The Management of Emergencies by the Primary Health Care Physician

World Health Organization
Eastern Mediterranean Regional Office

CHAPMAN & HALL MEDICAL

The Management of Emergencies by the Primary Health Care Physician

The Management of Emergencies by the Primary Health Care Physician

World Health Organization,
Eastern Mediterranean Regional Office

CHAPMAN & HALL MEDICAL
London · Glasgow · New York · Tokyo · Melbourne · Madras

Published by Chapman & Hall, 2–6 Boundary Row, London SE1 8HN

Chapman & Hall, 2–6 Boundary Row, London SE 1 8HN, UK

Blackie Academic & Professional, Wester Cleddens Road, Bishopbriggs, Glasgow G64 2NZ, UK

Chapman & Hall Inc., 29 West 35th Street, New York NY 10001, USA

Chapman & Hall Japan, Thomson Publishing Japan, Hirakawacho Nemoto Building, 6F, 1-7-11 Hirakawa-cho, Chiyoda-ku, Tokyo 102, Japan

Chapman & Hall Australia, Thomas Nelson Australia, 102 Dodds Street, South Melbourne, Victoria 3205, Australia

Chapman & Hall India, R. Seshadri, 32 Second Main Road, CIT East, Madras 600 035, India

First edition 1993

Typeset in 11/12½pt Palatino by VAP Enterprises, India
Printed in Great Britain by Page Bros, Norwich

ISBN 0 412 54800 3

A catalogue record for this book is available from the British Library

Library of Congress Cataloging-in-Publication data
The Management of emergencies for the primary health care physician/
World Health Organization, Eastern Mediterranean Regional Office. – – 1st ed.
p. cm.
Includes index.
ISBN 0-412-54800-3 (alk. paper)
1. Medical emergencies. I. World Health Organization. Regional Office for the Eastern Mediterranean.
[DNLM: 1. Emergencies. 2. Primary Health Care. WB 105 M2652 1993]
RC86. 7. M347 1993
616.02'5– –dc20
DNLM/DLC
For Library of Congress 93-8748
CIP

♾ Printed on permanent acid-free text paper, manufactured in accordance with the proposed ANSI/NISO Z 39.48-199X and ANSI Z 39.48-1984

Contents

Preface		viii
Acknowledgements		x
Units and abbreviations		xii
1	**Emergency life-saving procedures**	**1**
1.1	Airway management	1
1.2	Venous catheterization	11
1.3	Defibrillation	23
1.4	Use of military antishock trousers	24
1.5	Aspiration and drainage techniques	26
2	**Trauma**	**37**
2.1	Emergencies and resuscitation	38
2.2	Wounds	41
2.3	Cervicofacial injuries	50
2.4	Haemorrhage	59
3	**Burns**	**64**
3.1	Thermal burns	65
3.2	Chemical burns	69
3.3	Electrical burns	71
3.4	Sunburn	72
4	**Respiratory emergencies**	**74**
4.1	Infectious airway obstruction	74
4.2	Respiratory failure	76
4.3	Pulmonary oedema	84
4.4	Status asthmaticus	85
4.5	Haemoptysis	88
4.6	Cardiothoracic injuries	90

5	**Cardiovascular emergencies**	**103**
5.1	Chest pain	103
5.2	Shock	113
5.3	Anaphylaxis	124
5.4	Cardiopulmonary resuscitation	127
6	**Abdominal emergencies**	**142**
6.1	Acute abdominal pain	142
6.2	Food poisoning	149
6.3	Oesophageal varices	151
6.4	Abdominal injuries	152
6.5	Urological injury	159
7	**Central nervous system emergencies and head injuries**	**163**
7.1	Central nervous system emergencies	164
7.2	Psychiatric emergencies	177
7.3	Toxic confusional state due to acute alcohol withdrawal	181
7.4	The uncontrolled and potentially hostile patient	183
7.5	Head injuries	185
8	**Endocrine emergencies**	**189**
8.1	Diabetic ketoacidosis	189
8.2	Hypoglycaemia	190
8.3	Hyperglycaemic hyperosmolar non-ketotic coma	192
8.4	Thyrotoxic crisis	193
9	**Orthopaedic emergencies**	**196**
9.1	Fractures	196
9.2	Joint injuries	205
9.3	Dislocations	206
9.4	Fractures of vertebral bodies and spinal injuries	207
9.5	Hand injuries	210
9.6	Hand infections	213
9.7	Fishhook removal	215
10	**Fever**	**217**
10.1	Acute pyrexial illness	219
10.2	Heat stroke	225

10.3 Malaria 226
10.4 Typhoid 228
10.5 Septicaemia 230
10.6 Isolation procedures 231

11 Paediatric emergencies 235
11.1 Acute respiratory emergencies 236
11.2 Seizures 240
11.3 Cardiopulmonary resuscitation 241
11.4 Intravenous techniques 243
11.5 Endotracheal intubation 245
11.6 Fever 246
11.7 Otitis media 248
11.8 Pneumonia 249
11.9 Gastroenteritis and dehydration 250
11.10 Head injuries 251
11.11 Surgical emergencies 252

12 Poisoning 259
12.1 Drug overdose 260
12.2 Common forms of poisoning and intoxication 267
12.3 Bites and stings 275

13 Obstetric emergencies 279
13.1 Emergencies during pregnancy 280
13.2 Emergencies during labour 290
13.3 Emergencies following labour 296

14 Acquired immunodeficiency syndrome (AIDS) 298
14.1 Modes of transmission 299
14.2 Aetiology 301
14.3 Incubation period 302
14.4 Clinical manifestations 302
14.5 Clinical case definition 305
14.6 Prevention and control 305
14.7 Safety precautions for medical practitioners 309
14.8 Antibody testing 311
14.9 Management plan 311
14.10 Source references 314

Index 315

In the name of God, the Compassionate, the Merciful

Preface

One of the fundamental principles on which the Constitution of the World Health Organization was formulated more than four decades ago states: 'the extension to all peoples of the benefits of medical, psychological and related knowledge is essential to the fullest attainment of health'.

The Declaration of Alma-Ata in 1978 strongly influenced national health policies, which began to include the target of achieving good health for all by the year 2000 through primary health care. The declaration focused attention on the gross inequalities in health status between groups of people in both developed and developing countries, especially in rural areas. It emphasized that these inequalities could be removed with the cooperation and assistance of a number of sectors other than health. The main thrust was to rely on health care services provided by physicians, nurses and other health workers operating in basic and efficient health facilities located close to the target populations.

The availability, accessibility and affordability of health care services in peripheral areas continues to be an important concern for all countries of the Eastern Mediterranean Region. In view of this, WHO's Regional Office for the Eastern Mediterranean has taken a number of ambitious steps to establish strong foundations for primary health care in the region.

In 1990, the role of hospitals in the support of primary health care was reviewed in a WHO intercountry workshop held in Islamabad, Pakistan. In 1991, the subject of the integration of basic surgery into primary health care was discussed at the Thirty-eighth Session of the Regional Committee for the Eastern Mediterranean, in which the Committee urged member states to introduce training programmes to qualify newly graduated physicians in delivering basic surgery at primary health care and first-referral-level facilities. In 1992, a consultative meeting on the integration

of basic surgery into primary health care took place in Jordan. The many useful findings of these activities emphasized the vitality of the primary health care concept and the role that first-referral hospitals and physicians at the periphery can play in strengthening the primary health care system in order to provide efficient services to remote areas.

Central among target groups are those people whose lives are at stake, namely accident victims requiring immediate emergency medical or surgical treatment. Crucial minutes, or indeed seconds, can determine whether they live or die and, more often than not, it is the 'intermediaries', namely those mobilized to undertake this vital rescue mission, who determine the victim's fate. Therefore, these individuals need to be trained not only in the latest, but in the most effective first-aid care techniques known.

While much literature has been written on the subject, it has generally been aimed at the low and intermediate levels of first aid. There is very little aimed at the level of the primary health care practitioner, physician or qualified nurse. This provided the idea and motivation for this book, which is intended to fill the gap.

This book describes a number of medical and surgical procedures that have been chosen as appropriate for the primary health care physician who is *not* specialized in surgery or medicine. These procedures are considered essential for saving life pending referral to the nearest hospital.

I trust that this book will be an asset and will serve as an easy reference to primary health care physicians in managing medical, surgical and accident emergencies in remote areas of their countries, in order to provide immediate minimum emergency health care, and by so doing, to help to achieve the aim of good health for all by the year 2000.

Hussein A. Gezairy, M.D., F.R.C.S.
Regional Director
WHO, Eastern Mediterranean Region

Acknowledgements

A number of highly qualified health experts in various health and medical disciplines contributed their broad expertise and practical knowledge and experience to produce this much needed and vital addition to health literature.

WHO Regional Office for the Eastern Mediterranean acknowledges with thanks the valuable contributions made by prominent medical professionals in the preparation of this book. Especial thanks are owed to the following, who participated technically in preparation, review, editing and finalization of the work:

Professor Dr Galal El-Din Aref
Professor of Anaesthesia and Intensive Care Alexandria University, Egypt

Dr Hamed Mahmoud Arnaout
Professor of Surgery, Cairo University, Egypt

Dr Mohammad El Gundi
Professor of Surgery and Dean of the Faculty of Medicine, Alexandria University, Egypt

Professor Dr M. Shaker Abdullah
Professor of Paediatric Surgery, Alexandria University, Egypt

Professor Dr Ibrahim Lofty
Professor of Gynaecology and Obstetrics, Alexandria University, Egypt

Professor Morsy Arab
Professor of Internal Medicine, Alexandria University, Egypt

Dr A. Aly
Consultant, AIDS Information Exchange Centre, WHO, Alexandria, Egypt

Dr James Haworth
Chambesy, Switzerland

Ms E.J. Nicholson
External Editor, London, UK

Dr Sabry Mahmoud Shaban
Alexandria, Egypt

Dr. A. Barker
London, UK

Special acknowledgements are also due to Dr M.I. Al Khawashky, WHO Representative, Egypt, and to Dr Ataollah Amini, Regional Adviser for Health Systems Support, WHO/EMRO, for finalizing the book.

Units and abbreviations

°C	degrees Celsius
cm	centimetre
g	gram
h	hour
iu	international unit
kg	kilogram
kPa	kilopascal
l	litre
mg	milligram
min	minute
ml	millilitre
mmHg	millimetres of mercury
mmol	millimole
mosmol	milliosmole
µg	microgram
b.d.	twice a day
CNS	central nervous system
CPR	cardiopulmonary resuscitation
CSF	cerebral spinal fluid
CT	computed tomography
CVP	central venous pressure
ECF	extracellular fluid
ECG	electrocardiogram
EEG	electroencephalogram
ESR	erythrocyte sedimentation rate
i.m.	intramuscular
i.v.	intravenous
IPPR	intermittent positive pressure respiration
$Paco_2$	pulmonary arterial carbon dioxide saturation and tension
Pao_2	pulmonary arterial oxygen saturation and tension
PCV	packed cell volume
q.d.s.	four times a day
s.c.	subcutaneous
t.d.s.	three times a day

1

Emergency life-saving procedures

Summary of contents

AIRWAY MANAGEMENT 2

Control of airway with bag and mask 2
Oral airway insertion 3
Nasal airway insertion 4
Endotracheal intubation 5
Needle insertion into the cricothyroid membrane 9
Cricothyroidotomy 10

VENOUS CATHETERIZATION 11

Peripheral percutaneous venous catheterization 11
Peripheral central line placement 14
Venesection (cutdown) 16
Subclavian vein catheterization 19
Internal jugular catheterization 20

DEFIBRILLATION 23

USE OF MILITARY ANTISHOCK TROUSERS 24

ASPIRATION AND DRAINAGE TECHNIQUES 26

Thoracentesis 26
Chest tube insertion 28
Pericardiocentesis 30
Nasogastric tube insertion 31
Gastric lavage 32
Sengstaken–Blakemore tube insertion 33
Urethral catheterization 35

1.1 AIRWAY MANAGEMENT

1.1.1 CONTROL OF AIRWAY WITH BAG AND MASK

Indications

- Cardiopulmonary failure
- Respiratory failure
- Stridor or other partial obstruction of the upper respiratory tract

Position

The patient should be supine with the chin raised to ensure a clear airway (Fig. 1.1). Further advancement of the mandible downward and outward, thus translocating the temporomandibular joint, may be required to move the tongue to a point where it no longer obstructs the natural airway. Turning the head to either side with the jaw advanced may provide further aid.

Procedure

1. Clear the airway of dentures and debris.
2. Select the appropriate size of mask.
3. Position the head correctly.
4. Ventilate with the right hand at a rate of 10–15/min.
5. Observe the motion of the chest and absence of motion of the stomach.
6. Repeat until adequate oxygenation is achieved.

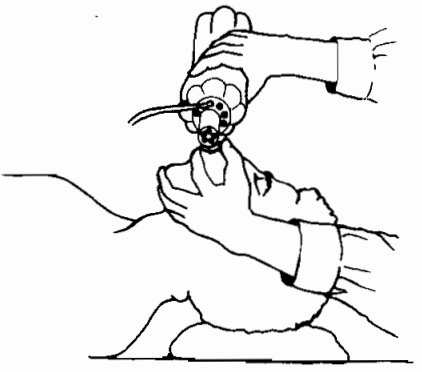


Fig. 1.1 Control of airway with bag and mask.

1.1.2 ORAL AIRWAY INSERTION

Indications

- Cardiorespiratory arrest
- Partial or total upper airway obstruction with or without nasal trauma

Contraindications

- Oral trauma
- Croup or other pharyngeal infection

Position

The patient should be supine with the chin raised, as for bag and mask ventilation.

Procedure

1. Open the patient's mouth by scissoring the jaw open with the thumb and forefinger of the right hand.
2. Remove dentures and clear the airway of debris.
3. Insert the airway so that the concave portions face away from the tongue (Fig. 1.2).
4. After insertion to the back of the pharynx, rotate the airway 180° and slide it inward to its full extent.

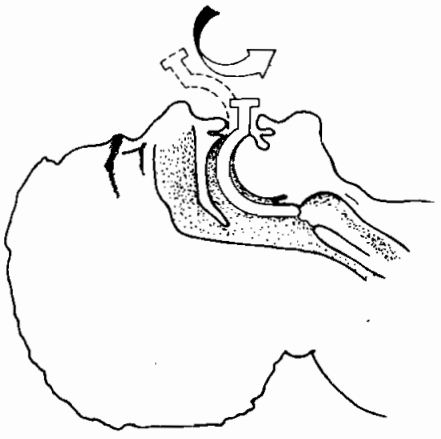


Fig. 1.2 Oral airway insertion.

1.1.3 NASAL AIRWAY INSERTION

Indications

- Cardiorespiratory arrest
- Partial or complete upper respiratory obstruction with or without oral trauma

Contraindications

- Nasal trauma
- Croup or other infections of the nasopharynx
- Enlarged adenoids

Procedure

1. Examine the nose for obstruction, foreign bodies or septal deviation.
2. Choose the nostril that seems to be the larger.
3. Clear the mouth and pharynx of dentures and debris.
4. Lubricate the anterior nostril and the nasal airway with water-soluble lubricant.
5. Insert the lubricated airway with the convex surface facing the convexity of the nasopharynx (Fig. 1.3).

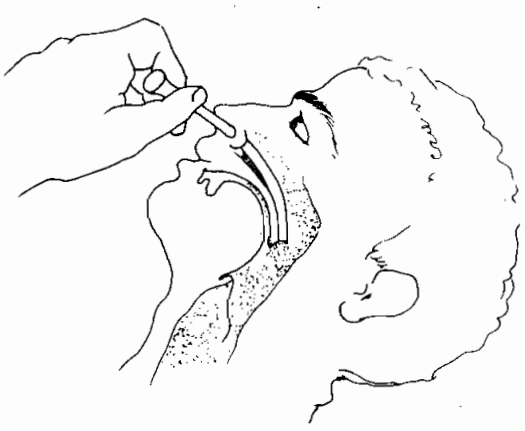


Fig. 1.3 Nasal airway insertion.

1.1.4 ENDOTRACHEAL INTUBATION

Indications

- Airway maintenance
- Cardiorespiratory failure
- Severe airway obstruction of a non-infectious nature
- Head and neck injuries with potential for obstruction or respiratory failure
- Superficial airway burns with potential for obstruction or respiratory failure
- Aspiration of gastric contents
- Protection from aspiration in unconscious patients

Contraindications

- Hypoxic patients before adequate oxygenation
- Known cervical spine injury

Methods

- Orotracheal intubation
- Nasotracheal intubation

OROTRACHEAL INTUBATION

Orotracheal intubation is preferable when a rapid emergency intubation is needed.

Equipment

General

- Intravenous access
- Working suction
- Resuscitation drugs
- Oxygen

Special

- Laryngoscope with desired blade attached
- Endotracheal tubes of appropriate sizes
- Malleable stylet
- Water-soluble lubricant
- 10 ml syringe
- Oropharyngeal airway

- Tape for fixation
- Tincture of benzoin

Position
The patient should be supine with the chin raised, as for bag and mask ventilation.

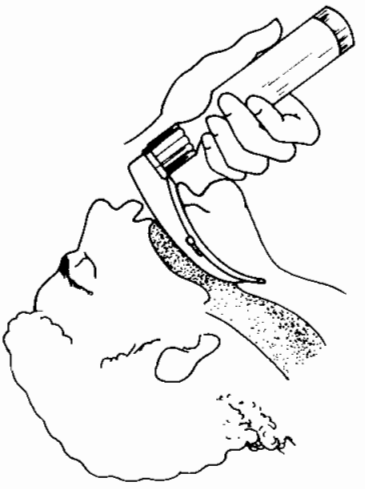


Fig. 1.4 Orotracheal intubation – insertion of laryngoscope.

Procedure
1. Provide adequate oxygenation of the patient, using bag and mask ventilation.
2. Select the appropriate-sized endotracheal tube.
3. Choose a laryngoscope blade and connect it to the handle. Check the light by opening the laryngoscope.
4. Check the cuff of the tube for leaks. Lubricate the tube and stylet. Insert the stylet into the tube with the appropriate curve.
5. Open the patient's mouth with the right hand.
6. Hold the laryngoscope with the left hand with the left wrist locked (Fig. 1.4).
7. Insert the laryngoscope as follows :
   - Insert the blade to the right side of the tongue, pushing the tongue to the left.
   - Advance the tip of the blade to the groove between the base of

the tongue and the epiglottis (curved blade), or advance the blade under the epiglottis (straight blade), and visualize the cords.

8. Insert the endotracheal tube as follows :
   - Hold the tube in the right hand with the bevel facing laterally.
   - Slide the tube between the cords (Fig. 1.5).
   - Remove the stylet.
   - Advance the tube 2 cm.
   - Remove the laryngoscope carefully.
   - Inflate the endotracheal tube cuff.
9. Ascertain the proper tube position:
   - Hold the tube stable; attach it to a positive pressure ventilation bag; apply intermittent positive pressure ventilation.
   - Ascertain bilateral chest expansion.
   - Conduct auscultation of air entry on both sides near the midaxillary line.
   - Ascertain the absence of breath sounds over the gastric area.
10. Secure the tube:
    - Place the oropharyngeal airway between the patient's teeth.
    - Apply tincture of benzoin to the cheeks.
    - Tape the tube at the level of the exit from the mouth and secure it to the cheeks.
    - Reconnect the tube to an appropriate ventilation device.

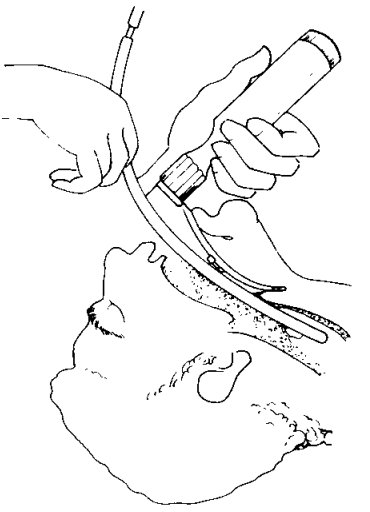


Fig. 1.5 Orotracheal intubation – insertion of endotracheal tube.

NASOTRACHEAL INTUBATION

Indications
- Patients with oral trauma
- Patients arranged for long-term nursing care
- Conscious patients with urgent need for airway protection

Contraindications
- Cases of nasal or upper facial trauma

Equipment and position
As for orotracheal intubation.

Procedure
Blind technique
1. Ascertain that all equipment is functioning properly.
2. Prepare the tube.
3. Ensure adequate oxygenation.
4. Insert the tube into the more patent nostril, and advance downward rather than backward into the pharynx.
5. Listen for respiratory sounds over the end of the tube and advance the tube slowly until they are heard.
6. Advance the tube through the glottis into the trachea during inspiration with a single rapid but gentle movement.
7. If the tube will not advance, observe the side of the neck in which the pressure of the tube protrudes outward, and rotate the tube in the opposite direction.
8. If the tube protrudes forward, a 360° rotation may facilitate passage through the glottis.

Direct visualization technique
1. Place the tube into the pharynx as above.
2. Visualize the glottis using a laryngoscope as in orotracheal intubation.
3. If necessary, grasp the tip of the endotracheal tube with Magill forceps and guide it into the trachea (do not grasp the balloon cuff) (Fig. 1.6).
4. Tube advancement should be done by an assistant.
5. Inflate the balloon cuff, ascertain proper tube position and secure the tube as for orotracheal intubation.

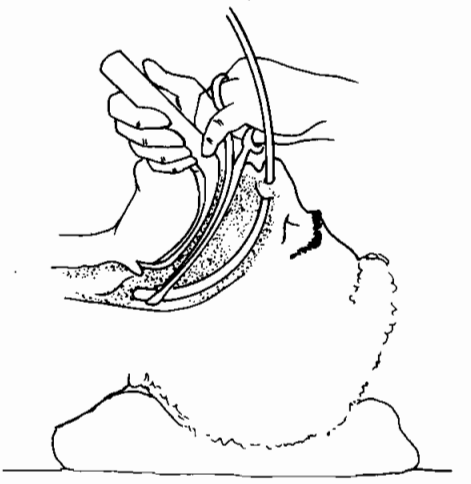


Fig. 1.6 Nasotracheal intubation.

1.1.5 NEEDLE INSERTION INTO THE CRICOTHYROID MEMBRANE

Indications

- Emergency establishment of a clear airway when other methods are contraindicated or equipment is not available.

Equipment

- Two large-bore needles (12 or 14 gauge with or without a plastic outer cannula)
- Sterile gloves
- Essential drugs

Position

The patient should be supine with the head in the neutral position. A small rolled towel should be placed between the shoulders.

Procedure

1. Prepare the skin.
2. Locate the cricothyroid membrane (just beneath the inferior prominence of the thyroid cartilage).

3. Insert the needle (with a plastic catheter over it if possible) through the skin and membrane into the trachea.
4. Remove the needle, leaving the catheter in place.
5. Tape the catheter or needle in place.
6. If the patient ventilates spontaneously, give oxygen at a rate of 6 l/min. Another needle may be placed alongside the first one to indicate exhalation.
7. If the patient is apnoeic, flush oxygen to the catheter for 1–2.5 seconds every 5 seconds, then disconnect from the oxygen source for 2.5–3 seconds. Repeat the cycle until the patient can breath spontaneously.
8. Convert to cricothyroidotomy or tracheostomy as soon as possible.

1.1.6 CRICOTHYROIDOTOMY

Indications

- Foreign body in the airway
- Oedema at or above the level of the vocal cords
- When orotracheal or nasotracheal intubation is not feasible

Contraindications

- Direct trauma to the larynx
- Fracture or crushed cricoid cartilage

Equipment

- Scalpel with a no. 15 blade
- Curved haemostat
- No. 6 or 8 cuffed tracheostomy tube or a 7·5 cm piece of intravenous tubing
- 1% lidocaine local anaesthetic; 3 ml syringe
- Suction apparatus
- Strong head light

Position

The patient should be supine with the arms restrained. Extension of the neck (if possible) facilitates the procedure.

Procedure

1. Palpate the cricothyroid space in the midline by running the left index finger along the trachea (Fig.1.7a).
2. Infiltrate the skin with 1% lidocaine if possible.
3. Keeping the index finger on the midline of the cricoid cartilage, make a 1.5 cm transverse incision directly through the skin. Make the skin incision deep enough so that the wound gapes (cutting the platysma muscle) (Fig.1.7b).
4. Slide the tip of the index finger into the incision with the fingernail resting on the cricothyroid membrane.
5. Slide the blade of the scalpel along the fingernail and poke the blade through the membrane.
6. Give the blade a twist to enlarge the opening (Fig.1.7c).
7. Keeping the finger in place, withdraw the scalpel and insert a curved haemostat in the opening (Fig.1.7d). Spread the blades of the haemostat to enlarge the opening and then insert a tracheostomy tube between the blades of the haemostat.
8. Tie the tracheostomy tube in place.
9. Suction of the trachea can now be carried out.

1.2 VENOUS CATHETERIZATION

1.2.1 PERIPHERAL PERCUTANEOUS VENOUS CATHETERIZATION

Position

The patient should be comfortably supine on the bed or on a stretcher.

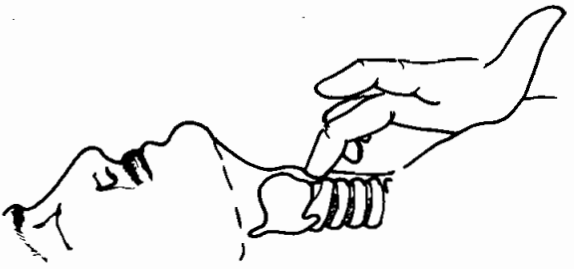
Procedure

Always explain the procedure to the patient before proceeding.

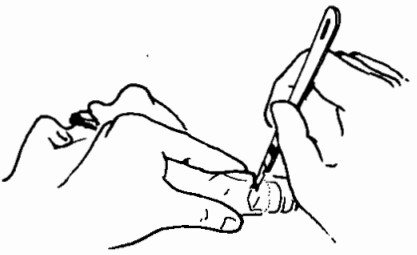
VEIN SELECTION

The two most important considerations when placing an intravenous cannula are the location of the vein and the size of the catheter. The following are commonly used intravenous sites:

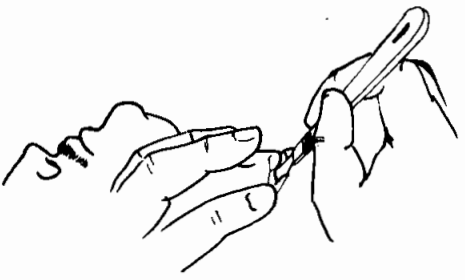
- Median forearm or basilic vein
- Superficial dorsal hand veins
- Cephalic or 'anaesthetist's' vein
- Volar wrist veins



(a)



(b)



(c)

(d)

Fig. 1.7 Cricothyroidotomy. (a) – (d) are explained in the text.

- Median cubital vein
- External jugular vein
- Saphenous vein

CATHETER SELECTION

At present, the following types of catheter system are available:

- Winged needle units (scalp vein, butterfly, etc.)
- Catheter-over-needle units (Angiocath, Medicut, Quikcath, etc.)
- Catheter-inside-needle units (Intracath, Desert subclavian catheter)

GAUGE SELECTION

Select the gauge appropriate to the patient, taking into consideration the purpose of the line and the fluid to be infused.

INSPECTION

Inspect and palpate the vein. A soft ballotable feel indicates that the vein is not phlebitic or sclerosed.

PRELIMINARIES

Ensure that all of the equipment and solutions to be used are readily at hand.

LOCAL ANAESTHESIA

Give a local anaesthetic if the patient is unduly anxious. Raise an intradermal wheal of plain 0.5% or 1% lidocaine.

TOURNIQUET

A tourniquet should be placed proximally and the area to be catheterized should be thoroughly prepared with a 70% isopropyl sponge or povidone iodine solution.

STABILIZATION

Place the finger on the skin to establish distal traction, thus firmly anchoring the vein in the subcutaneous tissue.

NEEDLE INTRODUCTION

1. Grasp the catheter between the thumb and forefinger, with the attached syringe resting against the palm of the hand.
2. The vein may be approached from above, at a venous junction, or from the side (the lateral entry is the most frequently used).
3. Holding the needle approximately 10–15° to the axis of the arm, briskly puncture the skin adjacent to the vein.
4. Once beneath the dermis, narrow the angle so that the catheter is nearly flush with the axis of the arm, and advance the catheter into the vein with the bevel up.
5. Once flashback of blood is seen in the needle hub, advance the catheter and needle along the course of the vein an additional 2–3 mm.
6. If a syringe is supplied with the catheter, aspirate it to ensure free return of blood, and advance the plastic catheter into the vein holding the needle stationary.
7. Pressurize the tip of the catheter to prevent backflow of blood, release the tourniquet, remove the needle completely from the catheter and attach the intravenous solution.

DRESSING

Place a small piece of gauze over the entry point, including the catheter hub. Tape the catheter securely into place.

1.2.2 PERIPHERAL CENTRAL LINE PLACEMENT

Indications

- Central venous pressure monitoring
- Swan–Ganz line placement
- Hyperalimentation access

Contraindications

- Patient with known superior vena cava syndrome

Position

The patient should be supine on a stretcher or bed, with the arm fully extended and abducted 90° from the shoulder in the same plane as the trunk in order to straighten the course of the axillary vein.

Procedure

VEIN SELECTION

The median vein or basilic vein distal to the antecubital fossa is selected, as its tributaries are in continuity with the great veins of the thorax. Apply a tourniquet to the upper arm.

PREPARATION

Strict asepsis is mandatory. Thoroughly prepare a wide area of skin around the vein with povidone iodine. Use sterile towels or paper sheets. Wear sterile gloves. The needle holder, skin sutures and scissors should be arranged in a convenient position on the sterile field.

CATHETER SELECTION

Select a catheter-inside-needle unit of the appropriate size. An assistant will be necessary to remove the catheter from its non-sterile container and pass it to the operator in an aseptic fashion.

ANAESTHESIA

Using a 25 gauge needle, raise a small wheal of 1% lidocaine at the site of catheterization.

NEEDLE INTRODUCTION

1. Loosen the needle and then reattach it to the hub of the plastic sleeve that guards the catheter.
2. Hold the introducing needle between the thumb and forefinger.
3. Gently introduce the needle into the vein. Successful introduction will result in a return of blood along the entire length of the catheter.

THREADING

1. Advance the introducing needle an additional 1–2 mm into the vein (Fig.1.8a).
2. Stabilize the needle and, with the other hand, advance the plastic catheter through the needle into the vein (Fig.1.8b).
3. Once the catheter is well into the vein, release the tourniquet.
4. Remove the needle from the vein and seat it firmly into the catheter hub (Fig.1.8c).
5. Place a needle-guard over the junction of the needle tip and the catheter.

6. Connect the catheter to the intravenous solution, directly (Fig.1.8d) or with an intervening three-way stopcock.
7. Instil additional lidocaine and suture the needle-guard and the hub of the catheter to the skin.

CONFIRMING POSITION

The intravenous bottle should be lowered below the edge of the bed to check for blood return before infusion. Obtain a chest X-ray to verify location of the catheter.

DRESSING

Dress the catheter with iodophor ointment and cover it with 5 × 5 cm sterile gauze. Tape the catheter securely into place.

1.2.3 VENESECTION (CUTDOWN)

Procedure

VEIN SELECTION

Select one of the following:

- Median basilic and median veins of the forearm
- Cephalic vein
- External jugular vein
- The origin of the saphenous vein in the ankle
- Saphenofemoral junction

TOURNIQUET

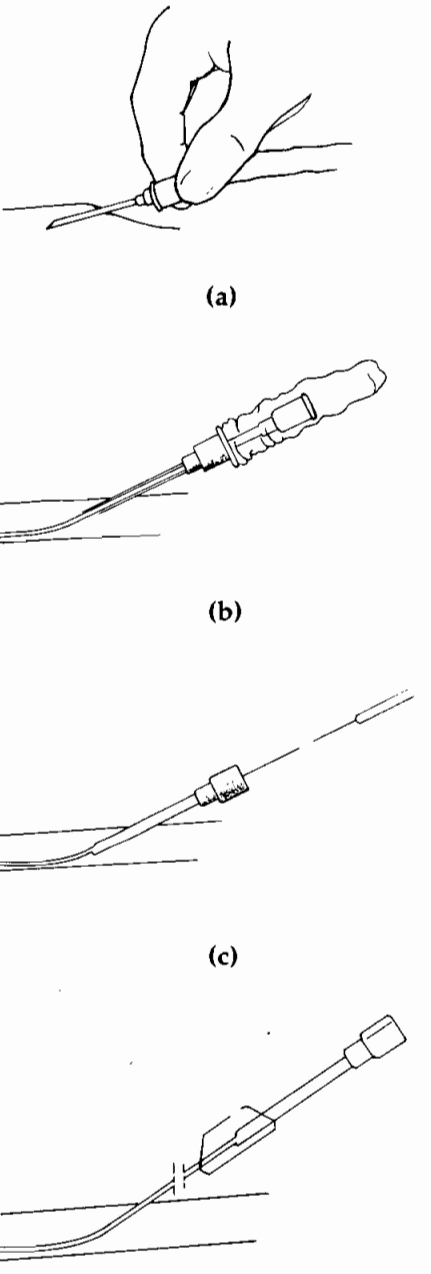
Apply a tourniquet proximally and thoroughly prepare the area to be cut down with a 70% isopropyl sponge or povidone iodine solution.

ANAESTHESIA

Give 5–10 ml of 1% lidocaine.

INCISION

1. Make an incision transverse to the axis of the vein. This type of incision should be used for all vessels with the exception of the



(a)

(b)

(c)

(d)

Fig. 1.8 Peripheral central line placement – threading. (a) – (d) are explained in the text.

saphenofemoral junction. In the upper extremity and the ankle an incision of 3–4 cm is sufficient.
2. Extend the incision until the subcutaneous tissue is free.

DISSECTION

Spread the tissues with a curved haemostat along the axis of the vein.

IDENTIFYING THE VEIN

The vein is a pulseless, thin-walled vessel whose blue colour blanches when distal traction is applied.

ISOLATION OF VEIN

1. Dissect the vein free from the surrounding tissue for a distance of 4 cm along its axis.
2. Pass fine ties proximally and distally along the vein.
3. Secure the distal tie and leave it long.
4. Leave the proximal tie loose and apply traction to it.

VENOTOMY

1. Pierce the midsection of the vein with a no. 11 scalpel blade.
2. Insert a fine haemostat a short distance into the vein.

INSERTION

1. Insert the catheter tip into the venotomy and gently advance it.
2. Flush the catheter with 5–10 ml of saline.
3. Connect the catheter to an intravenous solution.

CLOSURE

Once the catheter is placed and the infusion fluids are running easily, secure the proximal tie around the vein, including the catheter. Close the skin incision with three or four interrupted sutures of non-absorbable material.

DRESSING

Dress the wound with iodophor ointment and cover with a sterile gauze.

1.2.4 SUBCLAVIAN VEIN CATHETERIZATION

Indications

- Central venous pressure monitoring
- Swan–Ganz line
- Placement of percutaneous pacemaker

Contraindications

- Superior vena cava syndrome
- Thrombocytopenia
- Abnormal clotting studies

Position

Place the patient in the Trendelenburg position to distend the vein. Hyperextend the patient's shoulders. Place a rolled towel between the shoulder blades. Rotate the patient's head to the contralateral side.

Procedure

Either the right or left vein can be safely approached.

SKIN PREPARATION

Ensure the areas above and below the clavicle are thoroughly prepared. Wear sterile gloves.

LOCATION

Palpate for the angle formed by the intersection of the first rib and the clavicle at the junction of the middle and medial thirds of the clavicle. Move 1–2 cm laterally and inferiorly to this point. Slip the finger under the clavicle and locate the vein.

USE OF FINDING NEEDLE

1. Direct the needle in a plane 30–35° to the axis of the thorax, towards a point 2 cm above the suprasternal notch (Fig. 1.9a).
2. Attach the finding needle to a 10 ml syringe and advance it along this course to the inferior margin of the clavicle (Fig. 1.9b).
3. Walk the needle downwards until it will just pass beneath the clavicle.

4. Advance the needle towards the supraclavicular notch.
5. Maintain gentle negative pressure to detect the flashback of blood when entering the vein.

CATHETER INSERTION

1. Once the vein has been located, advance the needle and syringe an additional 2 –3 mm and hold firmly in place at the hub.
2. Remove the syringe, with the thumb of the non-dominant hand placed over the hub of the needle to prevent air aspiration.
3. Introduce the catheter into the needle and advance it until it is in a central position (Fig. 1.9c).
4. Withdraw the needle over the catheter.
5. Snap the needle-guard into place and attach the intravenous solution.
6. Suture the catheter to the skin of the chest wall midway between the clavicle and nipple, and dress with iodophor ointment and sterile gauze.

CONFIRMING POSITION

Lower the solution bottle below the edge of the bed, and observe the return of blood. Obtain a chest X-ray as soon as possible.

1.2.5 INTERNAL JUGULAR CATHETERIZATION

Indications, contraindications, equipment and position are the same as for subclavian catheterization.

Procedure

Prepare the skin with povidone iodine solution. Wear sterile gloves. Two approaches to the vein are possible: the high approach and the low approach.

HIGH APPROACH

1. Align the needle and syringe with the anterior border of the sternomastoid and adjacent to the triangle formed by the junction of the two bellies of the muscle (Fig. 1.10a).
2. Direct the needle approximately 30° posterior to the coronal plane, and towards the ipsilateral hip.
3. Palpate the pulse of the carotid artery and direct the needle lateral to this.

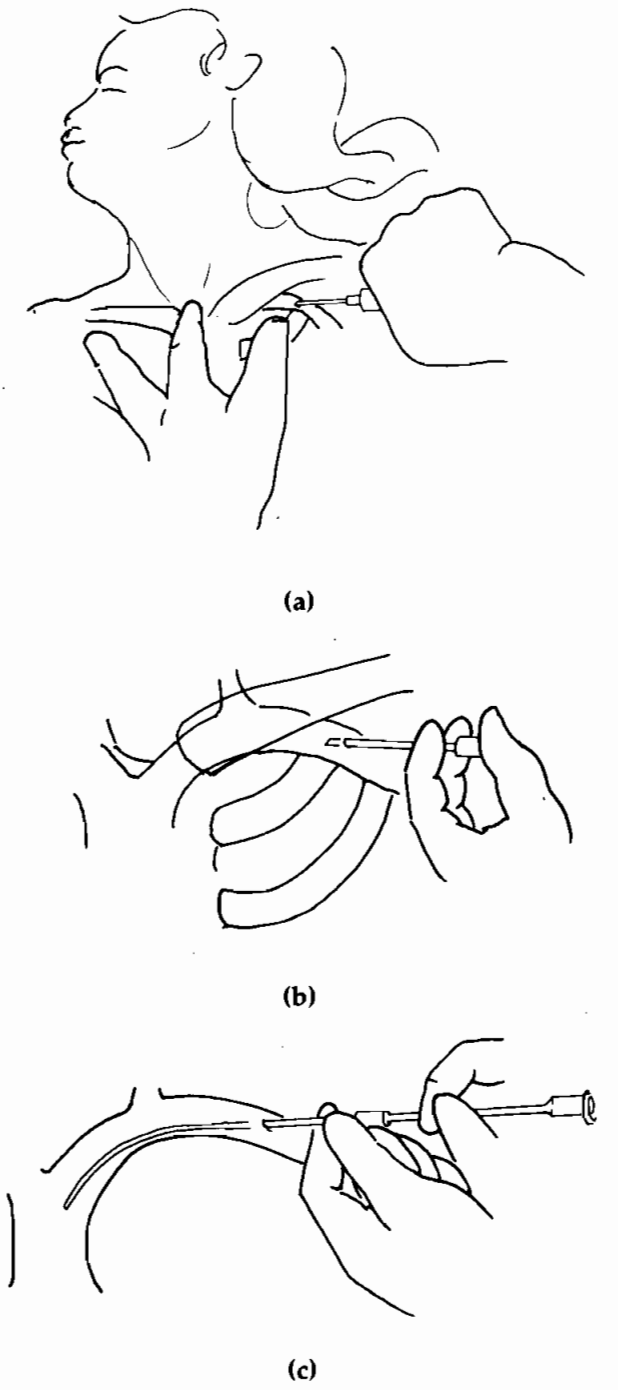
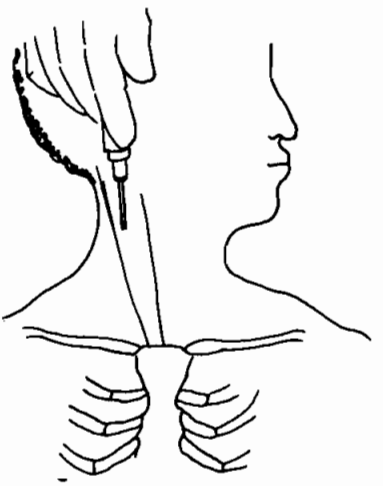


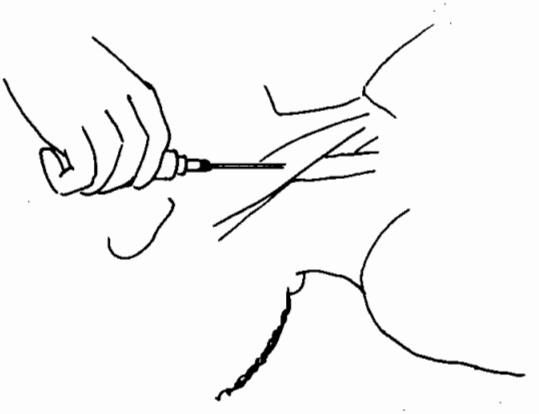
Fig. 1.9 Subclavian vein catheterization. (a) – (c) are explained in the text.

LOW APPROACH

1. The puncture site is 1 cm lateral to the lateral border of the sternal head of the sternomastoid, and 1 cm above the clavicular heads (Fig. 1.10b).
2. Maintain the axis of the needle and the syringe at 45° to the skin.
3. Direct the tip of the needle toward the xiphoid process.



(a)



(b)

Fig. 1.10 Internal jugular catheterization. (a) and (b) are explained in the text.

FOR BOTH APPROACHES

1. Apply slight negative pressure to the syringe. The flashback of blood indicates that the vein has been entered.
2. Advance the needle an additional 2–3 mm, remove the syringe and thread the catheter.
3. Check positioning of the catheter as before.
4. Secure the catheter to the skin with non-absorbable sutures, apply iodophor ointment and gauze and secure the catheter firmly in place with tape.

1.3 DEFIBRILLATION

Indications

- Ventricular fibrillation
- Conversion of supraventricular tachyarrythmias and ventricular tachycardia

Equipment

- A DC defibrillator with or without a monitor
- Paste or other material to reduce the electrical resistance of the skin

Procedure

1. Initiate basic life support until the defibrillator arrives.
2. Turn on the power and establish rhythm. Ensure that the unit is not in the synchronous mode used for cardioversion. If the defibrillator at hand does not include a monitor, a cardiac arrest victim should nevertheless receive an immediate DC shock. The level of shock to be applied is 20 – 100 J according to the age, weight and general condition of the patient.
3. Apply conductive material to the paddles to minimize burns to the patient's skin and to reduce the skin's electrical resistance.
4. Apply the paddles to the chest. The anterior (sternal) paddle is placed just beneath the right clavicle along the right sternal edge, the apical paddle is placed over the apex of the heart (at the anterior axillary line) (Fig. 1.11). The paddles should be placed on the chest with a slight twisting motion to distribute the electrode paste evenly.
5. Recheck the monitor, confirming the continued presence of ventricular fibrillation.

6. Clear the area and ensure that no personnel are in contact with the patient.
7. Firm pressure is applied to the paddle and the unit is discharged. Discharge buttons may be on both paddles requiring simultaneous depression, on a single paddle, or on the defibrillator itself. Evidence of effective discharge of the unit is manifested by skeletal muscle contraction.
8. Reassess rhythm and effective circulation. Check the monitor for rhythm. If ventricular fibrillation persists, repeat the above procedures immediately, increasing the level of shock as necessary. If an organized rhythm is present, check for the presence of a pulse. If no pulse is present, initiate cardiopulmonary resuscitation.

Remember: Always ensure that the patient is completely isolated before discharging.

Fig. 1.11 Placement of the defibrillator paddles.

1.4 USE OF MILITARY ANTISHOCK TROUSERS

Functions

- To provide haemostasis by increasing the pressure in the tissues surrounding a bleeding vessel

- To provide autotransfusion of the hypotensive patient
- To act as a splint of lower extremity and pelvic fractures, particularly with comminuted or unstable fractures

Indications

- Abdominal haemorrhage from blunt or penetrating trauma
- Severe lacerations or external blood loss
- Leaking or ruptured abdominal aneurysm
- Unstable femoral, pelvic and other lower extremity fractures in the patient with multiple traumas

Contraindications

- Pulmonary oedema
- Pregnancy
- Congestive heart failure and cardiogenic shock

Equipment

A pair of antishock trousers.

Position

The patient should be supine.

Procedure

1. Place the unit under the patient. The suit is ideally positioned with the upper aspect of the abdominal portion at the costal margin. The trousers should be adjusted to come to the level of the patient's ankle (Fig. 1.12a).
2. Secure the leg tabs to encircle the limbs with the garment and secure in place.
3. Connect the MAST unit to the inflation apparatus, inflate the trousers first (Fig. 1.12b), monitoring the patient's blood pressure constantly.
4. If the inflation of the leg panels is not sufficient to correct the haemodynamic abnormality, the abdominal portion of the garment should be inflated. For maximal autotransfusion effect, the pressure in the abdominal panels should not exceed the pressure in the leg panels.

1.5 ASPIRATION AND DRAINAGE TECHNIQUES

1.5.1 THORACENTESIS

Indications

- Hydrothorax
- Haemothorax
- Empyema

Contraindications

- Patient known to be taking anticoagulants
- Pneumonia (needle injury may cause empyema)

Position

- If fluid is expected the patient should be seated sideways on a chair with the arms draped over a pillow placed on a bedside stand.
- If air is expected the patient may recline, with the back up at a 30° angle and the hand placed behind the head.

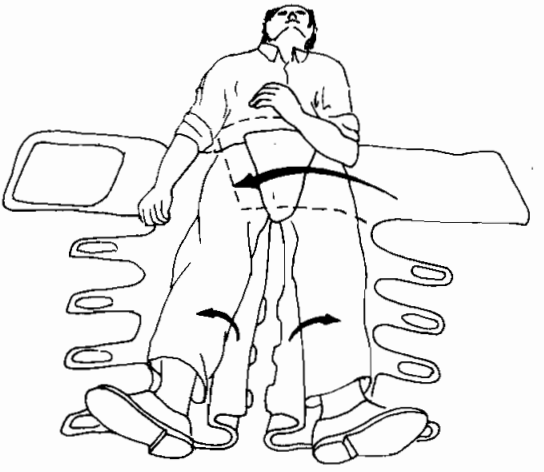
Procedure

PREPARATION

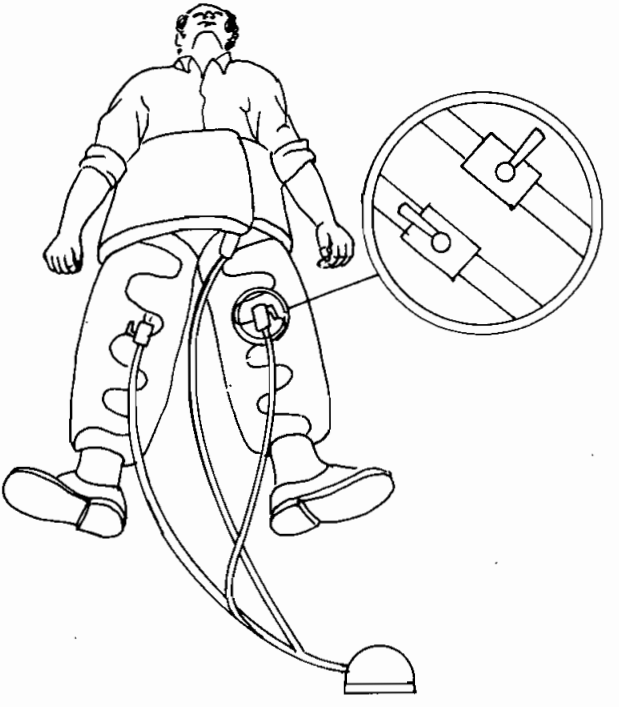
- Check the patient's name, the date and the site to be tapped on the X-ray film.
- Percuss the region to be tapped to locate the air or fluid level.
- Prepare the skin.

ANAESTHETIZING THE SITE OF TAPPING

1. Raise a skin wheal and anaesthetize with lidocaine.
2. Introduce the needle to touch the rib under the desired site and place 0.5–1 ml of lidocaine.
3. Walk the needle off the top of the rib border until it is free and advance it towards the pleura.
4. Place small amounts of anaesthetic and aspirate the syringe. If no material returns, advance the needle a further 0.5–1 cm.
5. Repeat until entry into the pleural space occurs (known by return of air or fluid on aspiration).
6. Pull the needle out until it is just outside the pleural surface.
7. Inject 1–2 ml of local anaesthetic to provide good pleural anaesthesia, then remove the needle.



(a)



(b)

Fig. 1.12 Use of military antishock trousers. (a) and (b) are explained in the text.

THORACENTESIS

1. Attach a small syringe to a large needle.
2. Advance the needle into the pleural space as described before.
3. When matter is returned, remove the syringe while the patient holds his or her breath and bears down against a closed glottis (Valsalva manoeuvre).
4. Cover the end of the needle with a gloved finger while the patient maintains the Valsalva manoeuvre.
5. Remove the needle over a catheter and attach a stopcock to the catheter hub.
6. Fluid or air may now be withdrawn slowly for as long a period as necessary using a large syringe.
7. Send a specimen from the early return to the laboratory.
8. When the procedure is complete, the entire unit should be removed and discarded.
9. Cover the wound with an adhesive dressing.

1.5.2 CHEST TUBE INSERTION

Indications

- Spontaneous pneumothorax
- Traumatic pneumothorax
- Haemothorax
- Hydrothorax

Contraindications

- Systemic anticoagulation
- Small, stable pneumothorax
- Empyema caused by acid-fast organisms
- Loculated hydrothorax or pneumothorax

Position

The patient should be supine with one or both hands behind the head.

Procedure

PREPARATION

Choose the site of insertion. The lateral chest wall behind the border of the pectoralis major muscle, the 5th or 6th intercostal

space which is usually lateral to the nipple, is an excellent site for insertion of tubes for either air or fluid drainage. Prepare the skin. Give local anaesthesia. Inject 5–10 ml of local anaesthetic immediately exterior to the pleura.

CHEST TUBE INSERTION

1. Choose the appropriate size of chest tube (18–20 French gauge catheter for air drainage, 28–32 F for fluid or blood drainage).
2. With the scalpel, make a small transverse incision 1.5 cm in length overlying the rib below the intercostal space.
3. Using blunt dissection with a haemostat, enter the pleural space, taking care not to enter more than 2.5 cm by accident.
4. Spread the haemostat to provide a satisfactory tract for the tube.
5. Insert a finger along the tube to ensure that the pleural space has been entered freely and that there is no adherent lung or diaphragm in the region of the selected placement.
6. With the patient holding his or her breath in full inspiration and bearing down slightly to increase intrathoracic pressure, remove the finger and place the catheter.
7. Place the haemostat on the tube to prevent excessive air entry into the chest.
8. Suture the tube in place using heavy non-absorbable suture material.
9. Connect the tube to a suitable drainage system.
10. Place a dressing around the catheter and tape the tube securely to the patient's chest.

DRAINAGE SYSTEM

The safest and simplest drainage system is the underwater seal. This consists of a bottle that is open to the air and has a tube that rests 1–2 cm below the surface of the saline-filled chamber. Air and fluid can easily exit through this. Drawing fluid up the chest cavity would necessitate an inspiratory force greater than 100 cmH_2O, which cannot be achieved by the diaphragm. All connections to drainage systems must be taped to prevent disconnection and air leakage (see also Chapter 4).

1.5.3 PERICARDIOCENTESIS

Indications

DIAGNOSTIC

- Pericardial effusion of unknown origin

THERAPEUTIC

- Cardiac tamponade
- Malignant pericardial effusion
- Uraemic pericardial effusion
- Traumatic haemopericardium
- Ruptured thoracic aortic aneurysm

Contraindications

- The use of systemic anticoagulants

Position

The patient should be supine.

Procedure

PREPARATION

Prepare the skin. Anaesthetize the skin and subcutaneous tissues: using a small syringe and needle and a local anaesthetic raise a skin wheal 2 cm below and 1 cm to the left of the xiphoid process. Advance the needle to contact the costal margin on the left side, and infiltrate local anaesthesia along this track up to and including the costal margin.

PERICARDIOCENTESIS

1. Attach a 10 ml syringe to a plastic catheter-over-needle unit.
2. Advance the catheter-over-needle unit and syringe to contact the left costal margin. This requires a course that would aim approximately for the middle of the left scapula (Fig. 1.13a).
3. After contacting the costal margin, insert the needle to the costal margin and advance it 0.5 cm at a time.
4. After each advancement infiltrate a small amount of local anaesthesia to clear the needle of tissue or thrombus, and attempt aspiration (Fig. 1.13b).

5. Once fluid is aspirated from the pericardium, advance the catheter-over-needle unit another 0.5–1 cm and remove the needle.
6. Aspirate the plastic catheter using a large syringe and a three-way stopcock.

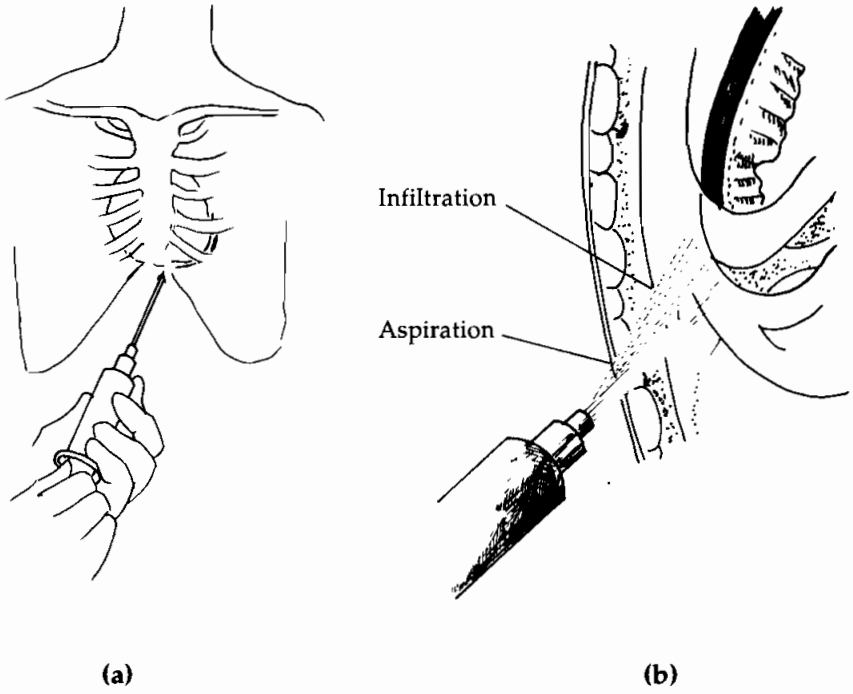


Fig. 1.13 Pericardiocentesis. (a) and (b) are explained in the text.

1.5.4 NASOGASTRIC TUBE INSERTION

Position

The patient should be sitting upright, or semireclining with the neck slightly flexed.

Procedure

1. Explain the procedure to the patient.
2. Measure the distance from the xiphoid to the top of the patient's head, and note the distance on the tube, relative to the black markers that are usually present.

3. Examine the nasal passage and select the most patent nostril.
4. Instruct the patient to breathe through the mouth.
5. Lubricate the distal 7.5 cm of the nasogastric tube liberally with water-soluble jelly.
6. Introduce the tip of the nasogastric tube into the nostril, and with one hand gently supporting the back of the patient's head, slowly advance the tube straight back, into the posterior nasopharynx.
7. Rotate the tube slowly if it will not easily advance further.
8. Ask the patient to swallow repeatedly as the tube advances past the soft palate and down the posterior pharynx. Once the patient has swallowed the tube past the glottis and into the upper oesophagus, the tube usually passes more readily. Continue to have the patient swallow as the tube is passed more rapidly into the stomach, supporting the head with one hand and advancing the tube with the other.
9. Advance the tube until the previously noted marker is at the level of the nose.
10. Check the proper position of the tube by aspirating obvious gastric contents with the catheter-tip syringe and/or injecting 20 ml air into the stomach by means of the same, while listening over the epigastrium with a stethoscope for a bubbling sound.
11. Fix the tube.
12. Connect the tube to the suction apparatus, and check again for proper function.

1.5.5 GASTRIC LAVAGE

Indications

- Massive gastrointestinal haemorrhage or bleeding
- Removal of known or suspected blood clot from the gastric lumen
- Removal of any matter not adequately aspirated by routine nasogastric intubation.

Contraindications

- Known or suspected oesophageal rupture, perforation, stricture or obstruction.

- Excessive facial trauma
- Cervical spinal cord injury

Position

The conscious patient should be sitting or semisitting. In the comatosed patient endotracheal intubation should be done first.

Procedure

1. Insertion of the Ewald tube is essentially the same as for insertion of a standard nasogastric tube.
2. Check the position of the tube.
3. Instil 100–200 ml lavage fluid (saline, iced saline or saline antacid slurry, as indicated) into the stomach through a series of catheter-tip syringes prepared by an assistant.
4. Aspirate the stomach by syringe or bulb-pump mechanism and discard the effluent in a second basin.
5. Repeat this process of instillation and aspiration until the aspirate is clear. In cases of ingestion, it may be advisable to instil a slurry of activated charcoal in saline as the final step before removal of the tube.

1.5.6 SENGSTAKEN–BLAKEMORE TUBE INSERTION

Indications

- Continued upper gastrointestinal haemorrhage known to be from oesophageal varices
- Massive and uncontrolled upper gastrointestinal bleeding from suspected varices in the known cirrhotic patient
- Continued bleeding from a documented Mallory–Weiss tear of the distal oesophageal and/or proximal gastric mucosa.

Contraindications

- Gastrointestinal bleeding from any site other than the distal oesophagus or gastric cardia
- Known or suspected oesophageal rupture or perforation
- Known or suspected oesophageal stricture or obstruction
- Obstructed nasopharynx
- Facial trauma
- Cervical spinal cord injury

Position

The preferred position is the patient sitting up (perhaps with the help of an assistant). A shocked patient should be supine with the head slightly raised.

Procedure

1. Sedate the patient with injectable narcotics, e.g. pethidine.
2. Test the Sengstaken–Blakemore (SB) tube by inflating each balloon with air under water and checking for any leaks. Then completely deflate the balloons.
3. Measure the nasogastric tube against the SB tube so that the tip of the nasogastric tube is at the upper end of the oesophageal balloon on the SB tube.
4. Anaesthetize the nasal passages and nasopharynx with local anaesthetic spray.
5. Lubricate the SB tube and the balloons with soluble jelly.
6. Insert the SB tube into the nose and gently advance it straight back into the pharynx. If the patient can cooperate, passage of the tube into and down the oesophagus is facilitated by having the patient swallow sips of water from a straw. Continue to advance the tube into the stomach until the 50 cm mark on the tube is at least at or in the nose.
7. Aspirate the gastric lumen; check the balloon is in the correct position.
8. Slowly inflate the gastric balloon with 50 ml of air only. Clamp the conical portion of the gastric balloon lumen, and gently withdraw the tube until resistance is felt, indicating engagement of the gastric balloon at the gastro-oesophageal junction.
9. Check the tube is in position again.
10. Advance the tube further down into the stomach before fully inflating the gastric balloon with an additional 200 ml of air and reclamping the lumen.
11. Withdraw the tube again until mild resistance is felt as the gastric balloon compresses the cardia.
12. Place the foam rubber cube around the tube and against the nose; tape it in place with adhesive tape.
13. Lavage the stomach through the gastric lumen to remove clots, and connect this lumen to gentle suction.
14. Insert the nasogastric tube through the opposite nostril and pass it into the oesophagus to the predetermined level.

15. Connect the nasogastric tube to another suction apparatus to remove blood and secretions from the now occluded oesophagus.
16. Ascertain the rate of bleeding. Gentle traction on the gastric balloon at the gastro-oesophageal junction may well gain control over the bleeding varices. Check the oesophagus aspirate (nasogastric tube) for fresh blood, and lavage the stomach through the gastric lumen of the SB tube to see if it clears. If bleeding continues, connect the oesophageal balloon lumen; slowly inflate the oesophageal balloon with air to 4 kPa (30 mmHg) pressure. Clamp the oesophageal balloon lumen to maintain this pressure and recheck at frequent intervals. If bleeding persists, the oesophageal balloon may be inflated to a maximum of 6 kPa (45 mmHg) of baseline pressure.

1.5.7 URETHRAL CATHETERIZATION

Indications

- Urinary retention
- Cross-haematuria with clots
- Sterile urinary collection for culture specimen
- Intermittent catheterization for neurogenic bladder

Contraindications

- Acute prostatitis
- Acute epididymitis
- Pelvic fractures with blood at the urethral meatus
- Recent transurethral resection of the prostate

Position

The patient should be placed supine with the thighs partially abducted. The female patient should draw up her knees into a frog-leg position.

Procedure

MALES

1. Apply a sterile drape around the penile shaft.
2. Prepare the glans and meatus with antiseptic solution.
3. Lubricate the catheter tip generously.

4. Advance the catheter up to the balloon sidearm. With the penis pulled taut and almost horizontal, the catheter glides easily with only slight resistance and some discomfort just as the catheter traverses the external sphincter. Never force a catheter in attempting to bypass an obstruction.
5. Obtain a urine specimen.
6. Inflate the retaining balloon with sterile saline (usually 5 ml). If there is pain, the catheter may be in the prostatic urethra; deflate the balloon and reposition the catheter.
7. Withdraw the catheter gently to seat the balloon against the bladder neck.
8. Connect the catheter to a drainage bag.
9. Secure the drainage tubing with tape to the medial thigh to prevent accidental dislodgement.

FEMALES

1. Prepare the perineum and vaginal introitus with antiseptic solution.
2. Expose the urethral meatus by spreading the labia.
3. Lubricate the catheter tip generously.
4. Introduce the catheter into the meatus.
5. Advance the catheter about half-way.
6. Obtain a urine specimen. Gentle irrigation with sterile saline will help ascertain proper positioning within the bladder.
7. Inflate the retaining balloon with 5 ml sterile saline.
8. Withdraw the catheter gently to position it against the bladder neck.
9. Connect it to a drainage bag.
10. Secure the drainage tubing with tape to the medial thigh to prevent accidental dislodgement.

2

Trauma

Summary of contents

EMERGENCIES AND RESUSCITATION 38

WOUNDS 41

Closed wounds 41
Contusion 41
Multiple bruises 41
Haematoma 42
Abrasion 43
Open wounds 43
Incised wounds 43
Punctures and stab wounds 44
Lacerated wounds 44
War wounds 46
Tetanus prophylaxis 49

CERVICOFACIAL INJURIES 50

Neck injuries 50
Facial injuries 52
Cut throat 55
Eye injuries 56

HAEMORRHAGE 59

Type 59
Management 61
Epistaxis 63

2.1 EMERGENCIES AND RESUSCITATION

Initial management of an injured patient is usually provided by an emergency room physician, and much of the prognosis depends on this initial care.

General principles of management

1. Assess the degree of injury rapidly but thoroughly.
2. Treat life-threatening conditions immediately: respiratory distress, severe bleeding and shock.
3. Improvise dressings, splints and transportation.
4. Arrange for prompt definitive treatment.

Patient evaluation

Ascertain the degree and type of damage and any serious underlying medical problem, e.g. cardiac disease.

RESPIRATORY DISTRESS

- Stridor and suprasternal or intercostal retraction indicate airway obstruction.
- Shortness of breath may be due to chest injury or shock.
- Respiratory depression may occur in head injury or severe shock.
- Cyanosis is always due to poor oxygenation, whatever the cause.

SHOCK

Typical signs are faintness, pallor, restlessness, cold clammy sweat, thirst, air-hunger, and a weak, usually rapid, pulse. Differentiate oligaemic from neurogenic shock.

FRACTURES AND DISLOCATIONS

Palpate carefully from head to foot, move all joints cautiously and exert gentle pressure on the spine, chest and pelvis. Pain, swelling, ecchymosis, deformity and limited movement are classic signs; few or none of these may be apparent immediately after injury.

BRAIN AND SPINAL CORD DAMAGE

Note the state of consciousness, gross skin sensation, and ability to move extremities actively.

INTERNAL INJURY

- Overt localizing signs are often minimal.
- Hypovolaemic shock in the absence of overt bleeding or severe tissue damage suggests internal haemorrhage.
- Chest pain with respiratory distress and abdominal pain with signs of peritoneal irritation point to visceral injury.

Life-saving procedures

Evaluation and restoration of vital functions should be the first step in the care of an injured patient and should follow this sequence of priorities:

1. Maintain adequate respiration.
2. Support the circulation.
3. Treat shock, if present.
4. Stop bleeding.
5. Check the state of consciousness.

RESPIRATION

Maintain a clear airway by:

removing any foreign body present in the mouth (broken teeth, clots or food debris); make sure the tongue itself is not obstructing the airway;

applying suction to remove secretions or blood;

insertion of an oropharyngeal airway or an endotracheal tube in cases of facial fracture.

Start artificial respiration if there are no respiratory movements.

CIRCULATION

Check blood pressure and pulse rate frequently. Establish an i.v. line, preferably by cutdown. Lactated Ringer's or normal saline solution may be started, and the solution and rate of infusion may be changed according to the condition. At the same time, take blood samples for typing and crossmatching, complete blood count, glucose and electrolytes.

SHOCK

Shock should be anticipated in any patient with multiple or serious injuries, and should be treated accordingly (see Chapter 5).

BLEEDING

Bleeding should be controlled by tourniquet or by compression using sterile gauze and pressure bandaging.

STATE OF CONSCIOUSNESS

It is important to record the patient's level of consciousness at the time of arrival in the emergency room, for further evaluation.

Management

WOUNDS

Cover all wounds with sterile dressings or towels. Cover exposed abdominal viscera with sterile saline dressings; make no attempt to return them to the peritoneal cavity in the emergency room.

MEDICATION

In cases of severe pain, i.v. pethidine hydrochloride or morphine may be given initially; i.m. medications may be poorly absorbed during the period of shock.

FRACTURES

Splint any obvious or suspected fractures.

CONSULTANTS

Call appropriate consultants after establishing emergency measures and completing the physical examination.

SUMMARY

The management of an injured patient may be summarized as follows:

1. Establish and maintain a clear airway and effective respiration.
2. Provide circulatory support.
3. Rapidly evaluate the patient's condition, including state of consciousness.
4. Do a thorough physical examination after undressing the patient.
5. Splint all obvious or suspected fractures immediately, and avoid injudicious movement of the patient.
6. Begin proper care of the wounds and administer appropriate pain medication as necessary.

7. Obtain blood and urine samples for appropriate tests, and order pertinent X-rays.
8. Obtain appropriate consultations.

Transportation of injured patients

Incorrect methods of moving patients can increase injuries. Where spinal injury is suspected, the use of a collar before moving the patient may be life-saving. Lift severely injured patients with care and improvise stretchers from blankets, boards and doors when necessary. Transport the following types of case in the recumbent position, on a stretcher, preferably in an ambulance: head and internal injuries; fractures of the spine, pelvis and long bones of the lower extremities; shock; major wounds in general.

2.2 WOUNDS

A wound is a disruption of the continuity of the tissues due to external violence.

2.2.1 CLOSED WOUNDS

Contusion

A contusion is an injury to the soft tissues while the skin remains intact. Damage to soft tissues is accompanied by blood vessel injuries and extravasation. The damaged area becomes swollen, discoloured and tense. The colour of the area varies with the duration of time that has elapsed since the occurrence of the trauma, due to blood derangement. First, it is red (oxyhaemoglobin), then blue (reduced haemoglobin), later greenish-yellow (haemosiderin), and finally the colour disappears and the area returns to normal. This condition is called a bruise or ecchymosis (e.g. black eye).

Multiple bruises

The presence of multiple bruises, ecchymoses or petechiae indicates a serious illness. Bruises or ecchymoses occur as a result of capillary bleeding into the tissues. They may be due to trauma or abnormal haematological states, such as platelet abnormality and deficiency of clotting factors. The common causes are:

- Trauma
- Bleeding disorders or haematological problems such as leukaemia,

haemophilia, von Willebrand's disease, idiopathic thrombocytopenic purpura
- Post-infectious conditions such as meningococcaemia
- Child abuse

TREATMENT OF CONTUSION AND MULTIPLE BRUISES

1. Sterilize the skin with an alcoholic antiseptic to avoid infection, and apply a pressure bandage to control pain and swelling.
2. If there is any suspicion of child abuse transfer the child to hospital.
3. Transfer suspected cases of bleeding disorder to hospital.

Haematoma

Haematoma is a localized collection of extravasated blood. It may be classified according to its anatomical site into:

- Subcutaneous (under the skin)
- Subfascial (deep to the fascia)
- Intrafascial (within the fascia)
- Intramuscular (within the muscles)
- Extracranial (outside the skull) including:
  subcutaneous (under the skin of the scalp)
  subaponeurotic (under the galea aponeurotica)
  subpericranial (under the pericranium)
- Intracranial (inside the skull), including:
  extradural (between the skull and dura)
  subdural (between the dura mater and arachnoid membrane)
  intracerebral haemorrhage
- Retroperitoneal (behind the peritoneum)
- Subperiosteal (under the periosteum)
- Haemoscrotum (extravasated blood in the scrotum)
- Haemoarthrosis (bleeding in a joint)

TREATMENT

1. If the haematoma is **small**, sterilize the skin and apply a pressure bandage.
2. If it is **large**, aspirate under aseptic conditions.
3. If it is **large and clotted**, evacuate by means of a small peripheral incision.
4. If it is **infected**, incise and drain as an abscess.

During the first 48 hours apply cold compresses on the site of the haematoma to help clotting. After 48 hours apply warm compresses to help absorption of the haematoma.

EVACUATION OF SUBUNGUAL HAEMATOMA

Accumulation of blood under the nail is a common consequence of a blunt trauma or crushing injury to the fingertips. It is very painful and can be relieved in the emergency room or health centre simply by making a hole in the nail to evacuate the blood.

To evacuate the haematoma, make a hole in the nail at the site of the haematoma with one end of a paperclip (heated red-hot on an alcohol sponge flame) or with the pointed end of a no. 11 blade. If the blood extends to the distal of the nailbed, it may be evacuated through a soft-tissue incision made under the distal end of the nail.

Abrasion

Abrasion is due to rubbing of the skin against a rough surface, resulting in the superficial layers of the skin being scraped away, exposing the sensory nerve endings and causing pain. The exposed area is liable to infection.

TREATMENT

Clean the skin with a bland antiseptic such as 1% cetrimide, and apply a dressing of penicillin cream for several days. Do not use tincture of iodine because it causes severe pain and much scarring.

2.2.2 OPEN WOUNDS

Incised wounds

An incised wound is a painful clean-cut wound which bleeds profusely; it is caused by sharp instruments. Deeper structures such as blood vessels may also be injured.

TREATMENT

1. Apply a sterile dressing over the wound.
2. Control external bleeding by applying a pressure bandage.
3. Transfer the patient to hospital.

Punctures and stab wounds

Punctures and stab wounds are due to penetration by sharp instruments, such as a knife or a nail. Such wounds are characterized by:

- being small but deep;
- possible severe damage to deeper structures;
- the possible presence of an exit wound;
- possible internal haemorrhage (in body cavities);
- possible retention of part of the broken instrument in the wound;
- liability to infection, which is usually deep.

TREATMENT

Treatment is similar to that for an incised wound:

1. Clean the area.
2. Irrigate the wound with sterile saline.
3. With the patient under local anaesthesia, make a cruciate incision of the skin corners and probe to determine the depth of the wound and the presence of a foreign body.
4. Remove any foreign material and any devitalized tissue.
5. Where the extremities are involved, careful evaluation for neurovascular damage is necessary.
6. Elevate the involved part.
7. Give tetanus toxoid.

Lacerated wounds

Lacerated wounds result from severe violence by a blunt instrument and from car accidents. They are usually associated with fractures and/or other injuries to the viscera, arteries and nerves. The wound is characterized as follows:

- The wound is irregular and the skin is lacerated.
- The surrounding tissues are swollen.
- The crushed tissues are insensitive.
- The blood vessels are torn across but their edges retract, therefore the amount of bleeding is usually small.
- Tissue destruction is great.
- Tearing of vessels may lead to sudden death of large masses of tissue (necrosis).
- Infection is very likely to occur.

TREATMENT

1. Remove any foreign bodies.
2. Apply a dry, sterile dressing and secure it in place.
3. Do not apply antiseptic solution or powder to the wound; it is not necessary to clean the skin around the wound with soap or antiseptic.
4. Arrange for the earliest possible cleaning and debridement, and closure under aseptic conditions.

TREATMENT OF LACERATED WOUNDS AT CERTAIN SITES

Fingertip injuries

Contusion and/or laceration of the distal phalanx of the finger is a common injury caused by the closing of a door on the finger; it usually involves the nail matrix. The aim of treatment is to restore normal sensitivity and protect the nailbed from further injury.

1. Apply a temporary dressing.
2. Obtain an X-ray to establish the presence of a fracture or foreign bodies.
3. If a fracture is suspected apply a splint and refer to a consultant.
4. When there is avulsion of the tip or suspicion of injury to tendons, consult a hand surgeon for further management.

Lacerations to the face

1. Close all lacerations to the face by anatomical layer rather than by a single layer or adhesive tape.
2. Suture the skin with small, closely placed sutures without infolding or unnecessary tension on the skin edges.

It is advisable to consult a surgeon for deep wounds because of the importance of obtaining good cosmetic results.

Lacerations about the mouth

1. Suture 'all-through' lacerations of the lip on the external surface, and leave the internal surface unsutured for drainage so that infection is not trapped.
2. Lacerations of the mucous membranes within the mouth that do not involve the vermillion border or the skin usually do not require suturing.
3. Lacerations of the mucous membranes of the hard or soft palate, posterior pharyngeal wall or floor of the mouth require special attention, and should be referred to an oral surgeon for repair.

4. Lacerations of the floor of the mouth may result in severe bleeding and may require surgical attention to control haemorrhage, but suturing is rarely required.

Lacerations of the tongue

Lacerations of the tongue do not usually require suturing; bleeding can be stopped by pressure (holding the tongue with a piece of gauze or holding an ice cube on it). However, a laceration on the edge of the tongue with obvious separation may be sutured with catgut sutures only.

2.2.3 WAR WOUNDS

Wounds sustained in armed conflict produce fractures that are nearly always accompanied by extensive destruction of the soft tissue. The type and velocity of the bullet or missile determine the character of the wound: high-velocity bullets cause a small entry wound but often result in a massive explosive exit wound, creating a shock wave that is widely destructive of tissues. Bones struck by such bullets are often shattered into fragments, which act as secondary missiles going in all directions.

The majority of war wounds are of the penetrating or the perforating type. In such wounds the skin loss is much smaller than that found in the deeper tissues. Disruption is soon followed by reactionary oedema, and within a few hours this stage passes into that of visible infection. Metallic foreign bodies carry infection and it is the extent and virulence of infection that really matters. The wound track is lined with devitalized and necrotic tissue, which forms an excellent culture medium for both anaerobic and aerobic organisms. Therefore the most essential measure in the emergency treatment of war wounds is the excision of dead and dying tissue, in order to prevent infection. The first principle of treatment is always **excise all dead and dying tissues.** The following types of wound may be encountered:

- Penetrating wounds or lodging wounds (no exit). These in particular may contain foreign bodies, such as bits of cloth, mud etc. which should be removed together with devitalized and necrotic tissue.
- Multiple superficial wounds. These only require dressing.
- Perforating wounds due to traversing bullets and missiles. Bullet wounds with a small entry and exit and without swelling of the intervening tissues and other signs of injury to important structures only need dressing. Wounds with a small entry and large exit require cone-shaped excision.
- Traversing wounds or tunnel wounds. If the tunnel is superficial, good drainage is recommended to avoid infection.

- Gutter wounds. These are very common and easy to treat, being clearly visible.
- Traumatic amputation where the limb is almost amputated and attached only by skin or devitalized tissue. Such cases usually require amputation.

Management of recent wounds

Eight hours is the maximum allowable time for wound excision during which suturing can be performed. After that infection will have begun to establish itself. The steps in treatment of war wounds to the limbs are as follows:

1. First aid:
   - Splint the wound.
   - Place the patient on a stretcher.
   - Control external haemorrhage.
2. War wounds require early operation. If the patient's condition permits, then the sooner it is performed the better. If the patient is in shock, he or she should be resuscitated and operated on as soon as possible. The following rules should be followed:
   - During the rush of cases, select those for resuscitation and those that need immediate operation, and arrange the work to get these cases to the theatre without delay.
   - Ensure correct preoperative care whatever the circumstances.
   - Ensure the patient is transferred to the operating theatre on the stretcher and lifted with care. If an X-ray is needed, use a portable X-ray unit.
   - Tourniquets should not be applied except for amputations and to control rapid blood loss.
   - If immediate amputation is needed, a second opinion should be sought to confirm that the procedure is unavoidable.
   - Guillotine amputation should **not** be performed.
   - Perform routine wound excision but avoid the common error of undue sacrifice of the skin.
   - Ensure adequate exposure of the wound and thorough debridement to excise devitalized and necrotic tissues.
   - Ensure proper recording of the operative details, whatever the circumstances.
3. After excision:
   - Support or immobilize the injured area to avoid or control oedema. Elevate the limb. Rest the injured part by dressing and bandage or closed plaster technique.

- Observe the circulation in the peripheral part of the limb.
- Give prophylactic antibiotics and chemotherapy.
- Give gas-gangrene serum and tetanus toxoid.
- Follow up.

Missile and bullet injuries to the chest

Missile and bullet wounds to the chest constitute about one-third of cases in the battlefield. Such wounds have a high death rate due to:

- damage to the heart and major vessels;
- associated severe injuries of the head or abdomen;
- pleural complications such as pneumothorax and haemothorax.

Injury may vary from a simple chest wound or fractured rib to severe laceration of the lung and mediastinum. The severity depends upon:

- the type of missile: a clean bullet is less damaging than shrapnel, which causes lacerations;
- the site of injury: the least fatal area is the middle third of the chest, rather than the upper third near the trachea and major vessels or the lower third where the diaphragm and abdominal viscera are found;
- whether the external wound is closed or open.

CLOSED WOUNDS

If the bullet entered with minimal damage to the chest wall, drilled a clean hole in the lung, caused a small air leak and a minimal pneumothorax, the wound is closed. If larger lung vessels or chest wall vessels are torn, haemothorax will occur. The bullet may pass in and out or be retained.

Treatment

Besides the general lines of treatment and treatment of the associated lesions (pneumothorax, haemothorax and fractured ribs), local excision of the wound track as far as the pleura is required.

OPEN (LACERATED OR SUCKING) WOUNDS

If the bullet explodes into shrapnel as it passes through the chest it will cause severe lacerating damage to the surrounding tissues. Shock and distress is always severe, with cyanosis due to tension

pneumothorax or ineffective respiratory effects. The wound is covered by a frothy mass of blood, with or without frothy sanguinous sputum.

Treatment

1. Apply a tight closure of the chest wall immediately to limit the resulting pneumothorax and allow effective respiration and coughing.
2. Give general treatment for shock.
3. Apply local treatment of the wound.
4. Excise the lacerated edges.
5. Remove metal fragments and cloth.
6. Perform quick pleural lavage and close the wound in layers, applying underwater seal drainage as necessary.
7. Give antibiotics (local and systemic).
8. This conservative treatment gives better results than that of formal thoracotomy. Later, under more appropriate conditions, undertake elective treatment for foreign bodies, resection of the damaged lung and repair of the chest wall. In fact, prompt treatment of shock, good care of the wound and the use of underwater seal drainage markedly reduce the incidence of complicating injections.

For treatment of pneumothorax, haemothorax and other complications, see Chapter 4.

2.2.4 TETANUS PROPHYLAXIS

Tetanus is absolutely preventable by prior active immunization. Prophylaxis against tetanus must be given to every patient suffering from a break in the body's epithelial barrier (skin or mucous membranes), as follows:

- Previously immunized persons who received the last dose of tetanus toxoid within the last 10 years: give 0.5 ml adsorbed tetanus toxoid. This booster dose may be omitted if the wound is clean and superficial.
- Previously immunized persons who received the last dose of tetanus toxoid more than 10 years ago: give 0.5 ml adsorbed tetanus toxoid for all wounds.
- Immunization status unknown: clean superficial wounds (not tetanus-prone) and give 0.5 ml adsorbed tetanus toxoid. All other wounds (potentially tetanus-prone) give 0.5 ml adsorbed tetanus toxoid plus 250 iu human tetanus immunoglobulin.

(Hyper-Tet). These two steps constitute combined active and passive immunization.

In addition, immediate meticulous care of the fresh wound is of prime importance. Removal of devitalized tissue, blood clots, foreign bodies, obliteration of dead space and prevention of tissue ischaemia in the wound are the objectives of the initial treatment. Wounds that are seen late or that are severely infected may be left unsutured after debridement, protected by sterile dressing for 3–5 days and then closed by delayed suture.

At the same time as tetanus prophylaxis is given, penicillin should be given in large doses to prevent infection.

Remember: Antitetanus serum (ATS) is no longer the standard treatment for tetanus prophylaxis. It can result in allergic reactions that may mount to anaphylaxis.

2.3 CERVICOFACIAL INJURIES

2.3.1 NECK INJURIES

Neck injuries are potentially dangerous because of the many vital structures liable to be affected. Penetrating wounds to the posterior neck endanger the vertebral column, the cervical spinal cord, the interosseous portion of the vertebral artery, and the neck muscles. Penetrating wounds to the anterior and lateral neck endanger the larynx, trachea, oesophagus, thyroid, carotid arteries, subclavian arteries, jugular vein and subclavian vein.

Blunt trauma may produce fractures of the cervical spine with or without spinal cord injuries. Traumatic occlusion of the carotid arteries, and laryngeal and tracheal injuries complicated by haemorrhage and airway obstruction, are commonly caused by blunt trauma to the neck front. In neck injuries, the head and chest must be carefully examined for possible traumatic complications.

Clinical picture

Laryngeal and tracheal injuries may be silent or may present with hoarseness, laryngeal stridor or dyspnoea due to airway obstruction or blood inhalation or both. These symptoms may be associated with subcutaneous emphysema in cases where the airway is torn. Oesophageal injuries rarely occur in isolation, but are usually associated with other injuries.

Injuries to the spine should be suspected in cases of deceleration

injury and in cases of violent trauma to the neck. In such cases the head and neck should be immobilized until a cervical X-ray can be taken to rule out cervical fracture, particularly in patients with cervical pain or tenderness.

Injuries to the major vessels are possible in penetrating wounds; the subclavian artery may be lacerated if the clavicle or first rib is fractured. The patient usually presents with external blood loss, haematoma formation and varying degrees of shock. In some cases bleeding may be contained and the injury may temporarily go undetected. Auscultation of the head may reveal bruits, which point to arterial injury.

Diagnosis

The location of trauma suggests the possible structure injured. The type of trauma (penetrating or blunt) will suggest the type of injury. X-ray of the soft tissues and cervical spine will confirm the presence of fractures, locate opaque foreign bodies and help to determine the track of a bullet. The main results of blunt cervical trauma are cervical fracture, cervical cord injury, vascular injury, laryngeal and tracheal injury.

Complications

The main complications in untreated cases are related to the individual structure injured. Injuries to the larynx and trachea may produce acute respiratory obstruction or late stenosis and sepsis. Oesophageal injuries may produce cervicomediastinal sepsis. Carotid artery injury may cause death from haemorrhage or brain damage. Major venous injuries may produce exsanguination, air embolism and arteriovenous fistula. Cervical fracture may result in paraplegia, quadriplegia or death.

Treatment

EMERGENCY OR FIRST AID

1. Clear the airway.
2. Control haemorrhage.
3. Treat shock.

DEFINITIVE TREATMENT

The general principles are as follows:

1. All penetrating wounds should be thoroughly explored.
2. A neurological deficit that is not clearly due to head injury is usually due to ischaemic brain infarction. Arterial reconstruction will worsen the condition by producing haemorrhagic infarction. In such cases the carotid artery should be ligated.
3. Blunt trauma of the neck producing contusion, haematoma, tracheal compression and respiratory insufficiency may necessitate tracheostomy.

Blood vessel injury

1. The vertebral artery should be ligated whenever injured, because it causes massive blood loss with high mortality. Ligation should be performed even though there is a 1.5% risk of fatal midbrain or cerebellar necrosis.
2. The subclavian artery is better explored by a cervicothoracic exposure; ligation of the artery is relatively safe, but repair is preferable.
3. Venous injuries are best ligated with the patient's head lowered throughout to avoid air embolism.

Laryngeal and tracheal injuries

Minor injuries need no treatment, but immediate tracheostomy should be considered when airway obstruction exists. Conveniently located small perforations of the trachea can be utilized for tracheostomy. Otherwise, the wounds can be closed after debridement, and a distal tracheostomy performed. Extensive circumferential tracheal injuries may require resection and anastomosis or reconstruction using synthetic material.

Nerve injury

Primary repair of nerves should be attempted.

Cervical spine and cervical cord injury

See Chapter 9.

2.3.2 FACIAL INJURIES

Importance of facial injuries

Soft-tissue injuries are usually associated with profuse bleeding, due to the abundant blood supply of the face. If not sutured properly, a wound may leave permanent ugly scars. Abrasions which occur as a result of road accidents may be contaminated with grit and dirt. If not thoroughly cleaned they will result in tattoo-like marks.

Injury to important structures such as the eyelids, eyeball, facial nerves and parotid duct is not uncommon.

Fractures of the facial skeleton, if not properly treated, may result in permanent deformities of the facial contour and disturbance in the normal system of dental occlusion.

Types of facial injury

Two main types of injury are recognized: injuries to the soft tissue and fractures of facial bones. These may occur as a result of everyday accidents or of extraordinary events. Everyday accidents typically produce mild injuries that consist of simple soft-tissue wounds and/or fractures of one or more of the facial bones. These injuries do not endanger the patient's life and there is no actual loss of either soft tissue or bone.

Extraordinary events, such as armed conflict and severe road traffic accidents, often result in severe laceration of the facial soft tissues and comminution of the facial bones. Such injuries may result in the loss of a major portion of the facial soft tissue or bones, or both. These injuries can endanger the patient's life and cause immediate death as a result of: obstruction of the respiratory passages; massive haemorrhage from a major vessel; major neurosurgical trauma; aspiration pneumonia. For emergency treatment of such injuries, refer to the section on Wounds, earlier in this chapter.

Management

Mild facial injuries are treated according to the same principles as wounds elsewhere in the body, with the following variations:

1. Exploration and debridement of the wound is essential, but this must be very conservative because the tissues of the face are very precious and the blood supply is adequate.
2. Early and primary suturing in layers of soft-tissue wounds must be performed in order to prevent infection.
3. Immediate reduction and fixation of the fractured bones should be performed.

TREATMENT OF SOFT-TISSUE INJURIES

1. Wounds to the face should be prepared by thorough washing with chlorhexidine solution 1.3%, and then irrigated with large amounts of sterile saline solution to wash away foreign bodies. The wounds should be debrided conservatively and closed primarily.

2. Larger wounds should be debrided and closed as quickly as possible.
3. Massive soft-tissue defects involving the mouth or nose should be debrided and the skin sutured to the mucosa to obtain closure and primary healing. Parotid duct injuries should be searched for and repaired over plastic cannulas. Wounds to the tongue should be debrided and closed with through silk sutures.
4. Eyelid injuries should be treated primarily with careful debridement and primary suturing, so as to prevent deformities. Local flaps and skin grafts should be used for reconstruction.
5. Wounds to the nose should be closed primarily when possible, but occasionally local flaps and ear-composite grafts are used to reconstruct a severely damaged nose later on.
6. Ear injuries should be repaired primarily whenever possible.
7. Facial burns should be treated by the open technique. The burns should be washed gently with Savlon solution and loose tissue debrided. A clean dry crust is then allowed to form over the burned area. Occasionally hot saline dressings to the face, or antibiotic ointment such as neomycin or oxytetracycline, are used in specific cases when there is evidence of infection; for deep burns skin grafting of the eyelids has to be performed to avoid healing by scarring and ectropion.

INJURIES INVOLVING THE FACIAL SKELETON

1. Wounds should be copiously irrigated and foreign material removed.
2. Bony fragments with periosteal attachment should be carefully preserved.
3. Fractured teeth that are devitalized should be extracted. Teeth in the line of fracture should be removed unless they are essential for reduction and fixation.
4. Fractures should be reduced and fixation by transosseous wiring performed. Occasionally a segment of devitalized bone is used to maintain the mandibular or orbital arch by interosseous wiring when soft-tissue coverage is possible. Bone grafts should not be performed at the acute stage, but only after reduction and fixation.
5. A watertight closure of the oral mucosa should be performed and an external dressing applied. Drains should be inserted, to be removed in 24–48 hours.

6. All patients should be placed on antibiotic therapy. Daily oral hygiene and saline irrigations should be carried out.

Mandibular fractures
Simple fractures of the mandible are treated by closed reduction with arch bar intermaxillary elastic fixation. Complicated or open fractures are treated by open reduction and interosseous wire fixation. In cases where there is massive loss of mandibular substance, arch formation and adequate positioning of remaining mandibular segments occasionally requires the use of Kirschner wires, tantalum splints, acrylic and arch bars to maintain proper alignment.

Midface fractures
Fractures of the maxillae are frequently associated with multiple and serious injuries of the facial bones. Zygomatic, maxillary and orbital rim fractures are treated by open reduction and interosseous wiring. Occasionally loss of bone is so great that continuity cannot be restored primarily, and soft-tissue coverage is carried out in the realization that reconstructive work will be needed in the area at a later date.

Emergency management of maxillofacial injuries

FIRST-AID

1. Ensure preservation of a satisfactory airway.
2. Control haemorrhage.
3. Ensure the patient is transported to hospital rapidly for primary care and disposition.

IMMEDIATE RESUSCITATION

1. Ensure a clear airway
2. Control haemorrhage.
3. Start intravenous fluids.
4. Take blood samples for typing and cross-matching.
5. Cut away clothing.
6. Remove dressings applied in the field.
7. Perform cutdown if necessary.
8. Give tetanus toxoid.
9. Obtain a brief history and record physical examination.

2.3.3 CUT THROAT

A cut throat may be :

- Accidental

- Homicidal (the most serious, usually transverse)
- Suicidal (the most common, usually oblique)

If the carotid and jugular veins are involved, rapid fatal haemorrhage will ensue. The skin only may be affected or the larynx and oesophagus may be injured.

2.3.4 EYE INJURIES

With severe eye injuries it is important for the non-specialist to avoid causing further damage by unnecessary manipulation, and to refer the patient immediately to an ophthalmologist. An injured eye has no defence against infection and any local anaesthetics, dyes or other medication used must be sterile. Examinations should be made with a well-focused light and with a magnifying glass or loupe if necessary.

Foreign bodies

Superficial metallic foreign bodies in the cornea or conjunctiva are common. Penetrating ocular foreign bodies are rare, and there is almost always a history of welding. X-rays are always indicated if an intraocular foreign body is suspected. Intraocular and deeply embedded foreign bodies should be removed by an ophthalmologist; superficial corneal foreign bodies may be safely removed by a general practitioner.

CONJUNCTIVAL FOREIGN BODIES

The only important conjunctival foreign bodies are those that lodge beneath the upper tarsal conjunctiva and rub on the cornea, causing extreme pain. Foreign bodies on the lower tarsal and palpebral conjunctiva seldom cause pain, and are usually washed away by the action of tears and blinking. A foreign body can be removed as follows:

1. Evert the lid, if necessary by pressing gently downward across its outer surface with a match or applicator stick while pulling upward on the eyelashes.
2. Remove free particles with a moist cotton-tipped applicator, or by gentle irrigation with warm sterile saline solution.

CORNEAL FOREIGN BODIES

1. Instil two drops of sterile 0.5% tetracaine solution or other sterile anaesthetic into the conjunctival sac.

2. Remove superficial particles with a moist cotton-tipped applicator.
3. Remove embedded particles with a delicate instrument, preferably a special 'hockey stick' sput. Instil an antibacterial ointment, e.g. polymyxin B, after the foreign body has been removed.

Most patients are more comfortable without a patch on the eye after removal of a corneal foreign body. It is essential to see the patient the next day to be certain that no infection has occurred, and that healing is under way.

Corneal abrasions

Corneal abrasion is a common injury which must be treated promptly and with great care in order to speed healing and to prevent infection and recurrent corneal erosions.

The history should be taken and visual acuity tested before treatment. A patient with a corneal abrasion complains of severe pain, especially with movement of the lid over the cornea. The surface of the cornea may be examined with a light and loupe. If an abrasion is suspected but cannot be seen, stain the cornea with sterile fluorescein. The area of corneal abrasion will have a deeper green stain than the surrounding cornea.

Instil polymyxin B and bacitracin ophthalmic ointment. Apply a tight pressure bandage to prevent movement of the lid and resultant irritation of the abraded corneal area. Bed rest may be necessary. The patient should be observed on the following day to be certain that the cornea is healing. Corneal abrasions heal in 24–72 hours if a pressure bandage is properly applied. In contrast to corneal foreign body wounds, there is little chance of infection. The main dangers are delayed healing and recurrent corneal erosion due to imperfect healing.

Burns to the eye

THERMAL

Thermal burns are usually associated with face or body burns. If the conjunctiva is involved adhesions may develop. If the cornea is severely damaged, perforation or infection is apt to occur and an ophthalmologist should be consulted.

1. Dilate the pupil with sterile 5% homatropine solution.

2. Instil an antibiotic ointment frequently.
3. Relieve pain with cold compresses and systemic analgesics or narcotics.
4. Keep the eye(s) closed.

ULTRAVIOLET

Ultraviolet burns may produce a superficial painful keratitis, which usually heals without complications within 24–48 hours.

1. Relieve pain with an initial instillation of sterile 0.5% tetracaine solution and subsequently with systemic analgesics or narcotics.
2. Apply cold compresses until the initial reaction subsides.
3. Keep the eyes bandaged.

In severe cases it may be necessary to instil corticosteroid drops locally to relieve inflammation and associated pain.

CHEMICAL

In cases of chemical burns, flush out the conjunctival sac immediately with tap water for several minutes. Neutralizing solutions are unnecessary and may even be harmful. If fluorescein stain after irrigation indicates that corneal damage has occurred, treat as for thermal burn and refer to an opthalmologist for definitive care.

Contusions of the eyeball

If ophthalmoscopic examination shows any evidence of internal haemorrhage or damage to the iris, lens or retina, specialist care is indicated. Any injury severe enough to produce anterior chamber haemorrhage (e.g. hyphaema) may cause secondary haemorrhage and intractable glaucoma.

Lacerations of the eyeball

Minor lacerations of the conjunctiva may be left alone or closed by fine silk sutures. Lacerations of the cornea or sclera require specialist care, since they may lead to blindness or loss of the globe itself. Emergency and temporary measures include dilatation of the pupil with sterile 5% homatropine solution, instillation of an ophthalmic antibiotic ointment three times daily, systemic antibiotics, analgesics or narcotics, and bilateral eye bandages. If the wound is extensive and loss of contents has been great enough to

preclude function, enucleation may be indicated. If uveal tissue has been injured and the eye is retained, sympathetic ophthalmia may occur 2 weeks to several years after the initial injury. Fortunately, this complication is rare.

2.4 HAEMORRHAGE

TYPE

Haemorrhages may be classified according to the origin of bleeding, site of origin, time of occurrence or aetiology.

ORIGIN OF BLEEDING

Arterial

Arterial bleeding is bright red in colour and occurs as forcible ejections synchronous with the heart beat. The larger the injured artery, the more powerful the ejection and the more profuse the bleeding.

Venous

Venous bleeding is dark blue-purple in colour and the blood flows in a continuous stream from the injured vessel.

Capillary

Where no large vessels are involved and there is capillary bleeding, there is a steady ooze of dark blood from the cut tissue.

SITE OF BLEEDING

External haemorrhage

External haemorrhage occurs either from a wound to the external body surface or from bleeding into the natural body passages which lead to the exterior, e.g.:

- Haemoptysis: the coughing of blood which is fresh, red and frothy because it is mixed with air and contains sputum.
- Haematemesis: vomiting of blood which is dark in colour with clots, and which may contain food particles and acid gastric juice.
- Bleeding per rectum: may consist of:
  fresh red blood which arises from the lower part of the gastro-intestinal tract, e.g. in piles, fissure, cancer of the rectum;

dark, tarry stools (melaena) from bleeding in the upper part of the gastrointestinal tract, e.g. in duodenal ulcer;
blood and mucus, e.g. in amoebic or bacillary dysentery.

- Epistaxis: bleeding from the nose.
- Haematuria: blood in the urine.

Internal haemorrhage
Internal haemorrhage is concealed (hidden) bleeding occurring in the body cavities, e.g.:

- Haemopericardium: blood in the pericardial sac around the heart.
- Haemothorax: blood in the pleural cavity.
- Haemoperitoneum: blood in the peritoneal cavity.
- Retroperitoneal haemorrhage and pelvic haematocele.
- Haemoarthrosis: blood in the joint cavity.
- Intracranial haemorrhage: bleeding inside the skull.

TIME OF OCCURRENCE

Primary haemorrhage
This may occur during an operation or at the time of an accident, as a result of injury to a blood vessel (artery, vein or capillary).

Reactionary haemorrhage
This occurs in the first 48 hours after a trauma or operation. It is due to the dislodgement of a clot, slipping of a ligature or untying of a suture.

Secondary haemorrhage
This usually occurs 7–10 days after injury or operation. It is due to infection eroding the ligatures or the vessel wall, which gives way under the effect of blood pressure. There is always a minor warning bleeding 24 hours before the actual bleeding, that may be as little as a bloody, stained ooze.

AETIOLOGICAL CLASSIFICATION

- Traumatic haemorrhage: (external or operative).
- Pathological haemorrage: due to the involvement of a vessel in an ulcer or malignant tumour.

- Spontaneous haemorrhage: usually occurs in conditions associated with an abnormal tendency to bleeding, e.g. thrombocytopenic purpura.

Mechanism of arrest of bleeding

1. Contraction of the muscular and elastic coats of damaged vessels and rolling of the internal endothelium with thrombus formation.
2. Clotting of extravasated blood.
3. Fall in blood pressure following loss of blood.

Management

Place the patient in the supine position unless this is contraindicated, to minimize fainting and to facilitate examination. Expose all bleeding sites before trying to control haemorrhage. Do not use a tourniquet unless other methods have failed.

Venous and minor arterial bleeding can be controlled by direct pressure on the wound with a sterile dressing or a clean cloth, and elevation of the part.

For major arterial bleeding, the following steps should be tried, in order, one after the other:

LOCAL MEASURES

1. Make a pressure dressing with a pad and bandage.
2. Elevate the injured part.
3. Apply direct pressure on the main artery.
4. Apply styptics, e.g. ice to produce spasm, and give coagulants.
5. Apply haemostatics topically.
6. Place a tourniquet between the wound and the heart.

Emergency tourniquet should be resisted. If it is lightly applied it increases bleeding and the patient may die from haemorrhage. If it is tight, the patient may die of shock and the crush syndrome, or gangrene may occur. Haemorrhage should be arrested by application of a thick pad bandaged firmly over the wound. Haemorrhage from a completely severed artery will stop quickly of its own accord as a result of reflex spasms. If a tourniquet must be applied, the time of application should be written down so that it is not forgotten. It should be released every 30 minutes for 5 minutes to

wash out the tissues. If, for any reason, a tourniquet is left in position for 6 hours or more, it should be released and the limb should be amputated at once at a more proximal level to prevent the crush syndrome.

GENERAL MEASURES

1. Restore the blood volume by blood transfusion or other fluids where necessary (glucose, saline, plasma, blood).
2. Give sedatives after certain diagnosis (barbiturates, pethidine, morphine).
3. Give vasopressor drugs, e.g. epinephrine and noradrenaline.
4. Give oxygen therapy.
5. Transfer the patient to hospital as quickly as possible for further treatment.

In certain areas of the body (head and neck, shoulder and axilla, groin and hip), it is not possible to apply a tourniquet or direct pressure to control haemorrhage. Bleeding should be controlled by applying pressure over the major arteries supplying the bleeding area.

Common carotid artery

1. Palpate the trachea and the thyroid cartilage in the middle part of the neck.
2. Move the fingers lateral to the thyroid cartilage, and feel for the arterial pulsation.
3. Apply pressure downwards towards the back of the neck with the first three fingers.

Brachial artery

1. Move the patient's arm at a right-angle to the body, and rotate it externally so that the palm of the hand faces upward.
2. In the midportion of the arm between the axilla and elbow, there is a groove bounded by the biceps muscle and the bone. Compress the brachial artery against the bone in this groove.

Femoral artery

1. Palpate the femoral artery in the upper part of the thigh about 2.5 cm below the inguinal ligament.
2. Compress the femoral artery between the hand and the pelvic bone.

EPISTAXIS

Sit the patient up and forward to prevent aspiration or inhalation of blood.

Anterior epistaxis

1. Apply pressure over the area (pinching the nose) for 5 minutes. This is usually sufficient to stop bleeding. It may be combined with packing of the bleeding nostril with cotton moistened with saline solution or 1:1000 epinephrine solution.
2. After active bleeding has ceased, or if pressure fails to stop the bleeding, a cotton pledget soaked with 2% lidocaine should be applied to the bleeding area and the bleeding vessel cauterized with chromic acid, a silver nitrate bead or an electrocautery needle. Chromic acid must be neutralized immediately with saline solution.
3. If the source of bleeding is far back or not controlled, the nasal chamber should be anaesthetized and packed with 2.5 cm selved gauze impregnated with cod-liver oil, or even vaseline. The packing is placed in layers. The pack can be left for 7 days provided antibiotics are given.

Posterior epistaxis

This will need a posterior nasal pack, which requires a specialist.

Uncontrolled epistaxis

This should be referred to hospital, as it necessitates blood transfusion or surgical interference.

Once epistaxis has been arrested, specific measures should be taken to treat the underlying cause as necessary.

3

Burns

Summary of contents

CLASSIFICATION OF BURNS 64

Thermal burns 65
Chemical burns 69
Electrical burns 71
Sunburn 72

A burn is the destruction of body tissues by heat, which may be dry or moist. A burn may also be caused by chemical substances (acids or alkalis), by physical agents (such as deep X-ray or electricity) or by thermal weapons (flame thrower, napalm, petroleum or nuclear explosion).

Classification of burns

Dupuytren graded burns according to the degree of destruction involved:

- First degree (50–60 °C): erythema of the skin (redness only)
- Second degree (60–100 °C): redness and vesicle formation
- Third degree: destruction of the superficial layer of skin, with exposure of the nerve endings
- Fourth degree: complete destruction of the skin
- Fifth degree: complete destruction of the skin and muscles
- Sixth degree: complete destruction of the bone

Wallace classified burns into two types:

- Incomplete (including first, second and third degree) with partial skin loss
- Complete or deep (fourth, fifth and sixth degree)

Burns may also be classified according to the percentage of the body surface area destroyed:

- Minor: less than 15–20% of the body surface area in adults, and 10–15% in children
- Major: more than 20% of the body surface area in adults, and more than 15% in children

The 'rule of nine' is used to determine the extent of damage to the body surface area:

Head and neck	9%
Each upper limb	9%
Each lower limb	18%
Front of trunk	18%
Back of trunk	18%
Perineum	1%

First aid

1. Remove the victim from the site of the incident.
2. Roll the victim up in a thick blanket.
3. Apply cold water; this is sufficient to relieve pain in minor burn cases;
4. Do not apply oily substances to the skin.
5. Start fluid therapy when possible.
6. Cover major thermal burns with clean dry dressings and transfer the victim to hospital as quickly as possible.

3.1 THERMAL BURNS

Moderately severe thermal burns can produce:

- loss of water, electrolytes and protein, which may result in hypovolaemia, haemoconcentration and shock;
- hyperkalaemia, secondary to destruction of tissue and red blood cells;
- loss of erythrocytes due to cellular destruction, thrombosis of blood vessels, trapping and haemolysis of red blood cells in burned tissue and late anaemia due to infection, negative nitrogen balance, and bone marrow suppression;
- renal shutdown during the first 24–36 hours, which is usually due to a decrease in blood volume, decrease in cardiac output, and renal vasoconstriction;

- increase in metabolism, which results from increased production of catecholamine and from evaporative heat loss;
- neurogenic shock.

For burns to the eyes, see Chapter 2.

Clinical picture

HISTORY

The causative agent and the possibility of smoke inhalation should be considered, together with any associated injuries. A history of drug allergy, immunization status and any current medication should be ascertained.

PHYSICAL EXAMINATION

1. Assess the status of the airway.
2. Look for circulatory failure.
3. Determine the extent of the burn.
4. Determine the degree of the burn.
5. Determine the associated injuries and patient's weight as soon as possible.

Management

MINOR BURNS

Minor burns include:

- first degree burns;
- second degree burns of less than 10% of the body surface that do not involve the face, hands, feet or genitalia;
- third degree burns of less than 2% of the body surface that do not involve the face, hands, feet or genitalia.

Treatment

Minor burns do not require hospitalization and can be managed on an outpatient basis as follows:

1. Apply cold compresses to burn areas immediately.
2. For **first degree** burns:
   - Topical antibiotics are not required.
   - The burned area may be wrapped in dry dressings for comfort.
   - Sedatives may be required in cases of severe pain.

3. For **second and third** degree burns:
   - Wash with a mild bactericidal agent.
   - Do not rupture the blisters, but loose skin may be removed.
   - Dress the wound with fine mesh gauze impregnated with a topical antibiotic.
   - Immobilize a burned extremity in an elevated position of function to minimize oedema.
   - Change the dressings daily.
4. Give tetanus toxoid.

MODERATE AND SEVERE BURNS

The following cases constitute moderate and severe burns:

- Burns to the head, neck, face, eyes, hands, genitalia, perineum or feet.
- Infants with a burn over more than 5% of the body surface.
- Second and third degree burns over more than 10% of the body surface.
- Children with chemical or electrical burns.
- Children with burns of the tracheobronchial tree.

Treatment

Cases of moderate or severe burns should be hospitalized. Initial care can be given in the emergency room using aseptic techniques.

1. Remove all the patient's clothing and wrap him or her in a clean cotton sheet.
2. Check adequacy of airway and give oxygen.
3. Check temperature, pulse, respiration and blood pressure.
4. Perform endotracheal intubation if there is significant respiratory distress.
5. Consider tracheostomy if intubation is required for more than 3 days.
6. Perform gastric decompression to avoid vomiting and aspiration in burns over more than 20% of the body surface, and if associated injuries are present.
7. Begin fluid therapy. Establish an i.v. line via cutdown with an i.v. catheter; start dextrose 5% in lactated Ringer's solution. Initial fluid requirement may be calculated as follows: 150 ml of fluid per 1% of body surface area for the first 24 hours. Give half this amount for maintenance after the first 24 hours. Give half of the estimated first day's fluid

requirement in the first 8 hours after the burn, and give one-fourth in each of the second and third 8-hour periods. Fluid loss replacement should not exceed 15% of the body surface area in the first 24 hours.

8. Give colloid solution after the first 24 hours. Give blood after the first 48 hours, unless blood loss has occurred from associated injuries.
9. For pain, give morphine sulfate, 0.1 mg/kg i.v. diluted in 2–3 ml of normal saline solution over a 3-minute period.
10. Insert a Foley catheter for urine output. Monitor urine output carefully.
11. Give tetanus toxoid.
12. Antibiotics should be given in all cases of second and third degree burns to prevent infection and scarring.

Care of the wound

1. Clean the wound with saline solution. Putting the burn victim in a tub and giving a saline bath removes the crusts and other purulent discharges. The use of hexachlorophene soap to wash large burn areas is no longer recommended.
2. Perform escharotomies (relaxing incisions), particularly during the first 24 hours, because circumferential full-thickness burns of the extremities or trunk may cause constriction as oedema progresses. This may result in vascular compromise to the hands or feet, or even in respiratory distress in the case of burns to the trunk.
3. Begin topical antimicrobial therapy. Continuous topical application of antimicrobial agents is useful in controlling bacterial infection of the wound. The use of silver sulfadiazine and zinc oxide ointments has proved effective.
4. Maintain a bedside progress chart for:

   - patient's temperature every 2 hours, since hypothermia is a frequent complication;
   - monitoring of vital signs, urine output and specific gravity every half hour initially, and hourly thereafter;
   - measuring serum electrolytes, total protein, haematocrit, blood gases, and urinary sodium content and osmolality every 6 hours in major burns;
   - recording intake and output of fluids;
   - patient's daily weight;

- daily chest X-ray – it may take up to 5 days for pulmonary infiltrates indicative of inhalation injury to appear;
- performing burn wound biopsies and obtaining quantitative cultures three times a week.

3.2 CHEMICAL BURNS

3.2.1 ACIDS

Acids produce burns when they come into contact with tissues, depending on the concentration of the acid and the duration of exposure. The fumes of concentrated acids are extremely irritating to the respiratory tract.

Clinical picture

On the skin surface, acids may produce burns similar to thermal burns.

Ingestion of acids produces:

- Corrosion or burn
- Severe burning pain in the gut, followed by vomiting and diarrhoea
- Marked thirst and shock
- Difficulty and pain in swallowing, breathing and speech
- Perforation of the stomach in severe cases
- Possible death from shock and collapse

Management

LOCAL

Wash thoroughly with water or weak alkalis such as sodium bicarbonate or lime water. Treat as for thermal burns.

SYSTEMIC

- Give milk, beaten eggs, or water
- Do **not** perform gastric lavage
- Give a sedative
- Begin supportive therapy
- Initiate steroid therapy
- Give a broad-spectrum antibiotic

3.2.2 ALKALIS

Caustic alkalis produce severe corrosive burns, and their ingestion requires vigorous and immediate treatment. When alkalis come into contact with skin or mucous membrane they produce burns or perforations. These deep burns heal with scarring.

Clinical picture

LOCAL MANIFESTATIONS DUE TO CONTACT

Skin: first, second or third degree burns, depending on the type of alkali and the duration of contact.
Eye: conjunctival and corneal ulceration.

SYMPTOMS DUE TO INGESTION

- Burning sensation in the gut, with difficulty in swallowing, and vomiting
- Possible respiratory difficulty and asphyxia
- Shock
- Perforation of the oesophagus

Management

LOCAL

For skin burns caused by an alkali, clean the skin with water or saline solution until the soapiness disappears.

For eye injuries, irrigate with water or saline solution for 5 minutes. Instil a local anaesthetic, and continue irrigation with water or normal saline solution for half an hour. Check for any injury to the cornea; if present, cover with an eye patch. Consult an ophthalmologist for specific management.

INGESTION

If alkalis are ingested, hospitalize the patient at once. In the meantime:

- Do **not** perform gastric lavage or give emetics
- Give diluted vinegar, lemon juice or orange juice to neutralize the alkali and olive oil to ease the pain.

In the hospital:

- Oesophagoscopy should be performed within 48 hours to determine future management.

- Give steroids to prevent oesophageal strictures.
- Give a broad-spectrum antibiotic.
- Bougienage may be started on the fourth day.

3.3 ELECTRICAL BURNS

The appearance of electrical burns may be deceptive: they may appear to be superficial, but may cause death because of the secondary necrosis of blood vessels and haemorrhage that usually occurs more distant to the apparent area of injury.

Electrical injury may be produced in two ways, either by arcing or a flash from short circuiting, or by direct electric current. If there is unusually secure ground contact and electric current is unusually prolonged, burns are produced at the site of contact. As the current passes through the body, various tissues or organs are affected:

- Blood vessels (thrombosis may occur)
- Brain (respiratory arrest may occur)
- Heart (ventricular fibrillation may occur)

Clinical picture

GENERAL MANIFESTATIONS

- Immediately after a severe electric shock, patients are usually comatose, apnoeic and in circulatory collapse due to ventricular fibrillation or cardiac arrest.
- Hypovolaemic shock appears in cases of high-tension electrical injury.
- Bone fractures may occur, either due to falls at the time of the accident or from convulsive muscular contractures.

LOCAL MANIFESTATIONS

- Thermal burn from arc or flash
- Coagulation, necrosis and haemorrhage
- Full-thickness burn and/or deep-tissue injury along the path of conduction
- Ventricular fibrillation
- Cerebral oedema

Management

At the accident site, the electric current should be switched off and

the patient removed from the site with a non-conducting material such as a wooden pole or a rubber sheet. Vital signs should be monitored and cardiopulmonary resuscitation performed if required.

CARE OF THE WOUND

Small electrical burns of the extremities can be treated on an outpatient basis.

Mouth burns

- Provide adequate hydration.
- Give systemic antibiotics.
- Antimicrobial ointment may be applied locally.
- Arrange for feeding by nasogastric tube if necessary.

Major electrical burns

- Early debridement of non-viable tissue is essential.
- Use a topical antibacterial ointment on the debrided area, and re-examine wounds daily for evidence of subsequent necrosis.
- Repeated debridement may be necessary to remove all necrotic tissue; the area is then covered by split-thickness or full-thickness grafts (tangential excision).

GENERAL MANAGEMENT

- Give tetanus toxoid 0.5 ml i.m. to previously immunized patients and human tetanus immunoglobulin (Hyper-Tet), 100–250 iu i.m., to those who have not been immunized previously.
- Fluid and electrolyte losses are not significant in electrical burns. However, the patient must be monitored carefully for the development of shock, respiratory failure or cardiac arrest.
- In patients with major electrical burns, watch for the development of acute renal tubular necrosis; this occurs more frequently in electrical burns than thermal burns.

3.4 SUNBURN

Sunburn results from the overexposure of depigmented or susceptible skin to sunlight (ultraviolet radiation). It occurs more frequently in fairskinned people, and in albinism, vitiligo and leucoderma. Symptoms usually begin 2–4 hours after exposure to

the sun, and reach a peak 14–48 hours after exposure. This may be local or systemic, depending on the intensity of the sun's rays and duration of exposure.

LOCAL MANIFESTATIONS

- First and second degree burns
- Possible pain, tenderness and swelling (with severe exposure)
- Tanning
- Desquamation and sloughing of the skin (during the recovery phase)

SYSTEMIC MANIFESTATIONS

These usually occur with a severe reaction: fever, headache, malaise, nausea and vomiting, and shock.

Mild sunburn

Treatment includes:

- Cold compress
- Topical application of ice
- Analgesics
- Topical application of bath oil for lubrication.

Severe sunburn

Hospitalize the patient for correction of fluid and electrolyte losses and for treatment of systemic manifestations. Treatment includes:

- Cold compress in tepid water
- Topical application of soothing lotions and creams
- Topical application of corticosteroid lotions, creams or spray in low strength
- Analgesics
- Adequate hydration
- Rest to the affected part.

4

Respiratory emergencies

Summary of contents

INFECTIOUS AIRWAY OBSTRUCTION 74

Ludwig's angina 75
Acute infectious epiglottitis 75

RESPIRATORY FAILURE 76

PULMONARY OEDEMA 84

STATUS ASTHMATICUS 85

HAEMOPTYSIS 88

CARDIOTHORACIC INJURIES 90

Thoracic injuries 90
Fractured rib 90
Flail chest 91
Closed pneumothorax 93
Open pneumothorax 93
Tension pneumothorax 94
Haemothorax 96
Rupture of the airways 98
Cardiovascular injuries 99
Cardiac tamponade 100
Contusion of the heart 100
Intracardiac rupture 100
Rupture of the aorta 101

4.1 INFECTIOUS AIRWAY OBSTRUCTION

The anatomy of the upper airway makes it vulnerable to obstruction, which may be produced by trauma, tumour, foreign body,

laryngospasm occurring with tetanus or hypocalcaemic tetany, soft-tissue haemorrhage, and oedema resulting from allergic reactions. Infectious processes resulting in airway obstruction usually involve the oropharynx, not the glottis.

4.1.1 LUDWIG'S ANGINA

Ludwig's angina is a cellulitis of the submaxillary space of the submandibular area. The submaxillary space is formed by the deep cervical fascia, the myelohyoid muscle, and the hyoid bone. Infection in the submaxillary space may be the result of oral lacerations or mandibular fractures, but most commonly it occurs with infections in the second and third molars. These molars are situated in potential communication with the submaxillary space, providing an accessible route for periapical or periodontal abscesses to reach the submaxillary space. Occasionally, infections of the submaxillary gland or suppuration from the submaxillary nodes may empty into the submaxillary space.

Clinical picture

Characteristically, the infection takes the form of cellulitis, beginning with unilateral involvement but soon spreading contralaterally into the neck and eventually entering the sublingual space as well, leading to oedema of the floor of the mouth, which pushes the tongue posteriorly, and cervical swelling (the familiar bull neck), with respiratory distress and dysphagia. Airway obstruction is the actual threat to the patient with Ludwig's angina.

Management

Prophylaxis is the most important treatment: patients with abscesses associated with disease in the posterior molars should receive antibiotic therapy as well as incisions for surgical drainage. Soft-tissue X-ray can provide an indication of the degree of tracheal involvement. Early elective tracheostomy is the treatment of choice in patients with full-blown Ludwig's angina. Broad antibiotic coverage should be given initially until culture results are available.

4.1.2 ACUTE INFECTIOUS EPIGLOTTITIS

Acute infectious epiglottitis is common in children, in whom it may be fatal. It may be produced by a variety of Gram-positive organisms, but the most common offender is *Haemophilus influenzae* type B.

Symptoms

The symptoms of acute infectious epiglottitis are chills, dysphagia, sore throat and choking, followed by respiratory difficulty or stridor. Hoarseness is not present. Fever, often high, is common and the patient may appear toxic.

Physical examination

- There is little evidence of pharyngitis, in contrast to the patient's symptoms.
- Occasionally, there may be cervical swelling or adenopathy.
- Respiratory obstruction is indicated by dyspnoea, cyanosis, stridor, flaring alae, accessory respiratory muscle activity, and suprasternal and intercostal retraction. Respiratory arrest follows within hours.
- Indirect laryngoscopy shows oedema and inflammation of the epiglottic and supraglottic tissues.
- Leucocytosis is usual, and bacteraemia is common.

Diagnosis

- Indirect mirror laryngoscopy
- Lateral neck X-ray

Treatment

1. Give oxygen.
2. Give hydrocortisone i.v.
3. Ensure high humidity.
4. Install endotracheal intubation if respiratory obstruction is severe with cyanosis.
5. Administer antibiotic therapy that covers both Gram-positive and *H. influenzae* organisms.
6. Transfer to hospital.

4.2 RESPIRATORY FAILURE

Diagnosis

Respiratory failure is defined in terms of altered blood gases – an arterial oxygen tension (Pao_2) of less than 8 kPa (60 mmHg) with or without an arterial carbon dioxide tension ($Paco_2$) of above 6.7 kPa (50 mmHg).

Both hypoxia (low Pao_2) and hypercarbia (raised $Paco_2$) are difficult to pick up clinically, for the following reasons:

- The classic signs of hypoxia are either non-specific (disturbances of consciousness, ranging from mild confusion to coma) or difficult to assess (cyanosis).
- Hypercarbia may give rise to a spectrum of mental changes similar to those of hypoxia. It may also cause a flapping tremor, peripheral vasodilation, papilloedema and early morning headaches, which again are not specific to a rising $PaCO_2$.

Arterial blood gas measurements are therefore mandatory if the diagnosis is suspected.

There are three patterns for respiratory failure:

- Pure ventilatory failure
- Hypoxaemic failure
- A mixture of ventilatory and hypoxaemic failure

4.2.1 PURE VENTILATORY FAILURE

Pure ventilatory failure gives rise to a raised $PaCO_2$ and a low PaO_2. Examples of this are:

- Depression of the respiratory centre by drugs
- Neurological conditions such as poliomyelitis, myasthenia gravis, acute infective polyneuritis (Guillain–Barré syndrome)
- Primary alveolar hypoventilation (Pickwickian syndrome)

There are occasions when the underlying problem is rapidly reversible, e.g. by administering naloxone to persons with opiate-induced respiratory depression. If no such specific therapy is available, the initial decision is when to institute artificial respiration. To make such a decision the appropriate measurements must be taken as follows:

1. Minute volume (measured with a Wright spirometer). If this is over 4 l/min the patient is very unlikely to require artificial ventilation.
2. Vital capacity (measured with a portable bedside vitalograph). If the vital capacity remains about 1.5 l, artificial ventilation will probably be unnecessary. The vital capacity should be measured at least daily in patients with progressive neurological lesions.
3. Blood gases. These should be measured if there is any doubt about the patient's respiratory status. If the $PaCO_2$ is raised, artificial ventilation should be instituted.
4. Physiotherapy should be given routinely to help prevent sputum retention and infection. In unconscious patients without a

gag reflex, or patients whose disease affects swallowing as well as breathing, inhalation of secretion or vomit must be prevented by installing an endotracheal tube. All this should be done in conjunction with the anaesthetist and chest physician.

4.2.2 HYPOXAEMIC FAILURE

Hypoxaemic failure is due to local disturbances of the ventilation/perfusion relationship. This gives rise to a low Pao_2, with a low or normal $Paco_2$. Examples of this are:

- 'Pure' emphysema
- Asthma in the initial stages of an attack
- Pneumonia
- Left ventricular failure
- Fibrosing alveolitis
- Adult respiratory distress syndrome (shock lung).

Treatment of the underlying disease should, of course, be initiated.

Oxygen may be given by means of a partial rebreathing mask (Fig. 4.1) which delivers a concentration of 50–60%, if the oxygen flow rate is 6 l/min. The reservoir bag fills with oxygen during expiration, so that high concentrations of oxygen are available for the subsequent inspiration. It is termed a partial rebreathing mask because the initial part of exhaled air containing some carbon dioxide and water vapour also enters the reservoir bag, to be rebreathed with the next inspiration. However, take care not to raise the Pao_2 too high (above 13.3 kPa, 100 mmHg) because high oxygen concentrations can be damaging to lung tissue.

Artificial ventilation should be resorted to if the Pao_2 cannot be maintained above about 6.7 kPa, (50 mmHg), or if the effort of breathing is becoming intolerable.

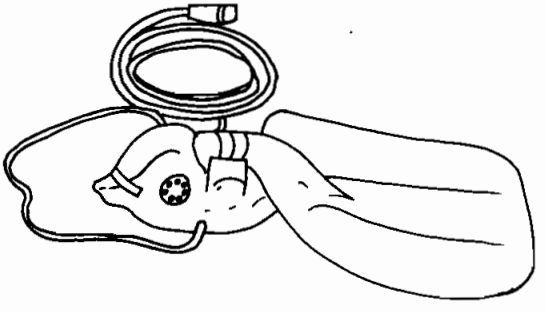


Fig. 4.1 Partial rebreathing mask.

4.2.3 MIXED VENTILATORY AND HYPOXAEMIC FAILURE

The combination of alveolar hypoventilation and a deranged ventilation/perfusion relationship produces a low Pao_2, with a raised $Paco_2$. The example of this type of failure is chronic bronchitis with emphysema. Such patients frequently have a permanently low Pao_2 and may have a permanently high $Paco_2$ (and therefore a high serum bicarbonate [HCO_3]. However, if, with a raised $Paco_2$, the serum HCO_3 is relatively normal (below 30 mmol/l), and the pH is therefore low, the implications are that renal compensation has not occurred, and the respiratory failure has come on over a short time. This further implies that there are reversible elements, such as an acute infection with associated sputum retention, increasing airway obstruction and, often, heart failure. This is the commonest clinical setting for respiratory failure.

Acute-on-chronic bronchitis precipitating respiratory failure

DIAGNOSIS

- The patient often has a history of increasing breathlessness, increasing volumes of purulent sputum and, occasionally, pleuritic pain. All this is in the setting of chronic obstructive airways disease.
- Examination reveals a breathless, often pyrexial patient, who may be confused, cyanosed and have a tachycardia.
- There may be evidence of hypercarbia.
- There will be a prolonged expiratory phase, with variable crackles and wheezes.
- Signs of collapse, consolidation, effusion or a pneumothorax must also be sought, as any of these can exacerbate the situation.
- Signs of right-sided heart failure (raised neck veins, oedema and a palpable liver) are often present.

MANAGEMENT

Initial investigations, in order of priority, are as follows:

- Arterial blood gases and pH
- Chest X-ray, most importantly to exclude a pneumothorax
- Culture of sputum and blood
- Knowledge of haemoglobin electrolytes and urea, although not often immediately useful, will be required.

The aim of management is to increase intracellular oxygen. The

Pa_{O_2} should be increased to at least 6 kPa (45 mmHg) and preferably to 6.7–7.3 kPa (50–55 mmHg), achieved preferably with a fall, or at least without substantial rise, in Pa_{CO_2}. If the intracellular oxygen status is not corrected, ventilatory and hypoxaemic failure will occur.

Infection

The commonest infection organisms are *Streptococcus pneumoniae* and *Haemophilus influenzae*. Both are usually sensitive to amoxycillin 250 mg 8-hourly (parenterally or orally), tetracycline 500 mg 6-hourly (i.m. or orally, not i.v.). If the infection has been contracted in hospital, or if for other reasons it is suspected that the infection may be caused by resistant staphylococci, add i.v. or i.m. flucloxacillin 500 mg 6-hourly.

If the sputum purulence has not decreased after 48 hours, consider changing the antibiotic, but consult the bacteriologist first. Remember that sputum culture and sensitivity tests may be misleading, so do not change antibiotics exclusively on the basis of information from these.

Sputum retention

A patient's outlook may be transformed if energetic and regular physiotherapy loosens the sputum. Initially physiotherapy must be given 2-hourly throughout the 24 hours. If necessary, teach both day and night nurses how to give appropriate physiotherapy. The sputum should be loosened by clapping the chest for 3–4 minutes, after which the patient should take a few quick deep breaths and then cough. Ideally this should be done in appropriate bronchial drainage positions (postural drainage). This is rarely feasible, but at least place the patient first on one side and then on the other. If the patient is too confused to cooperate, give physiotherapy after nikethamide or doxapram. The sputum may be sticky; intermittent humidification through a nebulizer can aid expectoration. If, despite these measures, the patient still cannot bring up sputum, it must be sucked up by nasotracheal suction, bronchoscopy or tracheal toilet.

Nasotracheal suction

Sit the patient up. With a gloved hand pass a soft catheter with a round end (to prevent traumatization of the pharynx and trachea) through a nostril and into the pharynx. A convenient arrangement for this is to attach the catheter to one limb of a Y connector, which is itself attached to a sucker. Suction is then applied by occluding

the other limb. To be of maximum benefit, the catheter must pass between the cords. Encourage the patient to cough and, as he or she exhales, advance the catheter, applying suction.

If the patient cannot talk the catheter is probably through the cords. Advance the catheter into each main bronchus in turn. This is a potent stimulus to coughing and the catheter should be left down until more sputum is forthcoming. If laryngospasm occurs, attempts to pass the catheter into the trachea should not be repeated.

Bronchoscopy
This should be undertaken if the patient continues to deteriorate despite nasotracheal suction, especially if the sputum retention produces lobar collapse. Flexible bronchoscopes have made this a much less traumatic event than previously and, given the circumstances, the patient's memory of the event is hazy.

Tracheal toilet
This is carried out by means of an endotracheal tube.

Airway obstruction
The reversible component may be due to sputum retention, mucosal inflammation or bronchospasm. The treatment of sputum retention and mucosal inflammation has been discussed. Bronchospasm must be assumed to be present and must be treated.

1. Give salbutamol (5 ml mixed with 3 ml saline) by nebulizer or intermittent positive pressure respiration (IPPR ventilator) over 3 minutes up to four times each day.
2. If salbutamol fails to give relief, give aminophylline i.v. 5 mg/kg initially and then 0.5 mg/kg/h thereafter. As well as being a bronchodilator, aminophylline may increase the force of diaphragmatic contraction. The dose may need adjusting in old or very ill patients.
3. Steroids may also be given (e.g. hydrocortisone 200 mg 4-hourly) although the efficacy of these is arguable.

Oxygen therapy
Oxygen should be given in sufficient concentration to raise the Pa_{O_2} to at least 6.0 kPa (45 mmHg), and preferably 7.3 kPa (55 mmHg). To aim higher than this is unnecessary and, in view of the potential danger of oxygen therapy in this type of ventilatory failure, undesirable.

The danger of oxygen therapy arises because patients with a chronically raised $Paco_2$ rely not only on a rise in $Paco_2$, as normal, but on a fall in Pao_2 to stimulate respiration – the so-called hypoxic drive. A sudden rise in Pao_2 may reduce this hypoxic drive, and thus depress ventilation. This causes a further rise in $Paco_2$, and may precipitate carbon dioxide narcosis.

Therefore, after measuring arterial blood gases, start with the 24% oxygen mask. Measure the arterial gases again after 1 hour.

- If the Pao_2 is above 7.3 kPa (55 mmHg) and $Paco_2$ has not gone up by more than 1.3 kPa (10 mmHg), continue using 24% oxygen.
- If the Pao_2 is below 7.3 kPa (55 mmHg) and the $Paco_2$ has not gone up more than 1.3 kPa (10 mmHg), progress to 28% oxygen by Ventimask (4 l/min). Measure the $Paco_2$ again one hour later, and if the situation remains poor progress to 35% oxygen by Ventimask (8 l/min).
- If the $Paco_2$ has risen more than 1.3 kPa (10 mmHg), there is grave danger of inducing carbon dioxide narcosis. Do not increase (or lower) oxygen concentration but intensify all other aspects of treatment, particularly the conjunction of physiotherapy and respiratory stimulants. If the $Paco_2$ goes on rising despite this, the decision as to whether or not to use IPPR must be taken. This can be a difficult decision and depends in particular on the usual respiratory status of the patient. If he or she is a respiratory cripple, then IPPR is unlikely to be of lasting benefit and coming off the ventilation may be difficult.
- Occasionally patients are given high oxygen concentrations by mistake, or out of ignorance. This may lead to the rapid development of carbon dioxide narcosis. It is always best to assume that deterioration in the condition of a patient with ventilatory and hypoxaemic failure is due to carbon dioxide narcosis.

Treatment of carbon dioxide narcosis

1. Do not immediately increase the inspired oxygen concentration.
2. Prevent anybody else from doing so.
3. Measure the blood gases.
4. If the chest signs have changed, repeat the chest X-ray to exclude a pneumothorax or massive pulmonary collapse.
5. Intensify physiotherapy.
6. If the Pao_2 is above 7.3 kPa (55 mmHg) and the $Paco_2$ either above 12 kPa (90 mmHg) or has risen by more than 1.3 kPa (10 mmHg) from the initial reading, reduce the oxygen to 24% by Ventimask, and use a respiratory stimulant.

7. If the Pao_2 is below 4.7 kPa (35 mmHg), as well as $Paco_2$ being high, give a respiratory stimulant without altering the oxygen until the $Paco_2$ has improved.
8. Keep measuring the blood gases; it may be necessary to use IPPR if the condition goes on deteriorating.

Respiratory stimulants
Respiratory stimulants are used to:

- Wake up the patient and help him or her to cooperate
- Counteract carbon dioxide narcosis (as above)
- Counteract respiratory depression.

DO NOT sedate patients in respiratory failure. Always write 'NO NIGHT SEDATION' on their charts, so that no-one else sedates them either!

The best drug to use is doxapram, in a dose of 1.5 mg/min, increasing by 0.5 mg/min at half-hourly intervals if there has been no improvement, to a maximum of 3 mg/min. Ethamivan 5% 2.5 ml i.v. may also be used, as may nikethamide 3–5 ml (0.5–1.25 mg) i.v. repeated half-hourly as necessary.

If respiratory depression is due to opiates, naloxone, which is a specific opiate antagonist, can be used in doses of 0.4 mg given i.v. over 3 minutes. This may be repeated to a total dose of 1.2 mg. As it has a shorter duration of action than the opiates, it may need to be repeated.

Heart failure in association with respiratory failure
The measures outlined above will result in substantial diuresis. However, in the presence of gross or persistent congestive heart failure, the following measures can be employed:

- Diuretics
- Digoxin, particularly if the patient has uncontrolled atrial fibrillation. Patients with respiratory failure have an enhanced sensitivity to digoxin, which is therefore best not used unless there is atrial fibrillation.
- Do not forget that weight is a useful indicator of fluid balance, so weigh the patient daily. In polycythaemia patients, diuresis may cause increased sludging of blood and precipitate thrombosis. This may be prevented by venesection of 3 units of blood, and replacement with an equal amount of Haemaccel or dextran 70. This in itself may be sufficient to improve renal blood flow and initiate a diuresis; this mode of therapy is considered desirable in men with PCV above 54% and in women with PCV above 50%.

4.3 PULMONARY OEDEMA

In pulmonary oedema, the passage of fluid from the vascular space into the interstitial and alveolar spaces results in reduced pulmonary compliance, airway obstruction and impaired gas exchange.

Aetiology

The most common cause of pulmonary oedema is obstruction of pulmonary venous outflow, due to either left ventricular failure or mitral valve disease, but any processes that damage alveolar cells or alter vascular permeability can produce this complication. Such processes include bacterial and viral infections, oxygen toxicity, inhalation of toxic gases, endotoxaemia, drowning, radiation therapy, uraemia, drug reactions (toxic or allergic), transfusion reactions, high-altitude exposure, increased intracranial pressure, seizures, opiate and salicylate intoxication, pulmonary embolism and volume overload.

Pathology

Early on, there is an increase in fluid in the pulmonary interstitial space. In this early stage, there is no auscultatory evidence of pulmonary oedema, and the only symptom may be tachypnoea due to impaired pulmonary compliance.

X-ray signs of interstitial oedema include:

- Kerley's septal lines (long [4 cm], curvilinear and non-branching) representing deep interlobular septa
- Perivascular and peribronchial densities
- Subpleural oedema
- Haziness of the hilar vessels.

Progression may lead to the classic bat's-wing or butterfly pattern of pulmonary oedema. The distribution of the fluid depends not only on the pulmonary anatomy and gravity, but also on underlying diseases, e.g. in neurogenic pulmonary oedema the upper lobes are most frequently involved.

The heart size cannot be relied on as a clue to the cause of pulmonary oedema.

Signs and symptoms

- Cough
- Sputum
- Dyspnoea
- Cyanosis

Prognosis

Pulmonary oedema is a major medical emergency which must be treated promptly. Fluid accumulation in the alveolar spaces is the end result of pulmonary oedema and will lead to bubble formation, resulting in airway obstruction, alterations in compliance and impaired gas exchange, hypoxia, metabolic (lactic) acidosis and carbon dioxide retention.

Management

The patient should be transferred to the hospital intensive care unit immediately. In the meantime, initial therapy should be directed towards improving gas exchange. The improvement should in large part lead to the correction of acid–base disturbances.

1. Give oxygen.
2. If oxygen does not correct hypoxia, or if there is foam formation or carbon dioxide retention, assisted ventilation should be used and even continuous positive pressure ventilation through an endotracheal tube.
3. Apply continuous endotracheal suction.
4. Treat the cause, e.g. sit the patient up; use rotation tourniquets in heart failure, and morphine and digitalis in cardiogenic pulmonary oedema.
5. Phlebotomy often improves the condition when other measures have failed.
6. Antihypertensives may be used cautiously.

4.4 STATUS ASTHMATICUS

Status asthmaticus is a true medical emergency, particularly when it occurs in young children, in whom it can lead to fatal respiratory failure. It results from a progressive worsening of bronchial asthma that does not respond to appropriate dosages of epinephrine hydrochloride. The cause is not clear. The precipitating factors are infection, acidosis, dehydration, sedation and narcotics, the presence

of significant hypoxaemia and failure to administer large doses of steroids in a patient who is steroid-dependent.

In asthmatic patients, the basic alteration of respiratory function is an increase in airway resistance brought about by three principal mechanisms: bronchospasm, mucosal oedema and accumulation of secretions. Other factors that may also contribute to increased airway resistance are thickening of airway walls by inflammatory infiltrates and glandular hyperplasia; intraluminal obstruction by cellular debris and inflammatory exudate; and airway collapse, due to either external compression or internal cohesion.

Varying degrees of airway obstruction and decreased compliance throughout the lungs cause regional differences in ventilation/perfusion ratios. As a result, the carbon dioxide tension rises and the oxygen tension falls in the blood perfusing the afflicted alveoli. If this process is unchecked, the normal compensatory mechanisms fail, resulting in hypoxia and acidosis.

Clinical picture

HISTORY

- Duration and severity of an attack
- Degree of dehydration
- History of fever and purulent sputum
- List of the doses of all medications and the time administered

PHYSICAL EXAMINATION

- General condition of the patient
- Any signs of infection
- Degree of dehydration
- Vital signs
- Ventilatory status, e.g. the presence of cyanosis and the degree of wheezing, dyspnoea, external retraction, and quality and symmetry of breath sounds

Management

Indications for hospital admission are:

- Failure to respond to adequate doses of epinephrine hydrochloride;
- Return to an emergency room or physician's office for therapy more than twice in 24 hours;

- A severely ill or dyspnoeic patient.

  Monitor vital signs frequently. At the time of admission, make up an asthma flow sheet containing the following information:

- Date and time of admission
- Clinical findings and vital signs
- Electrolytes, blood gases, pH
- Treatment and medication given in the emergency room.

GENERAL MEASURES

1. Give humidified oxygen.
2. Correct acidosis.
3. Rehydrate.

SPECIFIC MEASURES

After general measures have been taken, bronchodilator drugs should be tried in the following order:

1. Aerosolized sympathomimetic agents

Administer either a 0.05% isoproterenol solution or a combination of 0.04% isoproterenol and 0.06% phenylephrine solution for 5–10 minutes every half hour for the first 2 hours of therapy. Monitor pulse rate during administration. If the rate exceeds 120–140 beats/min, discontinue treatment temporarily. Avoid the use of a positive pressure ventilator (IPPR) because of the danger of inducing further bronchoconstriction and/or pneumomediastinum or pneumothorax.

2. Aminophylline

Give 3–5 mg/kg i.v. every 6–8 hours, maximum dosage not to exceed 15 mg/kg in a 24-hour period. When sympathomimetic agents and aminophylline are both used in therapy, they should be separated by an interval of 3 hours. Bradycardia is an early sign of overdosage and requires immediate cessation of the drugs. Other adverse effects include tachycardia, cardiac arrest, vomiting, haematemesis, convulsions and coma. Oxygen should be given simultaneously with sympathomimetic agents and aminophylline to prevent a decrease in Pa_{O_2}.

3. Adrenocorticosteroids

Betamethasone 0.3 mg/kg stat. followed by 0.3 mg/kg/24 h i.v; or hydrocortisone 7 mg/kg stat. followed by 7 mg/kg/24 h i.v. Administer corticosteroids promptly to all patients in status

asthmaticus; their use is mandatory in patients who have been receiving steroids before the attack. Tapering is not necessary if the total steroid course is 5 days or less.

Sedatives

If sedation is absolutely necessary, chloral hydrate (15 mg/kg every 6–8 hours given orally or rectally) is the drug of choice. Watch for hypoventilation after administration of the drug.

Epinephrine

Do NOT give epinephrine, since it failed initially to alleviate the attack and may provoke bronchial oedema.

MECHANICAL VENTILATION THERAPY

Indications are:

- $Paco_2$ above 8.7 kPa (65 mmHg)
- Pao_2 of less than 8 kPa (60 mmHg) on 100% oxygen
- Steadily rising $Paco_2$ in an exhausted patient.

In the management of patients in status asthmaticus:

- Do not treat the patient in the emergency room; however, hydration should be started immediately.
- Do not administer aminophylline in suppository form.
- Do not delay the administration of adrenocorticosteroids in therapeutic doses.
- Do not sedate the hypoxaemic patient.
- Do not use antihistamines.

4.5 HAEMOPTYSIS

Haemoptysis is the coughing up of blood.

Aetiology

- Trauma
- Foreign bodies
- Vascular lesions (bronchial varices of mitral stenosis, arteriovenous anomalies, telangiectasia, arteritis, hypertension, aneurysms and infarction)
- Infections and inflammatory lesions (pneumonia, bronchitis, bronchiectasis, lung abscess, cavitary or endobronchial tuberculosis, histoplasmosis and fungus balls)
- Neoplasia (primary or metastatic)

- Compression (right middle lobe syndrome)
- Immunological disorders (Goodpasture's syndrome, idiopathic pulmonary haemosiderosis, Wegener's granulomatosis)
- Coagulation disorders (scurvy, disseminated intravascular coagulation)
- In up to 15% of cases the cause may not be discovered

Common causes

- Tuberculosis
- Bronchiectasis
- Carcinoma

Rate of blood loss

The rate of blood loss may be difficult to evaluate due to swallowing or aspiration of blood to other areas of the lungs. In massive haemoptysis, blood loss is more than 600 ml within 48 hours. Nevertheless, smaller amounts of pulmonary haemorrhage can be life-threatening because of the danger of asphyxiation.

Site

The site of haemoptysis is determined by:

- the patient him or herself who, occasionally, can identify where the bleeding is;
- physical examination: (mitral stenosis, telangiectasis);
- serial chest X-rays, which can be used to demonstrate the degree of aspiration or to follow changes in the density of lung abscesses;
- bronchoscopy, which is the most important technique in evaluating pulmonary haemorrhage. It is also useful for clearing the airway of clots and occluding the offending bronchus. Also, tracheostomy can be performed safely over the bronchoscope to aid in maintaining a clear airway.

IMPORTANT

Nasopharyngeal bleeding is easily confused with pulmonary haemorrhage, so evaluation of the patient with haemoptysis should always include a careful examination of the nasopharynx.

Management

This consists mostly of general measures to stop the bleeding and allay the cough. Consult a specialist.

4.6 CARDIOTHORACIC INJURIES

4.6.1 THORACIC INJURIES

Chest wall and pleural injuries (closed and open) are serious because they are usually associated with altered intrapleural mechanics that have serious effects on the normal physiology of lung ventilation and cardiac function. Therefore, good results depend on a clear understanding of this disordered physiology rather than on applying routine measures.

Physiology

Normally the intrapleural pressure is slightly below the atmospheric. This partial vacuum is responsible for keeping the lung expanded. As the thoracic cage enlarges with inspiration, the subatmospheric pressure is further decreased. The elastic lung follows the chest wall as air is sucked in, and vice versa with expiration. The normal intrapleural pressure varies from -9 to -12 cmH_2O with expiration. This sucking action of the thorax during inspiration is vital for the return of venous blood to the vena cava and right heart. Once the integrity of the thorax is disturbed by trauma, the efficiency of the cardiorespiratory mechanism is interfered with, to a greater or lesser extent, according to the various factors, single or combined, provoked by injury. Any of the following conditions may be present: open pneumothorax, closed pneumothorax, haemopneumothorax, tension pneumothorax, haemothorax, fractured rib, and flail chest.

Management of thoracic injuries

Chest injuries should be managed in hospital by a specialized team.

FRACTURED RIB

Rib fractures are the commonest type of chest injury. They are usually complicated by underlying injuries. A rib fracture may result from direct or indirect trauma or, rarely, muscular violence.

Clinically, rib fracture presents with pain in the affected rib, which increases on respiration or coughing with local tenderness. Respiratory movements are short and shallow. There may be associated pneumothorax, haemothorax or haemopneumothorax.

Management

With a non-complicated fracture the aim is to relieve pain, since such fractures heal satisfactorily. This can be done as follows:

- Intercostal nerve block at the angle of the rib or at the posterior axillary lines is the quickest and surest way to relieve pain.
- Local infiltration: direct injection at the site of the fracture is effective but the relief of pain is neither as complete nor as long-lasting as intercostal nerve block. In addition, it predisposes to infection.
- Chest strapping is not recommended for a fractured rib, since it limits ventilation and does not always give full relief of pain.

FLAIL CHEST

A flail chest is an unstable thoracic cage due to double fractures of several ribs with costochondral separations or sternal fractures. A portion of the chest wall loses its continuity with the rest of the rib cage and moves inwards. This paradoxical movement interferes with the respiratory mechanism and physiology (Fig. 4.2).

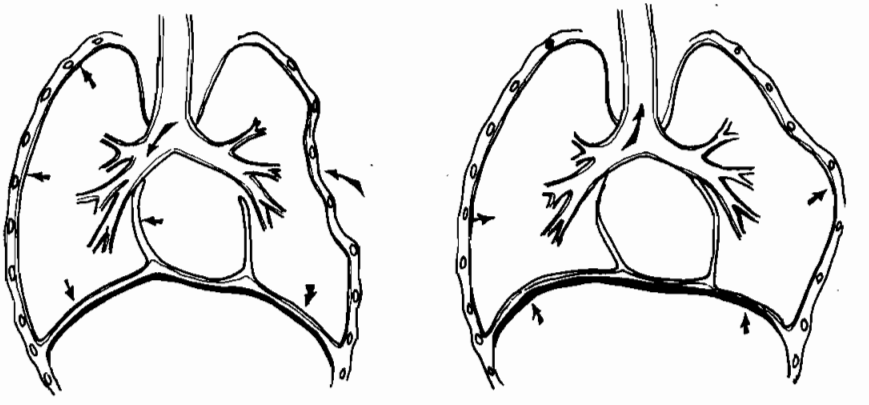


Fig 4.2 Paradoxical motion of the chest wall in flail chest.

Management

In order to delay or prevent the development of the flail where a small segment of the chest wall is involved, efforts should be made to:

1. Relieve pain. This will improve the ability to cough, thereby preventing lower airway obstruction.
2. Reduce the dead space, preferably by strapping the chest, which immobilizes the floating segment. Otherwise turn the patient on to the affected side or simply give manual support to the injured side, at the same time asking the patient to cough.

For **major flail** chest the steps are as follows:

1. Clear the airway.
2. Provide assisted ventilation (positive-pressure breathing) for cases with marked respiratory distress or severe paradoxical respiration (after excluding the presence of haemopneumothorax), by means of endotracheal intubation or tracheostomy plus assisted mechanical ventilation.
3. Stabilize the chest wall, either by means of external compression or by traction.

External compression

As a first-aid measure, firm circumferential adhesive strapping is the method of choice. The adhesive tape should be applied over a thick, firmly rolled pad of cotton wool or folded towels to conform with the size and shape of the mobile segment. Otherwise, the simplest and quickest way is to apply gentle but firm pressure of the palm of the hand against the floating segment. In lateral injuries, turning the patient on his or her side may prove effective. When available, a sandbag can be placed against the involved portion.

Traction methods

In an emergency, towel clips can be pushed through the skin to encompass one or more ribs, which can then be stabilized by manual traction. Better still, use stainless steel wire. In cases of anterior flail chest the sternum can be used for traction by passing Kirschner wire behind it. These procedures are better done under local anaesthesia.

Surgical fixation

This is the treatment of choice in cases requiring open thoracotomy.

CLOSED PNEUMOTHORAX

Closed pneumothorax is caused by blunt trauma to the chest wall.

Diagnosis

The main features of closed pneumothorax are dyspnoea, chest discomfort and chest pain; chest wall movement is diminished on the affected side, which is hyper-resonant on percussion, and breath sounds are distant on auscultation. Quick and definitive diagnosis can be done by thoracentesis.

Management

Mild cases heal spontaneously and can be left alone. Moderate or severe forms can be treated by repeated aspiration; however, intercostal tube drainage is the safest procedure. If these measures fail to give relief, look for an underlying cause, which may need thoracotomy.

OPEN PNEUMOTHORAX

Pathophysiology

Open pneumothorax (sucking wound) occurs when a wound to the chest wall results in loss of the negative pressure in the pleural cavity on the wounded side, causing the lung to collapse.

On inspiration the mediastinum is pulled contralaterally towards the non-wounded side by the increased negative pressure, and the contralateral lung (on the non-wounded side) is unable to expand. On expiration more air is pushed through the chest wall and the mediastinum swings back to the middle line or beyond (Fig. 4.3). This to-and-fro movement is called 'mediastinal flutter'. The mediastinum moves paradoxically to the intact chest wall, and there is a useless interchange of poorly oxygenated air between the two lungs across the tracheal bifurcation. This is called 'paradoxical breathing'. Thus the respiratory exchange of the good lung deteriorates, resulting in carbon dioxide retention, which stimulates the respiratory centre and accentuates the depth of respiration, aggravating the paradoxical breathing. Diminished oxygen in alveolar air will increase anoxia, dyspnoea and cyanosis.

Because of the impaired pumping capacity of the thorax, kinking of large vessels occurs with the mediastinal flutter and drainage is hindered, with consequent diminished cardiac output and circulatory failure. Therefore, an open chest is a serious emergency and must be closed as soon as possible as a first-aid measure.

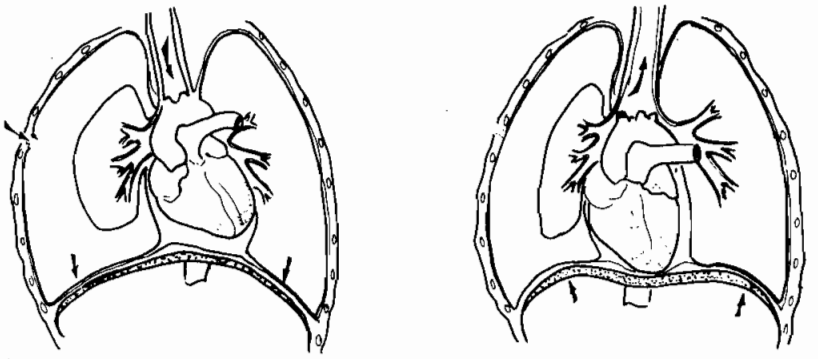


Fig. 4.3 Open pneumothorax – air enters the affected side of the chest through the hole in the chest wall rather than through the airway.

These sucking wounds, through which air moves in and out with a characteristic swishing sound, cause potentially fatal disturbances in cardiorespiratory dynamics. In addition, the foreign materials frequently carried into these wounds pose the danger of serious pulmonary and pleural infection.

First-aid treatment

Close the sucking wound immediately by any means available. Vaseline gauze is best, but as a temporary measure the open hand will do. The patient should be asked to cough or to strain in order to squeeze the air out of the pleural space. Place a dressing tightly over the wound to maintain lung expansion. In severe cases the patient's arm may be fixed firmly to the chest. This will at least partially occlude the opening. Tube drainage should be performed as soon as possible. Emergency skin suturing is not advised.

TENSION PNEUMOTHORAX

Pathophysiology

Injury or spontaneous rupture of the lung parenchyma may cause an air leak with a valve-like mechanism, resulting in tension pneumothorax or haemopneumothorax. Air enters the pleural cavity as the bronchi contract on expiration. The intrapleural pressure rises steadily, causing positive pressure within a closed hemithorax. The ipsilateral lung is compressed, the mediastinum is pushed over towards the contralateral lung which is finally compressed, and the venous return to the heart is impeded (Fig. 4.4).

Paracentesis of air, blood or both is the first-aid measure when there is evidence of mediastinal shift.

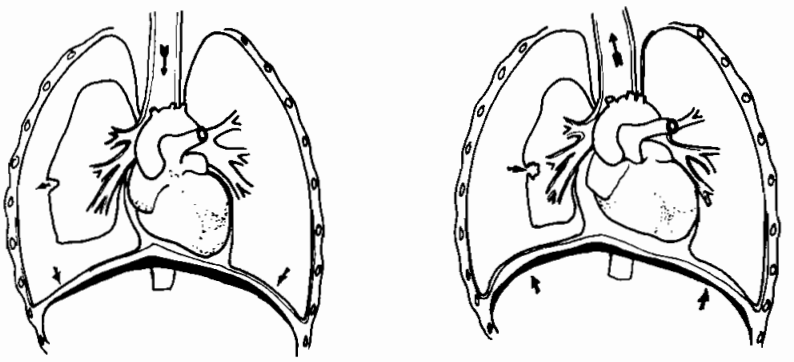


Fig. 4.4 One-way valve effect in tension pneumothorax – air escapes from the lung into the pleural space during inhalation but cannot re-enter the lung during exhalation.

Paradoxical respiration may accompany tension pneumothorax, and occurs when the integrity of the chest wall is lost. Normally in inspiration, all parts move outwards and the diaphragm downwards; when a part of the wall is flail it moves in the opposite direction to normal, being pulled inward on inspiration and outward on expiration, with diminished ventilatory efficiency. This is transmitted to the mediastinum, causing harmful effects similar to those of severe open pneumothorax.

Diagnostic features

Cyanosis, chest pain, hyper-resonance, marked respiratory distress and tracheal shift to the opposite side. Perform a diagnostic tap with a syringe: the plunger will be pushed out by the intrathoracic pressure (Fig. 4.5).

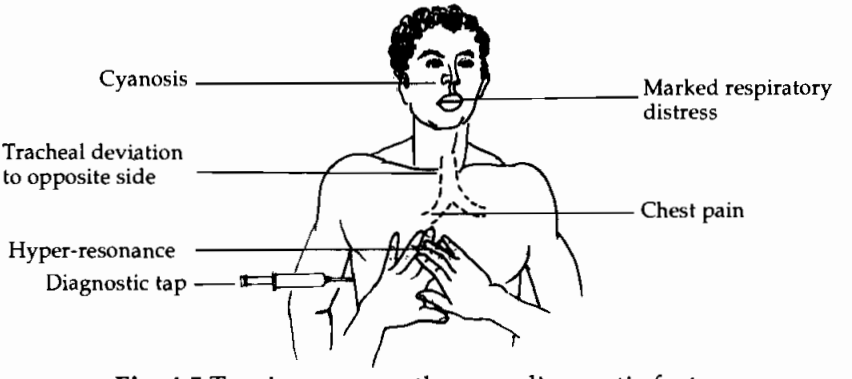


Fig. 4.5 Tension pneumothorax – diagnostic features.

First-aid treatment

The increased intrapleural pressure must be relieved at once. This can be done with a large-bore needle. Attach a flutter valve to the end of the needle by tying a perforated finger cot or condom to the hub of the needle. If this is not available, an opening should be made in the chest wall, preferably at about the second space in the midclavicular line, using any semisharp tool, e.g. a screw driver or even a nail file.

The treatment of choice is underwater seal drainage of the pleural cavity. If air continues to leak into the pleura, search for a cause which may require surgical repair.

HAEMOTHORAX

The seriousness of haemothorax and the treatment indicated will depend on the volume of blood in the pleural cavity (Fig. 4.6).

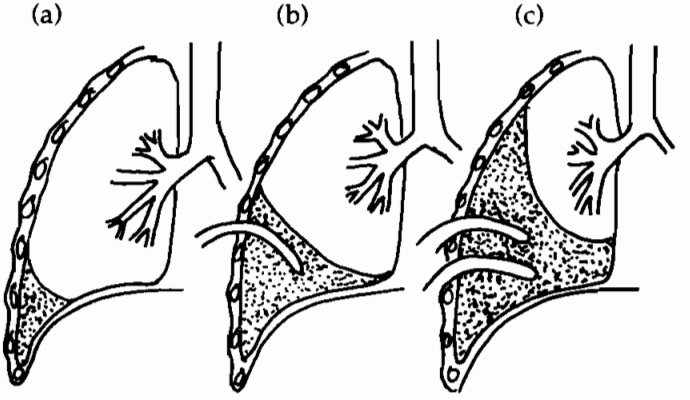


Fig. 4.6 Haemothorax. (a) Minimal (up to 350 ml); blood is usually resorbed spontaneously; thoracentesis is rarely necessary. (b) Moderate (350–1500 ml); thoracentesis and/or tube draining usually suffices; drainage is preferred initially. (c) Massive (over 1500 ml); two drainage tubes inserted since one may clog; thoracotomy may be necessary.

MINIMAL HAEMOTHORAX

Haemothorax is considered to be minimal when the accumulation of blood amounts to less than 350 ml; obliteration of the costophrenic angle on an X-ray may be the only positive sign. No treatment is

necessary, since resorption is rapid. However, all patients should be observed closely because bleeding may occasionally continue.

MODERATE HAEMOTHORAX

A total accumulation of blood of up to 1500 ml will produce shortness of breath due to lung compression, and there may be shock. X-ray in the upright position will show a shadow curving upwards and laterally on the affected side. Physical signs, including dullness and distant breath sounds, will depend on the amount of bleeding and the position of the patient. Diagnostic confirmation is obtained by aspiration.

Treatment

Thoracentesis, which may yield 500 ml of blood initially, should be repeated every 6 hours. Preferably, however, tube drainage should be instituted. The pleural cavity is entered at the 5th or 6th interspace in the midaxillary line.

Since minimal and moderate degrees of haemothorax are usually caused by bleeding from the lung parenchyma, a haemopneumothorax is usually present, both air and blood being obtained from the pleural space. Since the bleeding is from low-pressure vessels it will almost always stop spontaneously. Therefore, exploratory thoracotomy should not be considered, except rarely if rapid accumulation of blood continues.

MASSIVE HAEMOTHORAX

Rapid and continuous bleeding into the pleural cavity usually comes from larger, high-pressure vessels, such as the internal thoracic (internal mammary) artery. Less common sources are the liver and spleen in thoracoabdominal wounds.

Signs and symptoms

Compression of the lungs and the main veins produces cyanosis, tightness and severe chest pain, and engorgement of the veins in the neck. The trachea may be felt to be displaced to the contralateral side in the suprasternal notch. The affected side will show dullness, absence of breath sounds and limitation of respiratory excursion. Profound shock is produced by the loss of circulating blood.

Treatment

Blood volume must be restored and the pleural cavity emptied immediately. Blood may be removed from the pleural cavity by

needle and syringe as a temporary expedient, but a large-bore tube should be inserted and connected with a continuous negative suction underwater seal drainage as soon as possible. Two tubes should be used, since clotting in a single tube will eliminate an important means of estimating the rate and volume of blood loss which, if undetected, may lead to disastrous results.

In addition, the necessity for prompt exploratory surgery should always be considered in these patients. The decision in the individual case will depend on the location of the wound, the amount and rapidity of blood accumulation as shown by serial X-ray, and particularly by observation of the drainage bottles an intervals of 10 or 20 minutes.

Profound or increasing shock, despite active treatment with intravenous fluid and blood, calls for prompt thoracic exploration.

Underwater seal drainage

The most effective way of emptying the pleural space and keeping it clear is by underwater seal drainage. This can be carried out without suction. The chest drainage tube is attached to a bottle, partially filled with water, that stands on the floor, well below the patient's chest. The chest is sealed off from the contamination of the outside air by the water in the bottle. A negative pressure greater than the 15 cmH_2O negative pressure within the chest is produced by the column of water in the tube.

This system has several disadvantages, the most important of which is the tendency of thoughtless personnel to lift the bottle in order to empty it. If this is done without first clamping the drainage tube, fluid will siphon back into the chest.

RUPTURE OF THE AIRWAYS

Severe blunt trauma to the thorax may result in rupture of the trachea or major bronchi. The patient's clinical condition following rupture of the airway depends upon the size of the tear in the tracheobronchial tree, the level of the injury, the magnitude of the injury to the other lung, and on whether the flow of air from the tracheobronchial lumen to the pleural space is two-way, one-way, or has ceased because the traumatic defect of the airway has become obliterated.

Patients with major unidirectional air flow are most likely to be severely symptomatic, dyspnoeic and hypotensive, because they frequently develop tension pneumothorax. The symptoms in

patients with bidirectional flow are directly related to the size of the hole and the level of injury to the tracheobronchial tree. Patients with a defect of the airway sealed by adjacent tissue are usually relatively asymptomatic, unless the injured area of the trachea has resulted in an unstable segment which collapses, particularly during expiration, and thus impairs ventilation.

The clinical picture in patients with injury of the tracheobronchial tree may vary from very grave, characterized by severe dyspnoea, cyanosis and hypotension with or without massive subcutaneous emphysema, to virtually asymptomatic, except for some haemoptysis and perhaps minimal subcutaneous emphysema. Rupture of the tracheobronchial tree should be suspected in patients who experience severe blunt trauma to the chest with the above clinical manifestations. It is even strongly suggested in the very ill patient in whom the insertion of one or two chest tubes does not result in improvement and expansion of the involved lung (because there is massive air leak), or in the patient who has persistent lobar atelectasis, although otherwise clinically doing quite well.

Once rupture of the tracheobronchial tree is suspected, bronchoscopy should be done and the diagnosis established by visualizing the site of rupture. Repair of the traumatic defects should be performed as soon as possible: results are usually gratifying and the damaged lung is preserved.

4.6.2 CARDIOVASCULAR INJURIES

Physiology

Acute haemopericardium with cardiac tamponade is caused by small penetrating wounds of the heart wall, or of the intrapericardial portions of aorta, pulmonary artery or venae cavae. The trapping of blood in the pericardial sac results in three major physiological alterations:

- On the venous side, the increased intrapericardial pressure causes compression of the vena and the auricles.
- On the arterial side, cardiac compression lowers the cardiac output. This reduces coronary filling, predisposing to myocardial hypoxia and failure.
- On the systemic side, the reduced cardiac output is initially compensated by a generalized vasoconstriction. Blood pressure is thus temporarily maintained at near normal or even higher

than normal levels. Eventually, the blood pressure will fall suddenly.

Such injuries should only be handled by a specialist in a cardiothoracic centre.

Management

CARDIAC TAMPONADE

Extensive lacerations or large-calibre gunshot wounds to the heart are rapidly fatal, due to sudden and voluminous blood loss. However, small wounds as from a penknife or even a small-calibre bullet, should rarely cause death because the blood becomes trapped within the pericardium, thus tending to arrest the haemorrhage (see Chapter 5 for signs and symptoms).

Once aspiration has been carried out, the relief must be considered temporary and the patient observed closely because bleeding, which may prove rapidly fatal, may recur within minutes or days.

CONTUSION OF THE HEART

Contusion of the heart may lead to signs and symptoms similar to myocardial infarction, and ECG is required. Therefore, serial electrocardiograms should be done in patients who have sustained blunt trauma to the thorax, to detect both the early and the late ECG changes. Unfortunately, the electrocardiographic abnormalities caused by cardiac contusion are relatively non-specific, since they can be precipitated by other conditions not infrequently present in injured patients.

Other laboratory studies have not been found to be of great diagnostic value. Serum enzyme levels are so frequently elevated in concomitant liver, lung and skeletal muscle damage, or in haemorrhagic shock, that they are not very useful in establishing the diagnosis.

INTRACARDIAC RUPTURE

Rupture of the cardiac valves or interventricular septum should be suspected in patients who sustain severe blunt trauma and develop congestive heart failure. The symptoms of cardiac decompensation in these patients follow a relatively asymptomatic period of days or even years after the injury. Rupture of the cardiac valves or ventricular septum should be strongly suspected in

patients who develop a valvular regurgitant murmur, or the murmur of ventricular septal defect, with or without symptoms of cardiac decompensation, following severe blunt trauma. A cardiologist should be consulted.

RUPTURE OF THE AORTA

Rupture of the aorta is one of the most lethal of cardiovascular injuries resulting from blunt trauma. The vast majority of patients with aortic rupture die at the scene of the injury or while being moved to hospital. However, they can be treated successfully if the diagnosis is made early and treatment is instituted promptly. The usual site of aortic rupture is the aortic arch, although rarely it may occur in the distal descending or abdominal aorta. The patient with aortic rupture is usually desperately ill from other coexisting musculoskeletal, abdominal or heart injuries, though he or she may be asymptomatic, with no evidence of external injury. These coexisting injuries may mask the signs of aortic injury, therefore a high index of suspicion is required in order to establish the diagnosis of aortic rupture.

Symptoms and signs

The symptoms of chest pain, dyspnoea, back pain or inability to move the lower extremities, of which the patient with rupture of the aorta usually complains, are not specific and can be due to other injury.

The signs of rupture of the aorta are more specific. A triad suggesting aortic rupture consists of :

- increased pulse amplitude and blood pressure of the upper extremities;
- decreased pulse amplitude and blood pressure of the lower extremities;
- radiological evidence of widening of the mediastinum.

However, it must be remembered that widening of the mediastinal shadow without upper extremity hypertension might be the only finding. Equally, increased pulse amplitude of the upper extremities, without widening of the mediastinal shadow, may also be the only manifestation. For this reason, when the above triad is present in a patient who has sustained severe blunt trauma, the diagnosis of ruptured aorta is certain but the site of rupture still needs to be confirmed by aortography. This examination should therefore be performed as soon as possible in all casualties of blunt thoracic

trauma who exhibit the above triad, or part of it. The danger of exsanguination should be considered in such injuries. The results of surgical treatment are usually most gratifying.

LUNG BLAST INJURY

A nearby explosion may cause a wave of immense pressure, resulting in extensive bruising and haemorrhage in the lung, which are most marked in the underlying intercostal spaces and costophrenic sinuses. Clinically, shock, dyspnoea and cyanosis manifest in varying degrees, without signs of external injury. Unless rapidly fatal, lung blast injury tends to resolve spontaneously. There is therefore no specific line of treatment. It is important, however, **not** to give an anaesthetic.

5

Cardiovascular emergencies

Summary of contents

CHEST PAIN 103

Acute myocardial infarction 106
Right heart failure 107
Cardiogenic shock 107
Cardiac tamponade 108
Pulmonary embolism 109
Hypertensive crisis 111

SHOCK 113

ANAPHYLAXIS 124

CARDIOPULMONARY RESUSCITATION 127

5.1 CHEST PAIN

Chest pain should always be considered as being of cardiac origin and, as such, an emergency until proved otherwise. Myocardial infarction, pulmonary embolism and aortic dissection account for more than 90% of all sudden deaths. Thus, the patient with chest pain presents a special challenge to the emergency physician who has to distinguish these cases from the many who have a sometimes equally painful but less significant cause of chest pain. Chest pain is often precipitated by anxiety, temperature extremes and exertion.

Causes of acute chest pain

- Cardiovascular

Angina
Myocardial infarction
Pericarditis
Dissecting aortic aneurysm
Pulmonary embolism

- Pulmonary
  Pleurisy
  Pneumonia
  Pneumothorax
  Mediastinitis
- Oesophageal
  Oesophagitis with or without hiatus hernia
  Oesophageal spasm
  Oesophageal rupture
- Abdominal
  Acute cholecystitis
  Perforated peptic ulcer
  Acute pancreatitis
- Neuromusculoskeletal
  Cervical spondylitis
  Herpes zoster
  Tietze's syndrome
  Myositis

Diagnosis

The patient presenting with chest pain must be examined promptly.

PAIN DIFFERENTIATION

The characteristics of chest pain are often sufficient to present an initial diagnosis. Thus, the middle-aged man or woman with substernal squeezing discomfort radiating to the chin and inner aspect of the left arm is a case of coronary pain. Coronary pain is usually described as constricting or a sense of fullness under the sternum; rarely, it is knife-like or stabbing and frightening. Occasionally, it is accompanied by nausea and sweating.

Chest wall pain is usually more sharply localized, frequently to the costochondral junction. Pain sharply localized to the area of the cardiac apex and more consistent with deep inspiration is mostly chest wall pain or anxiety.

Radiation of pain to the inner aspects of the arms and the jaw is common with ischaemia. Radiation to the back points to peptic ulcer, pancreas and dissecting aorta.

The effect of various movements and positions, chest wall pain can often be reproduced by directly compressing the painful area or the costochondral junction. Pain that increases with respiration or coughing is pleuritic in origin. In pericarditis, pain is increased by lying down supine or on the left side, and is relieved by sitting up and leaning forward. An increase in the pain with swallowing suggests oesophageal spasm, pericardial inflammation or mediastinitis.

Pain that appears on exposure to cold is suggestive of coronary artery disease.

DIFFERENTIAL DIAGNOSIS

- Blood pressure should be measured in both arms and the presence or absence of both carotid pulses should be noted, since these signs may provide the only clue to a proximal aortic dissection.
- Tachypnoea in the presence of a clear chest is often the only clue to pulmonary embolism.
- Temperature greater than 38°C excludes coronary artery disease and may be due to pneumonia or mediastinitis.
- Chest palpation may elicit subcutaneous emphysema due to a ruptured oesophagus. A friction rub may help to diagnose pleurisy or pericarditis. Retrosternal dullness is an early sign of pericardial effusion.
- Abdominal tenderness, guarding and absence of bowel sounds excludes chest conditions.
- Absent femoral pulse may be the only sign of a dissecting aorta. Calf or thigh tenderness, or inequality in the size of the lower limbs, would increase the likelihood of pulmonary embolism.
- The presence of a characteristic rash is diagnostic of herpes zoster. Tenderness elicited by pressure on the xiphoid cartilage points to Tietze's syndrome.

INVESTIGATIONS

The possibilities can usually be reduced to two or three by a careful history and examination. However, it is always wise to ask for an ECG, chest X-ray and serum enzymes, bearing in mind that a normal ECG and enzyme pattern do not exclude myocardial infarction. Therefore, whenever in doubt, admit the patient to hospital for observation and further investigations.

5.1.1 ACUTE MYOCARDIAL INFARCTION

An acute myocardial infarction (AMI) or 'heart attack', occurs when a portion of the cardiac muscle is deprived of coronary blood flow, with its essential oxygen and nutrients, long enough for the muscle to die (necrosis). Atherosclerotic diseases and a generalized reduction of blood flow from any cause (e.g. shock, dysrhythmias, pulmonary embolism) are factors that diminish flow through the coronary vessels.

Clinical picture

The typical patient with acute myocardial infarction is middle-aged (45–65 years old). Chest pain occurs in 80–90% of patients.

PHYSICAL EXAMINATION

- General appearance: does the patient appear anxious, frightened, in pain?
- Vital signs: is the pulse strong or weak?
- State of consciousness : is the patient fully alert or confused?
- Is the skin pale, cold or clammy?
- Are there signs of left heart failure (wheezes or rales)?

SIGNS AND SYMPTOMS

- Extreme restlessness, agitation
- Confusion
- Severe dyspnoea and tachypnoea
- Tachycardia
- Elevated blood pressure
- Rales and often wheezes
- Frothy sputum in severe cases

Treatment of uncomplicated acute myocardial infarction

Treatment should be initiated immediately in any middle-aged or older patient with chest pain.

1. Give 100% oxygen by mask or nasal cannula.
2. Sit the patient up, with legs dangling.
3. Start an i.v. lifeline with dextrose 5% in water.
4. Monitor.
5. Administer medication on the spot as available:

- Morphine sulphate
- Nitroglycerine
- Frusemide
- Aminophylline.

5.1.2 RIGHT HEART FAILURE

Right heart failure (congestive heart failure) most commonly occurs as a result of left heart failure. As blood backs up from the left heart into the lungs, the right side of the heart has to work harder to pump blood into the already engorged pulmonary vessels. If the right heart is unable to keep up with the increased work load, it fails.

Signs and symptoms

- Distended neck veins
- Oedema of the lower limbs
- Enlargement of the liver

Treatment

- Make the patient comfortable, preferably in a sitting or semisitting position.
- Monitoring is indicated in any patient with significant cardiac disease.
- If left heart failure is present, treat it.

5.1.3 CARDIOGENIC SHOCK

Cardiogenic shock occurs when the heart is so severely damaged that it can no longer pump an adequate volume of blood at an adequate pressure to maintain tissue perfusion. Cardiogenic shock indicates that there has been extensive injury to the myocardium, and accordingly it carries a very high mortality.

Signs and symptoms

- Patient is confused or comatose
- Skin is pale and cold
- Respirations are rapid and shallow
- Pulse is racing and thready
- Blood pressure is decreased, usually below 80 mmHg systolic

Emergency treatment

1. Secure an airway and administer 100% oxygen by mask.
2. Place the patient in a supine position.
3. Start an i.v. lifeline with dextrose 5% in water to a keep-open rate.
4. Apply monitoring electrodes.
5. Prepare a dopamine infusion. Add one ampoule of dopamine (200 mg in 5 ml) to a 250 ml bag of dextrose 5% in water, to yield a concentration of 800 µg/ml.
6. Sodium bicarbonate may be given to combat the acidosis.
7. Methyl prednisolone is used in some areas for various types of shock and may be administered in a dose of 500 mg slowly i.v.
8. Transport the patient to hospital, supine.

See the section on Shock, p.113, for therapy.

5.1.4 CARDIAC TAMPONADE

Cardiac tamponade results from an accumulation of blood in the pericardial sac and may be caused by disruption of a coronary artery, rupture of the myocardium, or severe contusion. Most commonly, cardiac tamponade is seen after stab wounds to the heart, but blunt chest trauma and even acute myocardial infarction with cardiac rupture may cause this condition as well.

As blood fills the pericardial sac, the function of the heart is progressively compromised by the pressure of the surrounding fluid. Because they are compressed by the pericardial fluid, the ventricles cannot fill completely during diastole. As a consequence, stroke volume is reduced and cardiac output along with it.

Signs and symptoms

- Thready, rapid pulse
- Hypotension out of proportion to blood loss
- Narrow pulse pressure (systolic minus diastolic)
- Distended neck veins
- Muffled heart sounds

Treatment

1. Ensure a clear airway.
2. Administer 100% oxygen.
3. Transport without delay to the hospital.

4. On the way: start an i.v. with normal saline, and run the i.v. wide open.
5. If required, give isoproterenol by infusion, starting at 2μg/min.
6. If the patient becomes unconscious and the pulse is not palpable, start cardiopulmonary resuscitation.

5.1.5 PULMONARY EMBOLISM

Definition

Pulmonary embolism is the occlusion of one or more vessels in the pulmonary arterial tree by matter from a source extrinsic to the lung. It is usually acute. The obstruction is usually a clot from the distal venous system; however, it can result from fat, amniotic fluid, air, or particulate matter from intravenous injection.

Clinical picture

Depending on the size and number of the emboli, the clinical features may range from mild tachycardia and anxiety to shock and sudden death. Diagnostic techniques have improved recently, so fewer cases are missed.

Incidence

Approximately 90% of pulmonary emboli occur in those over 50 years of age. There are many conditions which increase the incidence of pulmonary embolism: congestive heart failure; venous thrombosis after major surgery, especially pelvic and lower-extremity; leukaemia; polycythaemia; sickle-cell anaemia; massive obesity; immobility; cancer, particularly of the lung, pancreas and stomach; pregnancy. The use of oral contraceptives and the early postpartum period may also be factors.

Differential diagnosis

Pulmonary embolism can easily be confused with acute myocardial infarction, pneumonia, congestive heart failure and bronchial asthma.

Symptoms and signs

- Anxiety
- Dyspnoea

- Palpitation
- Fever
- Tachycardia
- Syncope
- Clear evidence of thrombophlebitis (50% of cases)
- Pain (pleuritic chest pain in 25% of cases, or angina-type pain)
- Pleural friction rubs and haemoptysis (10% of cases)
- Additional signs: cyanosis; hypotension; right-sided congestive heart failure; increased dullness to the right of the sternum on percussion; distended neck veins; pulmonary systolic murmur and thrill with accentuated pulmonary second heart sound and wheezing. Wheezing is typically localized to the involved side and occasionally to the site of the embolus.

Hypoxaemia is almost invariably present in acute pulmonary embolism. Patients with normal heart and normal lungs can compensate by increasing their inspiratory capacity.

Laboratory tests and investigations

- Lactate dehydrogenase (LDH) is increased.
- Serum bilirubin is increased.
- Serum glutamic oxaloacetic transaminase (SGOT) is normal.
- Chest X-ray: there are non-specific changes of pulmonary infiltrate and/or pleural effusion in two-thirds of cases.
- ECG changes are: atrial arrhythmias, prominent P-waves, an S in lead I and a Q in lead III, right bundle-branch block, right axis deviation and ischaemic changes.

Prognosis

Patients with massive pulmonary embolism die within the first few hours, while those with smaller emboli generally live. The period of resolution varies depending on the clot size, age, and number of clots.

Treatment

1. Give 100% oxygen.
2. Treat the shocked patient and relieve pain.
3. Full heparinization: give 10 000 iu i.v. as initial dose.
4. Transfer the patient to the hospital intensive care unit.
5. Give thrombolytics, e.g. streptokinase or urokinase.

5.1.6 HYPERTENSIVE CRISIS

Hypertensive crisis is characterized by:

- Severe progressive impairment of function in the kidneys, eyes, and brain.
- Sustained or sudden rise in diastolic blood pressure, usually to levels greater than 120 mmHg.

Incidence

The incidence of hypertensive crisis ranges from 1%–7% among the known hypertensive population but, on rare occasions, the condition will arise in a patient with no previous history of hypertension. Crisis is seen most often in the 40–60-year-old age group, and in known hypertensives of 2–10 years' duration.

Associated diseases and circumstances

- Essential hypertension
- Chronic pyelonephritis
- Glomerulonephritis
- Other: unilateral renal artery stenosis; toxaemia of pregnancy; post-irradiation of the renal area; congenitally small kidneys; hydronephrosis; tuberculosis of the kidney; hyperadrenalcorticism; phaeochromocytoma; renin-secreting tumour of the kidney; ingestion of ephedrine, amphetamines or foods rich in tyramine by patients being treated with monoamine oxidase inhibitors for psychotherapy.

Clinical picture

Renal, eye or central nervous system changes may dominate the clinical picture, but changes in all three are usually seen.

OCULAR CHANGES

Early ocular changes may include soft exudate, haemorrhages and/or papilloedema. Varying degrees of blurred vision are present and usually resolve with appropriate treatment. Blindness is the result of neuroretinitis and/or severe obliterative arterial spasm.

HYPERTENSIVE ENCEPHALOPATHY

This may be sudden or gradual in onset, and is usually preceded by or accompanied by a severe headache. Neurologic findings are

variable and frequently result in seizures and coma. The primary underlying pathology is multiple small thrombi in the brain, with associated focal or generalized cerebral oedema. This process results from the cerebral vasoconstriction accompanying high arterial blood pressure.

A 30–50 mmHg rise in diastolic pressure may indicate hypertensive encephalopathy in some patients with toxaemia of pregnancy, and in children and adolescents with acute nephritis.

RENAL FAILURE

Renal failure may dominate the clinical picture of hypertensive crisis. The pathology is that of necrosis and endarteritis of the preglomerular arterioles and interlobular arteries of the kidney, with resultant ischaemia and necrosis of glomeruli and renal failure.

Hypertensive crisis can present as acute oliguric renal failure with or without encephalopathy.

Although a general worsening of renal function is usually seen with the initial lowering of blood pressure, renal function will, in most patients, improve in subsequent weeks. The effects of lowering the diastolic blood pressure do not appear for some months.

Management

Reduction of the diastolic arterial pressure to 100 mmHg is all that should be attempted. The blood pressure may be lowered rapidly with one of the following:

- Sodium nitroprusside: freshly prepared 50 mg in 500 ml of dextrose 5% given as a continuous infusion. This is the most rapid means of lowering the blood pressure, but there is a risk of cyanide poisoning, which is a rare complication and only occurs with overdosage.
- Hydralazine: 10 mg i.v. to be repeated every 30 min as necessary.
- Diuretics: frusemide 40–80 mg i.v.
- β-blockers: better reserved for patients who can take by mouth; nifedepine 10 mg given sublingually starts to lower the blood pressure within 10 minutes and can be repeated as required. Atenolol 100 mg is an alternative.

If the patient is comatose, transfer him or her to hospital. In the meantime, take the necessary measures for care of the comatosed patient.

5.2 SHOCK

Shock is a medical emergency that demands rapid action and constant attention by doctors and nurses to prevent irreversible cell damage and death. Shock is a syndrome of failure of the cardiovascular system to pump blood in sufficient quantity or under sufficient pressure to maintain the pressure/flow relationship necessary for adequate tissue perfusion. It leads to progressive organ dysfunction which, unless rapidly reversed, results in irreversible organ damage and death.

Types and causes of shock

HYPOVOLAEMIC SHOCK

The relative or absolute depletion of intravascular volume leads to a decrease in venous return, which leads to a decrease in ventricular output with a compensatory increase in vascular resistance. The patient thus appears cold, clammy and pale with evidence of organ hypoperfusion, confusion and oliguria as well as tachycardia and tachypnoea (secondary to metabolic acidosis due to tissue hypoperfusion and anaerobic metabolism).

Absolute hypovolaemia

The following are the causes of absolute hypovolaemia:

- Blood loss as a result of acute haemorrhage in excess of 1 l in an adult;
- Plasma loss as a result of thermal injury (burn) or third-space losses, e.g. bowel obstruction or infarcts;
- Extracellular fluid loss through:
  protracted vomiting, diarrhoea, fistula;
  excessive diuresis, salt-losing nephropathy, diuretic therapy;
  water loss due to diabetes insipidus or excessive insensible loss such as excessive sweating and fever.

Relative hypovolaemia

Relative hypovolaemia is due to a disparity between the circulatory blood volume, which is not altered, and the capacity of the vascular bed, which is markedly increased by widespread vasodilatation. It is caused by:

- reflex vasomotor paralysis in response to severe pain, excitation or trauma to certain trigger areas of the body (neurogenic shock);
- sympathetic blockade due to spinal cord injury or spinal analgesia (spinal shock);

- capillary paralysis due to release of histamine and other vasoactive substances (anaphylactic shock).

CARDIOGENIC SHOCK

A decrease in myocardial contractility leads to a fall in ventricular output, accompanied most commonly by an increase in preload and vascular resistance. The patient often presents hypotensive, cold and clammy, with evidence of organ hypoperfusion. Increased preload leads to pulmonary and peripheral oedema, organomegaly (liver) and systemic venous congestion.

Cardiac auscultation may reveal gallop rhythm. Tissue hypoperfusion results in metabolic acidosis similar to hypovolaemic shock.

Causes

- Defective cardiac filling: e.g. arrhythmias
- Defective cardiac emptying due to:
  depressed contractility – myocardial infarction of more than 40% of cardiac muscle mass
  valvular heart diseases
  outflow obstruction (obstructive shock) from massive pulmonary embolism or tight aortic stenosis

SEPTIC SHOCK (HYPERDYNAMIC SHOCK)

This is caused by sepsis, mostly Gram-negative bacteria (e.g. *E. coli, Proteus vulgaris, Pseudomonas*) and liberated endotoxins. The toxic part is a lipopolysaccharide molecule fixed to the bacterial outer membrane which activates the release of vasoactive substances.

Mechanism

- Diminished venous return:
  Extensive vasodilatation causes relative hypovolaemia.
  Increased capillary permeability causes absolute hypovolaemia.
- Contractility is depressed due to:
  coronary hypoperfusion resulting from diastolic hypotension (the most important cause)
  decreased preload
  underlying cardiac disease
  myocardial depressant factors, which are polypeptide substances produced by the pancreas and demonstrated in sepsis endotoxaemia

- Microcirculation:
  Opening of anatomical arteriovenous shunts
  Uncoupling of oxidative phosphorylation of cells (histotoxic anoxia) so the high cardiac output is often accompanied by a decrease in oxygen utilization.

Two distinct haemodynamic patterns can be distinguished in septic shock, depending on the volume status of patients: warm shock and cold shock (Table 5.1).

Monitoring of shock

BLOOD PRESSURE

In the early stages of shock, blood pressure remains normal and the body reacts with tachycardia only. When more than 20% of the

Table 5.1. Haemodynamic patterns of septic shock

Warm shock	Cold shock
In patients who are normovolaemic prior to the onset of sepsis	*In patients who are hypovolaemic prior to the onset of sepsis*
Early phase of hyperdynamic pattern:	Late phase of hypodynamic circulatory pattern:
Fever, chills	Progressive mental confusion
Hypotension (mild) but full pulse pressure	Fever or hypothermia
Peripheral vasodilatation results in warm dry extremities	Severe hypotension with weak thready pulse
High cardiac output, normal or increased blood volume, normal or increased CVP	Peripheral vasoconstriction causes cold, clammy, cyanotic extremities
Little or no change in urine output	Low cardiac output, blood volume and CVP
Hyperventilation: respiratory alkalosis	Oliguria or anuria
Thrombocytopenia	Metabolic acidosis; lactate level above 8 mmol/l
Very high increase in white cell count	Thrombocytopenia
	Depletion of clotting factors
	Disseminated intravascular coagulation
	Positive blood culture
	ECG indicates ischaemia (coronary hypoperfusion)

blood volume is lost, blood pressure starts to fall and heart rate rises. Auscultatory blood pressure may not accurately reflect central aortic blood pressure. Peripheral cuff pressure may be unobtainable due to the severe peripheral vasoconstriction.

Direct intra-arterial measurement of blood pressure is indicated in severe shock. The use of vasoactive substances in the treatment of shock needs continuous monitoring of blood pressure. Blood samples for grouping and cross-matching, chemistry and blood gases can be taken easily by arterial cannula as often as necessary.

In the absence of peripheral vascular disease, it is preferable to perform percutaneous cannulation of the femoral artery with a 16 gauge cannula. It is rapid, safe and clean and it avoids cutdown to expose the vasoconstricted radial artery and possible CNS complications of axillary artery cannulation.

Figure 5.1 shows the effects of low blood pressure, the symptoms and the follow-up action that should be taken.

CENTRAL VENOUS PRESSURE

Central venous pressure (CVP) (normal: 5–13 cmH_2O) measures primarily the right ventricular end diastolic pressure or filling pressure. It represents the relationship between venous return and right ventricular output. In the presence of pulmonary disease or left ventricular disease, CVP cannot reflect left heart filling pressure. So, pulmonary artery wedge pressure (PAWP) should be monitored.

PULMONARY CAPILLARY WEDGE PRESSURE

Pulmonary capillary wedge pressure (PCWP) measures left ventricular end diastolic pressure or filling pressure. It is indicated:

- when the patient does not respond to the initial fluid challenge and left ventricular function has to be assessed;
- when myocardial infarction is suspected as the cause of shock.

It assesses the adequacy of treatment with inotropics and vasodilator therapy. It is also important to test for mixed venous oxygen tension, which is a good indicator of tissue oxygenation.

URINE OUTPUT

A Foley catheter should be inserted to monitor urine output per hour. Shock states produce urine with the following characteris-

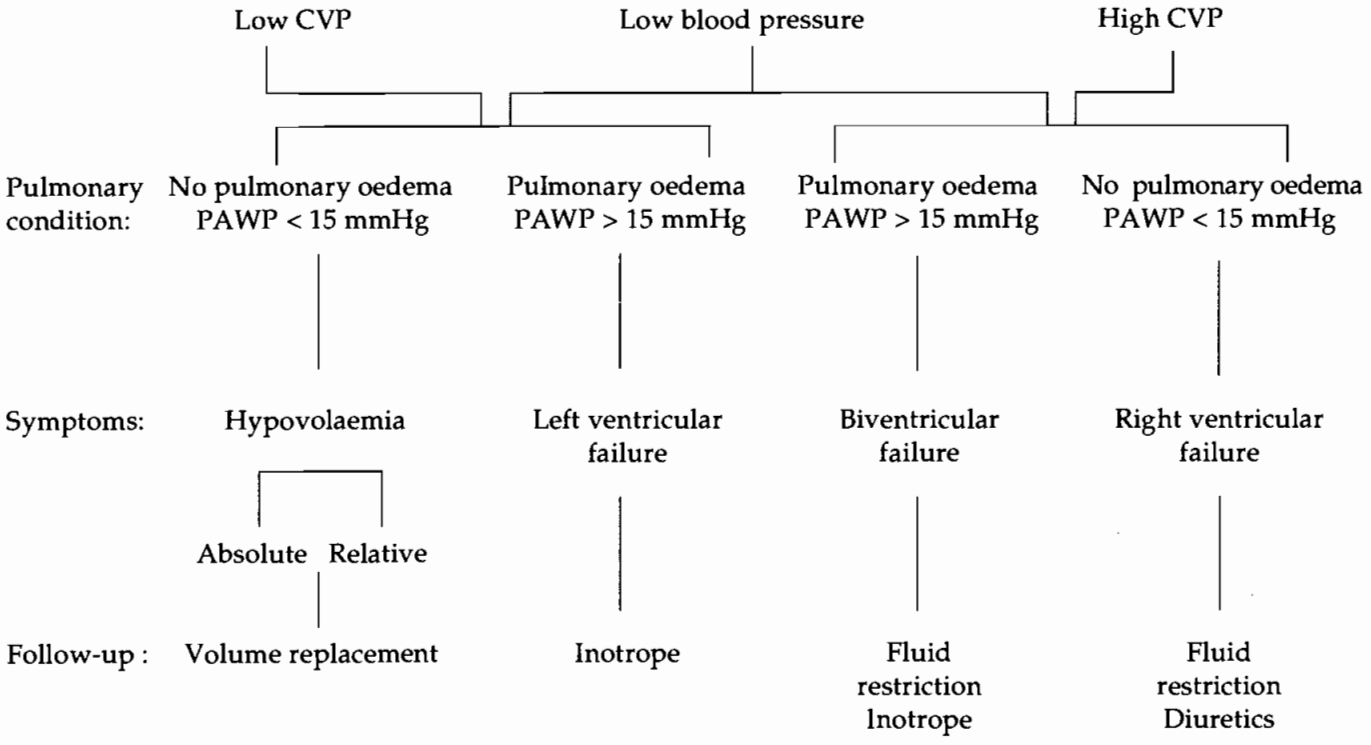


Fig. 5.1 Effects of low blood pressure.

tics: oliguria less than 0.5 ml/min, low sodium content (below 10 mmol/l) and concentration above 400 mosmol/kg. In the absence of intrinsic renal disease, the restoration of normal urine output is the best indicator of adequate tissue perfusion.

ARTERIAL BLOOD GASES

Mixed venous oxygen tension must be above 4 kPa (30 mmHg), and can be used to assess the efficiency of therapy.

HAEMATOCRIT AND HAEMOGLOBIN

- Measurements of haematocrit and haemoglobin provide a rough guide for the type of fluid loss. Loss of whole blood causes Ht below 30% (but normal in the first 24 hours). Plasma or extracellular fluid (ECF) loss leads to haemoconcentration (Ht above 35%). Blood transfusion is indicated in Ht below 30%, Hb below 10g/100 ml. Each unit of transfused blood is expected to raise Ht by 1.5–3% if there is no ongoing bleeding.

Management of shock

INITIAL MANAGEMENT

1. Arrest bleeding if possible, e.g. pressure on bleeding wounds, tourniquet, elevation of the wounded limb.
2. Give adequate pulmonary oxygenation to maximize oxygen content of the blood delivered to hypoperfused organs; oxygen supplement by nasal cannula or face mask or artificial ventilation is mandatory.
3. Provide sedation and pain relief (pethidine 1 mg/kg) in painful and stressful conditions.

Mechanical methods can be employed as follows:

1. Elevation of the legs is a rapid, effective, reversible means of increasing the circulating intravascular volume. It may be used as a diagnostic trial of volume expansion.
2. Pneumatic garment.
3. Military antishock trousers (MAST suit) increase blood pressure by increasing systemic vascular resistance and shifting some blood to central circulation.

Warmth: blankets should be used to preserve body heat but artificial warmth is to be avoided, since it causes vasodilatation.

Adequate venous access: at least two large-bore cannulae (14 gauge) are needed to ensure adequate venous access.

Transportation : great care should be taken in cases of suspected spinal fracture – immobilization using a specially designed mattress is strongly advised. In cases of suspected cervical spine fracture, a plastic collar should be fitted and skull traction applied before any attempt is made to intubate the patient.

HAEMODYNAMIC MANAGEMENT

Volume expansion

Whole blood transfusion is indicated when shock results from blood loss (Ht below 30%). Disadvantages include:

- 30–45 minutes required for typing and cross-matching
- Risk of hepatitis, AIDS (see Chapter 14)
- Risk of transfusion reactions.

The following precautions should be taken in transfusion:

1. Blood must be warmed first.
2. Regular measurements of K^+ and pH should be made after transfusion.
3. 1–2 units of fibrinogen-free plasma (FFP) must be given for every 8 units of banked blood.
4. 1 ampoule of calcium gluconate must be given for every 6–8 units of blood.

Colloids are substances of high molecular weight which do not readily diffuse from the capillary membranes. They are retained in the intravascular compartment for longer than crystalloids. Colloids include:

- plasma: fibrinogen-free plasma (FFP);
- 5% albumin and 6% hydroxyethyl starch, which act similarly as volume expanders;
- 25% albumin which increases the i.v. volume using the endogenous extravascular water (50 ml of 25% albumin recruits 200 ml extravascular fluid).

Dextran is an effective plasma expander that:

- reduces blood density and improves microcirculation;
- interferes with the platelet function coagulation mechanism;
- results in high urine specific gravity.

Dextran should **not** be given:
- before sampling for cross-matching;
- before completion of surgery (risk of bleeding);
- in more than 1 l/24 h.

Crystalloids include normal saline and lactated Ringer's solution. Three to four times the volume of blood lost is required. The major difference between normal saline and lactated Ringer's solution is negative ions, chloride versus lactate. Resuscitation with a large volume of normal saline may result in a lower pH than with lactated Ringer's solution. Approximately 50% of lactate in lactated Ringer's solution is converted to bicarbonate with normal liver function.

Risks of volume expansion

Pulmonary oedema: when volume expansion is performed according to proper haemodynamic criteria, both colloids and crystalloids are equal in their effect on pulmonary function. This is true in the presence of either normal or altered capillary permeability. If the proper criteria are not adhered to, pulmonary oedema may result.

Coronary perfusion pressure: if the proper haemodynamic criteria are not adhered to coronary perfusion pressure (CPP) may be decreased, especially in those with coronary ischaemia (CPP = diastolic pressure minus left ventricular end-diastolic pressure). If this occurs contractility should be increased. Positive inotropic therapy is indicated if there is hypotension, if cardiac output is low, and if the preload is optimal or elevated.

β–ADRENERGIC AGENTS

- Epinephrine is of choice in hyperdynamic shock; it has major inotropic effect. Give 40–100 μg/kg/min. In doses over 100 μg/kg/min vasoconstriction predominates.
- Dopamine: less than 3 μg/kg/min has dopaminergic effect (renal vasodilatation). In doses of 3–10 μg/kg/min a β effect predominates. In doses of 10–40 μg/kg/min an α effect predominates.
- Dobutamine (5–20 μg/kg/min) is of choice in cardiogenic shock because it:
  - is predominantly β stimulant;
  - is less chronotropic than dopamine;
  - decreases systemic vascular resistance, resulting in afterload reduction.

A similar haemodynamic effect is obtained by a combination of dopamine and nitroprusside.

- Isoproterenol is used mainly for its chronotropic effect in the treatment of hypotension associated with heart block or sinus bradycardia.
- Amrinone is a new non-glycoside, non-catecholamine, and is used for congestive heart failure that does not respond to other lines of treatment. Dose: 0.5 mg/kg i.v. slowly, followed by 5–10 μg/kg/min infusion.

VASODILATORS

Vasodilators reduce afterload and overcome the impedance against forward ejection of both ventricles.

Arteriolar vasodilators

Hydralazine has a delayed onset of action (10–30 min) and is of prolonged duration. It is not given by infusion. Dose 2.5–20 mg i.v.

Venular vasodilators

Nitroglycerine is predominantly a venodilator in doses below 1.5 μg/kg/min. In higher doses it also produces arteriolar vasodilatation. It decreases preload. In pregnancy it causes oedema. It also decreases filling pressure without reducing cardiac output or blood pressure. However, in hypovolaemic shock it decreases cardiac output and blood pressure. It also causes pulmonary and coronary vasodilatation.

Combined arteriolar and venular vasodilatation

Sodium nitroprusside results in both afterload and preload reduction. It has rapid onset and short duration. Dose 0.5–5 μg/kg/min. Also trimetaphan camsylate. Dose 0–10 μg/kg/min.

Important: the use of vasodilators is usually confined to patients with:

- high vascular resistance (better used based on such monitoring);
- low cardiac output;
- normal or high preload;
- adequate perfusion pressure.

Adverse effects of vasodilator therapy

- Coronary ischaemia due to diastolic hypotension
- Intracranial hypertension in patients with decreased cerebral compliance

- Hypoxaemia due to inhibition of pulmonary hypoxic vasoconstriction

INCREASE IN PERFUSION PRESSURE

Perfusion pressure to coronary and cerebral circulation through an increase in mean and diastolic blood pressure is increased by the use of vasopressors.

Coronary perfusion pressure = diastolic pressure minus left ventricular end-diastolic pressure. Diastolic pressure is raised by phenylephrine and left ventricular end-diastolic pressure is raised by nitroglycerine.

In hyperdynamic septic shock with persistent hypotension not responding to fluid resuscitation, vasopressors may be used. However, their use must be balanced against the potential hazards of increased afterload, increased myocardial oxygen demand and regional vasoconstriction leading to organ hypoperfusion (splanchnic–renal) e.g. noradrenaline 1–8 μg/min i.v.; phenylephrine 5–40 μg/min i.v. infusion.

CORRECTION OF ACIDOSIS

Sodium bicarbonate ($NaHCO_3$):

mmol required = base excess × body weight × 0.3.

Half the dose is given initially, then check pH and serum K^+.

SPECIFIC MANAGEMENT

Hypovolaemic shock

Primary therapy

1. Control losses.
2. Restore intravascular volume.
3. Correct acidosis.

Secondary therapy

1. Give positive inotropic support.
2. Reduce afterload.

Assess the adequacy of volume therapy by:

- monitoring volume status (CVP, PCWP, response to fluid challenge);
- monitoring haemodynamic efficacy (blood pressure, cardiac output and urine output).

Septic shock (hyperdynamic shock)

Primary therapy
1. Treat infection.
2. Install surgical drainage.
3. Give antibiotics (aminoglycosides, semisynthetic penicillins, cephalosporines). Antibiotics may be continued for up to 14 days according to renal status.
4. Maintain intravascular volume to optimize preload (PCWP above 15 mmHg); this is the basis for therapy. Volume requirements may exceed 1–2 l/h. If blood pressure remains unacceptably low despite optimal preload, there are two options: inotropic agents to increase cardiac output, or vasopressors to increase peripheral resistance (vasopressors are preferred in patients with known or suspected coronary artery disease).
5. Correct acidosis.

Secondary therapy
1. Give positive inotropic support – epinephrine is of choice.
2. Give vasopressor support.

Cardiogenic shock

Primary therapy
1. Optimize preload. If the patient is not in pulmonary oedema, volume is administered while monitoring CVP, PCWP, haemodynamic status, blood pressure, cardiac output, urine output. Patients whose haemodynamic status improves on fluid administration but then develop pulmonary oedema should be considered for elective intubation ventilation rather than diuresis, because reduction of preload may cause vascular collapse. Also, such patients may suffer cardiac arrest from excess respiration before diuresis takes effect.
2. Give positive inotropic support: dobutamine is of choice.
3. Reduce afterload under close monitoring of preload and vascular resistance.
4. Correct acidosis.
5. Manage arrhythmias.
6. Reassess haemodynamic interventions after each step of therapy.

Secondary therapy
1. Surgical procedures in cases of acute revascularization.
2. Intra-aortic balloon counterpulsation: the balloon, which is positioned in the descending thoracic aorta, inflates during diastole and deflates just prior to systole.

Diastole inflation augments the pressure gradients and increases coronary perfusion.

Systolic deflation decreases resistance to ventricular ejection (afterload reduction).

Such a balloon appears to be of value when myocardial dysfunction is anticipated to be transient, as in patients prepared for emergency cardiac surgery.

3. Vasopressors: if there is cardiogenic shock, decreased peripheral vascular resistance and decreased diastolic blood pressure, vasopressors may help.

MAIN COMPLICATIONS OF SHOCK

- Acute tubular necrosis (ATN)
- Disseminated intravascular coagulation (DIC)
- Adult respiratory distress syndrome (ARDS)

5.3 ANAPHYLAXIS

Anaphylaxis is a severe life-threatening allergic reaction. It is the typical immediate hypersensitivity reaction (type I) mediated by immunoglobulin E (IgE) in atopic patients, e.g. allergic individuals with personal or family histories of asthma, rhinitis or eczema. Atopy is a constitutional factor which predisposes a person to anaphylaxis. The term anaphylactoid reaction denotes an identical or very similar reaction that is not mediated by IgE. Systemic anaphylaxis falls into three main clinical patterns:

- Acute airway obstruction leading to laryngeal oedema or acute bronchospasm
- Urticaria
- Vascular collapse leading to vasodilatation of capillaries and postcapillary venules; changes in inotropy; and increased capillary permeability.

In a sensitive person these manifestations are usually rapid in onset and follow parenteral injection of the antigen. Reaction may be immediate or may be delayed for 2–15 minutes.

Anaphylaxis falls into two categories with regard to mechanism: IgE-mediated (true) anaphylaxis and anaphylaxis not mediated by IgE (anaphylactoid).

IgE-mediated anaphylaxis results in the release of biological mediators including:

- Histamine
- Eosinophilic chemotactic factors of anaphylaxis (ECF-A)
- Slow-reacting substance of anaphylaxis (SRS-A)
- Kinins
- Prostaglandins.

Types of allergen include:

- Proteins, e.g. antiserum (tetanus antitoxin)
- Polysaccharides, e.g. dextran
- Haptenes, e.g. antibiotics (penicillin) and vitamins (thiamine)
- Untested drugs.

Mechanisms not mediated by IgE (anaphylactoid) may be:

- complement-mediated through IgG or IgM, e.g. transfusion reaction, or through lipopolysaccharides, e.g. endotoxins in septic shock;
- arachidonate-mediated, e.g. anaphylaxis of aspirin;
- direct mast-cell and basophil-releasing agents, e.g. most narcotics, D-tubocurarine and radiographic contrast media;
- idiopathic.

DIAGNOSIS

- Personal or family history of atopy (rhinitis, eczema, asthma)
- Immediately or very rapidly following parenteral injection of antigen
- Clinical pattern: urticaria; laryngeal oedema and/or bronchospasm; vascular collapse. These are all usually rapid in onset but symptoms may vary from mild pruritis to irreversible shock.

Treatment

IMPAIRED ABSORPTION OF ANTIGEN

1. Give epinephrine hydrochloride 1:1000, 0.3 ml (300 μg) injected into the relevant site.
2. Apply a tourniquet proximal to the injection site.

LARYNGEAL OEDEMA AND BRONCHOSPASM

1. Give epinephrine hydrochloride 1:1000, 0.5 ml subcutaneously, into the non-occluded extremity.
2. Give humidified oxygen by controlled flow.
3. Give aminophylline, 500 mg diluted into 250 ml dextrose 5% in water intravenously over 1 hour.

4. Apply an IPPR ventilator with nebulized isoproterenol 1: 200/0.5 ml, and saline 1.5 ml.
5. Install endotracheal intubation with assisted respiration if adequate oxygenation cannot be established in 1–3 minutes.

HYPOTENSION

1. Start generous crystalloid infusion (Ringer's solution).
2. Give hydrocortisone sodium succinate, 100 mg i.v. stat. bolus with 100 mg added to 250 ml dextrose 5% in water. Higher doses may be required.
3. Give dopamine hydrochloride 5–20 μg/kg/min.
4. Give norepinephrine, 2 ml of 0.2% solution added to 500 ml dextrose 5% in water (4 μg/ml). Rate: 2 ml/min.

Prophylaxis

Minimization of anaphylaxis requires anticipation of:

- the sensitivity of the recipient
- the antigenicity of the drug
- the dose and rate of absorption of the drug.

History taking is mandatory. All patients must be questioned regarding previous drug reactions, with specific reference to the drug or test dose to be administered, e.g. before treatment with heterologous serum. No previous reaction to the drug is never a guarantee of non-reactivity, therefore a skin test should be performed. Intradermal testing with 0.2 ml of 1/10 dilution of serum should be negative before routine administration. A skin test is negative if there is no local reaction, or if reaction is very slight – less than 5 mm local reaction.

If there is reaction to a skin test, substitution therapy is imperative e.g. the use of human tetanus immunoglobulin (tetanus toxoid) instead of horse serum for passive immunization. Tetanus toxoid is, in any case, the vaccine of choice nowadays because of the risk of anaphylaxis.

For radio-opaque contrast media (RCM) a routine intravenous test dose should be given initially i.v. because skin test reactions correlate poorly with clinical experience. Prophylactic administration of prednisone 50 mg every 6 hours should be given in three doses ending 1 hour before the RCM test, and diphenhydramine 50 mg i.m. 1 hour before RCM administration.

Patients treated for anaphylactic shock should be advised subsequently to undergo desensitization therapy.

5.4 CARDIOPULMONARY RESUSCITATION

The chest contains the most vital of life-sustaining organs – the heart and lungs. Therefore, chest trauma seriously compromises the function of these organs and, unless treated on the spot or within a short period of time, death often occurs rapidly. The lives of over 80% of patients with critical but reversible chest problems can be saved with the application of a few simple measures. Therefore, all physicians must be familiar with the main diagnostic signs, their altered physiology and the measures that can be taken. The immediate danger to life from thoracic trauma is usually due to respiratory and/or circulatory derangement. Respiratory insufficiency immediately after trauma may be due to:

- Upper airway obstruction
- Impaired gas exchange at the alveolar level due to direct lung injury
- Lower airway obstruction due to aspiration of blood, vomitus, foreign material, bronchospasm, excessive secretions, etc.

Circulatory derangements are usually due to:

- Blood loss
- Cardiac tamponade
- Cardiac failure from rupture or contusion of the heart.

For cardiopulmonary resuscitation first aid, the vital steps to be followed are: airway, breathing and circulation. Causes of respiratory and cardiocirculatory arrest are shown in Fig. 5.2.

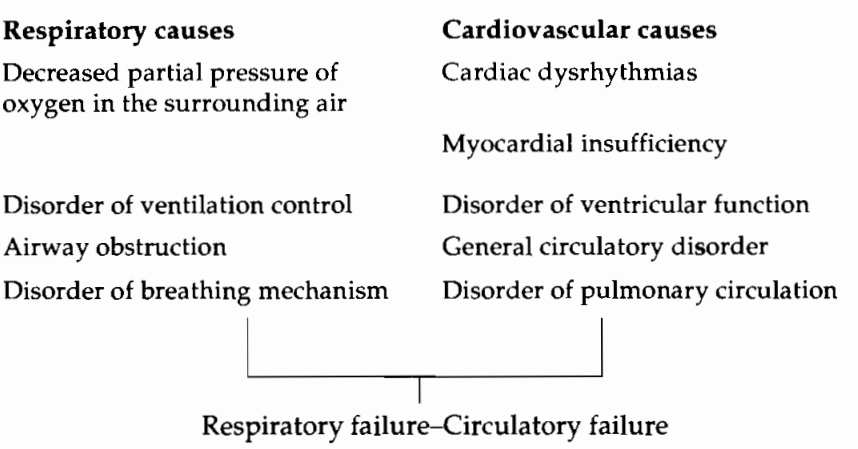


Fig. 5.2. Causes of respiratory and cardiocirculatory arrest.

5.4.1 UPPER AIRWAY OBSTRUCTION

Diagnostic features

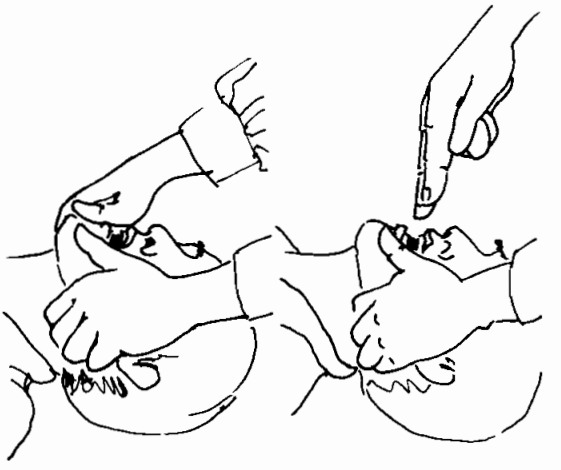
The patient with upper airway obstruction is cyanotic or ashen grey, and on each inspiration there is:

- strong inspiratory effort with 'crowing' respiration
- contraction of cervical muscles
- suprasternal, supraclavicular, intercostal and epigastric retraction.

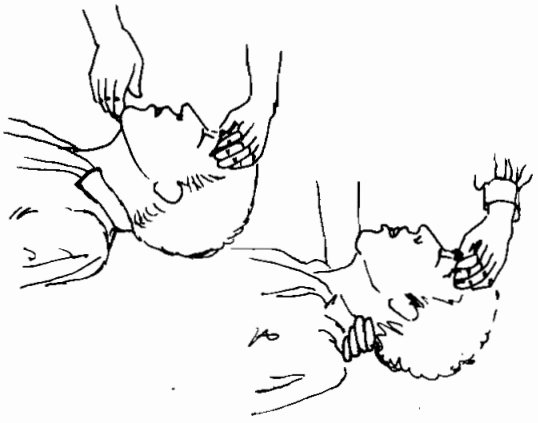
Management

Clear and maintain the airway and give oxygen as follows.

1. Clear the airway of blood, mucus, vomitus and all foreign materials with a finger, a gauze sponge or if necessary by catheter suction.This must be carried out rapidly and efficiently (Fig. 5.3a). The possibility of the tongue slipping back into the throat can be eliminated by turning the head to the side.
2. If the mouth and throat are clear, or have been cleared and respiration is still laboured, the airway may be obstructed by faulty positioning of the neck and jaw. This can be corrected by extending the neck and holding the jaw forward. This procedure, with or without an oxygen mask, may be sufficient for efficient ventilation (Fig. 5.3b).



(a)



(b)

Fig. 5.3 Clearing the airways. (a) and (b) are explained in the text.

If the above procedures fail and respiration is still laboured, with retraction of the suprasternal notch, or if signs of laryngeal stridor are present, the next step will depend on the equipment at hand.

An **oropharyngeal airway** is a quick and easy way to provide a clear airway in the unconscious or comatose patient. This method will ensure airflow, but it will not prevent inhalation.

A **nasopharyngeal airway** passed via the nose to the oropharynx will prevent complete blockage of the pahrynx by the tongue, but will not prevent aspiration.

Cricothyroid stab is resorted to in cases of complete upper airway obstruction when the above procedures fail. The cricothyroid membrane must be opened at once, using any relatively sharp instrument. The transverse stab is done 13 mm below the thyroid cartilage because it is a relatively avascular area. Once the trachea is opened a piece of rubber tubing, if available, may be inserted into the airway to keep the aperture open. Wide-bore needles may be inserted but are usually insufficient.

Endotracheal intubation is the treatment of choice if available. It is the quickest and easiest way to ensure a clear upper airway. Moreover, it passes the larynx and eliminates vocal cord obstruction. A cuffed tube will prevent tracheal aspiration and permit positive pressure ventilation. This tube can offer an adequate airway for hours or days, and can also be installed following a tracheostomy. Once in place, the physician can look for other serious problems. The main advantages of the tube are that it:

- ensures a clear upper airway;
- prevents inhalation;
- diminishes airway resistance;
- allows positive pressure ventilation;
- allows aspiration of the lower airway for secretion;
- facilitates induction of anaesthesia.

Tracheostomy may be life-saving in chest trauma but is time-consuming and more difficult than endotracheal intubation.

These procedures are illustrated in Chapter 1, Figures 1.2–1.7.

5.4.2 LOWER AIRWAY OBSTRUCTION

Causes

- Inhalation of blood, vomitus, etc.
- Bleeding from injured lung tissues
- Loss of ciliary function of the bronchial tree (mechanical or reflex)
- Bronchospasm
- Excessive mucus, transudates
- Drowning

Clinical picture

The main signs are:

- Cyanosis
- Dyspnoea
- Ineffective cough
- Rales, wheezes, rhonchi
- X-ray signs of atelectasis and/or pneumonia
- Increased $Paco_2$
- Decreased Pao_2
- Decreased pH

Management

The first-aid treatment will depend upon the patient's condition and the facilities at hand.

CONSCIOUS PATIENT

The best way to clear the airway is by voluntary cough. In a conscious patient cough should be encouraged while manual sup-

port is given to the chest to decrease pain. When the ribs are fractured pain can be relieved by intercostal nerve block or pain-relieving drugs.

UNCONSCIOUS PATIENT

Nasotracheal suction

Use a small rubber tube or catheter. After lubrication pass it down to the larynx via the nostril to a maximal depth. Apply suction for 3–4 seconds. It is essential to interrupt suction to allow the patient to inhale, otherwise hypoxia and even cardiac arrest may occur. Well performed nasotracheal suction will save the patient from bronchoscopy and/or tracheotomy (Fig. 5.4).

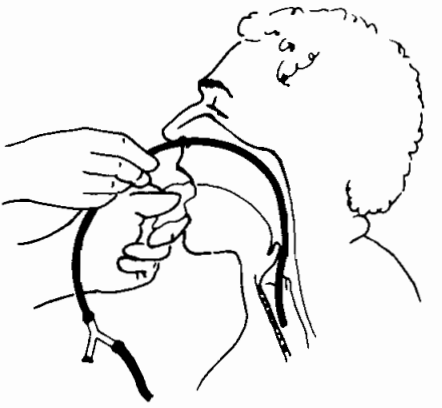


Fig. 5.4 Nasotracheal aspiration.

Bronchoscopy

Bronchoscopy enables the removal of foreign bodies, diagnosis of any tracheal or bronchial tears and identification of the source of bleeding. Oxygen can be given with the ventilating bronchoscope. Bronchoscopy is essentially needed in suction of inhaled gastric contents and in cases with atelectasis.

Tracheotomy

This is indicated in cases with multiple rib fractures, flail chest and/or contusion pneumonitis complicated by retained secretions and head injuries. This procedure decreases airway resistance and respiratory dead space. It also facilitates suction and lung ventilation.

Positive pressure breathing
This helps to overcome airflow resistance and expands the alveoli of stiff lungs.

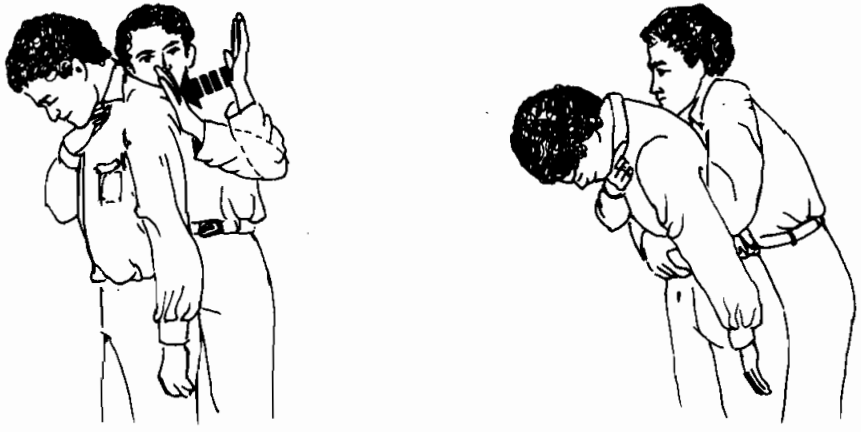
Drugs
Narcotics (morphine etc.) should be used cautiously because they cause depression of cough and/or respiration. Bronchodilating drugs such as isoproterenol and aminophylline are of value. Appropriate antibiotics should be given without delay in the hope of preventing obstructive atelectasis and secondary pneumonia.

5.4.3 COMPLETE AIRWAY OBSTRUCTION (SUFFOCATION)

Complete obstruction to the airway usually occurs during eating, as a result of inhalation of food particles which obstruct the laryngeal opening or even the trachea. Suffocation may occur in infants and children when playing with small objects, e.g. marbles, buttons and uninflated balloons.

The condition is usually of sudden onset. The patient is unable to speak, cough or breathe, and usually puts a hand to the neck. The patient turns blue (cyanosis), and if not treated immediately will lose consciousness and the heart will stop.

Treatment



(a) (b)

Fig. 5.5 Complete airway obstruction in the conscious patient (a) back blows (b) abdominal thrusts.

ADULTS

The conscious patient

If possible, remove the obstruction by putting the finger into the mouth and then try slapping the patient forcibly between the shoulders several times (Fig. 5.5a). If this fails stand behind the patient with one foot between the patient's feet and apply the Heimlich technique or abdominal thrusts. Put your arms around the patient's waist in such a way that your fists lie on his or her stomach. Press inwards and upwards quickly and strongly 5–10 times, then relax (Fig. 5.5b). This results in the escape of air from the lungs through the larynx, forcibly pushing the foreign body into the mouth. Repeat the process several times until the foreign body is ejected, or until the patient loses consciousness. If the patient loses consciousness, place him or her carefully on his back and proceed as follows.

The unconscious patient

With the patient lying on the back, try to clear the airway by hand and then by back blows (Fig.5.6a). If this is not possible, close the nose and start mouth-to-mouth artificial respiration. If this does not succeed in ventilating the patient and the airway is still obstructed, try the Heimlich technique. Kneeling beside the patient, place the heels of your hands one above the other on the patient's stomach, just below the sternum, and push forcibly and rapidly upwards and backwards several times (Fig. 5.6b). If this manoeuvre succeeds, open the patient's mouth and extract the foreign body, then start mouth-to-mouth breathing. Check the patient's pulse. If it cannot be felt, start cardiopulmonary resuscitation.

Fig. 5.6 Complete airway obstruction in the unconscious patient. (a) back blows; (b) abdominal thrusts.

CHILDREN

For infants: place the infant on his adbomen stretched on one of your arms so that his head is lower than his abdomen. Insert your index finger into the infant's mouth to keep it open. You can use your thigh to prevent the infant falling. With the heel of the other hand, slap the infant between the shoulders forcibly several times (Fig. 5.7). Repeat until the foreign body is ejected.

Fig. 5.7 Back blows in infants.

Mouth-to-mouth breathing should then be performed to ensure oxygenation. Check the pulse to ensure the efficiency of the circulation. External cardiac massage may be indicated.

For children over 1 year of age perform the Heimlich technique by abdominal thrusts as for adults. See also Chapter 10 for more detail on these techniques in children.

5.4.4 CARDIAC ARREST

The brain can survive a lack of oxygen for 3–4 minutes only. Active energetic support of both ventilation and circulation must be started immediately, even if some doubt as to the condition exists. Figure 5.8 summarizes the steps to be taken in cardiac arrest. The vital steps are: airway, breathing, circulation, in that order (Figs 5.9 and 5.10). If help is available, attention should be given to ventila-

tion and circulation at the same time. Otherwise, the first priority must be given to ventilatory support – airway and breathing. In other words adequate upper and lower airways must be established.

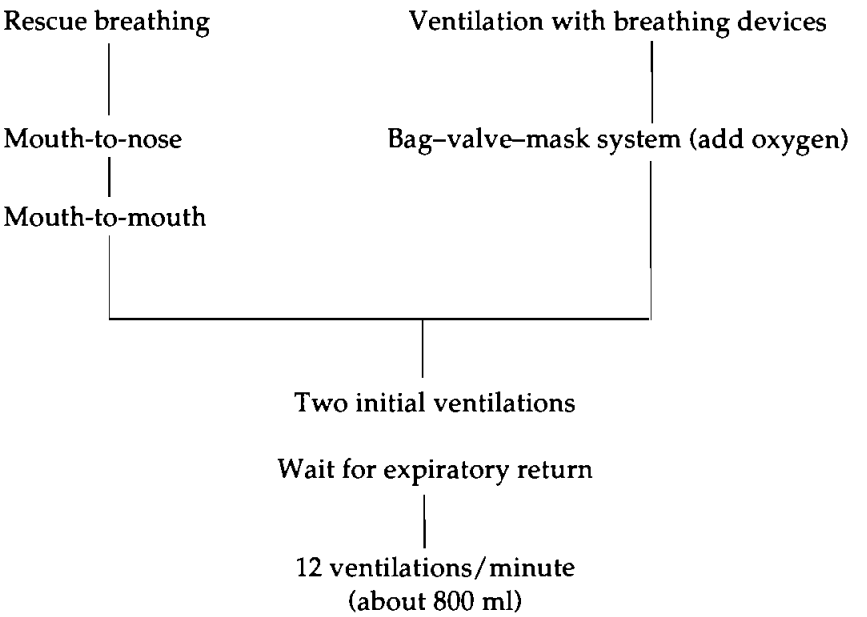
Symptoms
↓
Cardiocirculatory arrest
↓
Supine position
↓
Clear the airways
↓
Continued respiratory arrest
↓
Ventilate twice
↓
No carotid pulse
↓
Cardiopulmonary resuscitation
↓
Ventilation/cardiac compression
↓ ↓
One-rescuer Two-rescuer

Fig. 5.8 Summary of management of cardiac arrest.

1. **Clear the airway**
↓
   - Inspect the oropharynx
   - Remove foreign bodies
   - Tilt the head back
   - Pull chin forward
   - Heimlich manoeuvre for bolus obstruction

↓
2. **Maintain the airway**
   - Nasopharyngeal airway
   - Oropharyngeal airway
   - Endotracheal intubation

Fig. 5.9 Basic principles of cardiopulmonary resuscitation (airways management).



Check
Control ventilation. Is it effective?
Do chest and abdomen rise and fall?
Is there an expiratory return of air?

Fig. 5.10 Basic principles of cardiopulmonary resucitation (breathing).

Artificial respiration

In an emergency, once the upper airway is cleared, start mouth-to mouth resuscitation as follows:

1. Hyperextend the patient's head (Fig.5.11a), pull the jaw forward and occlude the nostrils with the fingers.
2. Cover the patient's open mouth with your own lips and exhale with a force and depth that will noticeably expand the patient's chest (Fig.5.11b). Withdraw the mouth, allowing the patient's chest to contract. Repeat intermittently and regularly at a rate of 20 times per minute (Fig. 5.12).
3. In cases of severe maxillofacial injury initiate mouth-to-nose resuscitation (Fig.5.13).

A self-expanding Ambu bag, if available, is far superior to the mouth-to-mouth or mouth-to-nose method.

(a) (b)

Fig. 5.11 Mouth-to-mouth artifical respiration. (a) and (b) are explained in the text.

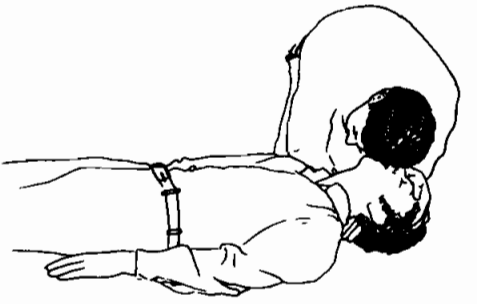
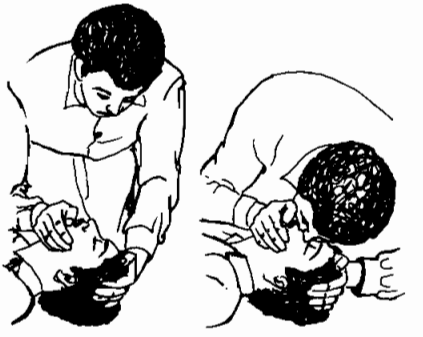


Fig. 5.12 Checking the effect of artificial respiration.



(a) (b)

Fig. 5.13 Mouth-to-nose artifical respiration. (a) and (b) are explained in the text.

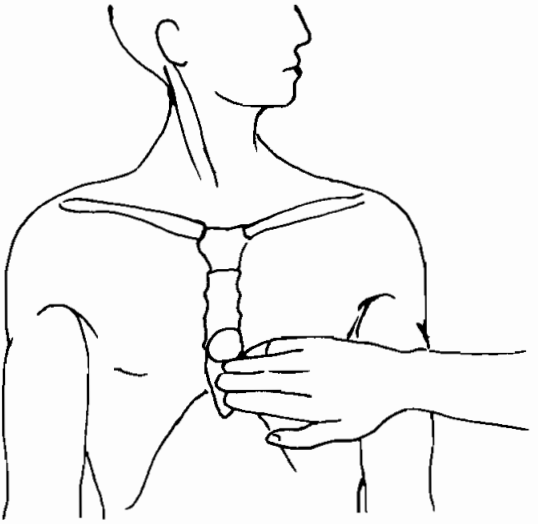


Fig. 5.14 Locating the point of cardiac compression.

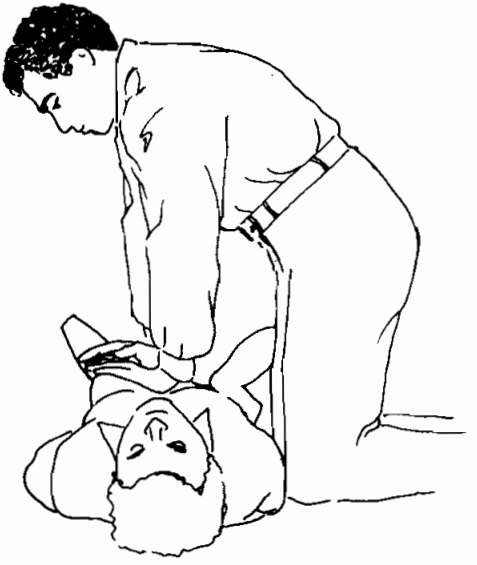


Fig. 5.15 Execution of external cardiac compression.

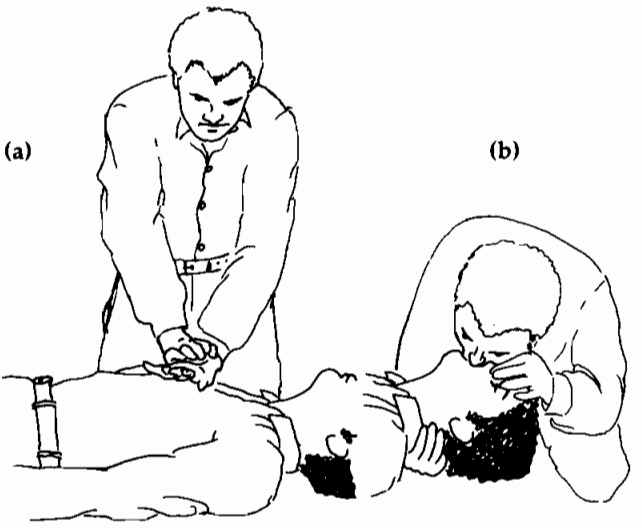


Fig. 5.16 CPR – one-rescuer method. (a) and (b) are explained in the text.

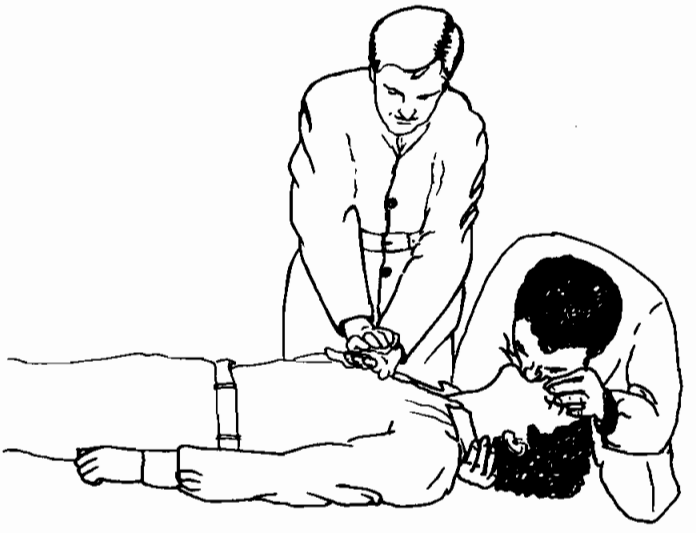


Fig. 5.17 CPR – two-rescuer method.

Cardiac massage

Figures 5.14 and 5.15 show the location and execution of closed chest cardiac compression (massage).

Procedure

1. Place the patient supine on a firm level surface.
2. The shoulders of the person applying pressure should be high, so that force can be applied with the arms straight and the elbows stiff.
3. Place the heel of one hand, with the other superimposed, on the lower third of the sternum. Apply sharp downward force with the weight of the body, sufficient to depress the sternum 2.5–5 cm. Release pressure promptly.
4. Repeat these short, sharp compressions rhythmically at a rate of 60–80/min.
5. After every three compressions, pause momentarily to permit mouth-to-mouth resuscitation and maximum lung expansion. If this is done properly a carotid or femoral pulse should return and a blood pressure of at least 60–70 mmHg obtained.

Figures 5.16 and 5.17 show how to perform cardiopulmonary resucitation, singly or in a pair.

Contraindications

- Certain chest injuries, such as a stab wound
- Wounds to the heart and major vessels, because bleeding and/or tamponade will be increased
- Tension pneumothorax
- Extensive traumatic diaphragmatic herniation
- Fractured ribs

These conditions require immediate thoracotomy and internal cardiac massage, along with pulmonary resuscitation to establish and maintain ventilation, and simultaneous efforts to control haemorrhage and/or other underlying causative factors.

OPEN CARDIAC MASSAGE

When external compression proves ineffective within a few minutes or when it is contraindicated:

1. Perform a rapid thoracotomy via the left fifth interspace with a sterile scalpel, and after quickly sterilizing the skin.

2. Separate the ribs rapidly.
3. Thrust the hand into the chest. Grasp the heart by placing the fingers behind and the thumb in front and rhythmically compress it through the intact pericardium against the sternum. Compress at a rate of about 70 compressions per minute using a gliding motion from the apex to the base. Complete relaxation must be permitted between each compression to allow for adequate cardiac filling.
4. If this is not immediately effective, or if there is tamponade, the pericardium should immediately be opened longitudinally, anterior to the phrenic nerve. Evacuate the blood and control bleeding from myocardial wounds by digital pressure. A myocardial wound may then be sutured. If major vessels are ruptured, massage is started and continued and the chest opened sufficiently to permit identification and control of the source of bleeding.

OTHER MEASURES

If the heart fails to beat in spite of adequate massage, 0.5–1 ml of 1:1000 epinephrine hydrochloride should be injected directly into the heart to trigger its action.

Continued cardiac massage. If myocardial contractions are restored but are feeble, 5–10 ml of 10% solution of calcium chloride, or 1–2 ml of isoproterenol hydrochloride should be injected directly into the heart, or into a vein, to improve both myocardial and contractile force.

Intravenous sodium bicarbonate ($NaHCO_3$), is mandatory to combat metabolic acidosis; it depresses myocardial contractile force and sensitizes the heart to the action of epinephrine hydrochloride and isoproterenol hydrochloride. For each estimated minute of arrest give 44.6 mmol sodium bicarbonate i.v. (one ampoule).

Electrical defibrillation is used in the presence of ventricular sodium bicarbonate fibrillation (20–100 J). A single shock often proves effective. If not, a series of two or three shocks in rapid succession should be given. To be effective the heart must be maximally oxygenated and in good tone.

6

Abdominal emergencies

Summary of contents

ACUTE ABDOMINAL PAIN 142

FOOD POISONING 149

OESOPHAGEAL VARICES 151

ABDOMINAL INJURIES 152

Signs and symptoms 153
Radiological studies 154
Laboratory examination 155
Other studies 155
Emergency management 156
Specific injuries 157

UROLOGICAL INJURY 159

6.1 ACUTE ABDOMINAL PAIN

Abdominal pain is the most common complaint presenting in patients with acute surgical or medical emergencies of the abdomen. The task of the physician is to decide the most likely diagnosis and to prove that the diagnosis is correct, so as not to miss dangerous situations that may cost patients' lives. The physician should at least categorize the case as medical or as surgical needing intervention.

In examining patients with abdominal pain, it is important that no analgesics or sedatives are given until a diagnosis is reached. Patients should be placed under observation and their condition followed up; at the same time an i.v. line should be established.

History

It is of utmost importance in patients with acute abdominal pain to take a detailed history.

SITE OF PAIN

Certain visceral organs provide reasonably good localization of the pain they generate (Table 6.1).

RADIATION OF PAIN

- Biliary pain radiates around the right side of the back to the angle of the scapula and/or shoulder.
- Pancreatic pain radiates directly through the back.
- Appendicular pain may start as periumbilical and ultimately settles at the right lower quadrant.
- Ureteric pain radiates to the corresponding testis and root of thigh.
- Perihepatic inflammation radiates to the right shoulder.
- Adnexal and rectal pain radiates to the lower back.

TYPE OF PAIN

The pain may be colicky or a constant ache. Colicky pain is caused by:

- hollow organs when they become obstructed, e.g. intestinal obstruction;
- ureteral stone;
- dysmenorrhoea;
- increased intraluminal pressure in a hollow organ without obstruction, e.g. enteritis due to increased peristalsis; superior mesenteric artery thrombosis, which may start with severe colicky pain.

Constant pain comes and goes frequently and is usually the result of inflammatory or neoplastic involvement.

DURATION OF PAIN

Acute appendicitis will not persist as a local process for more than 5 days, whereas acute cholecystitis will not persist for more than 48 hours before a complication ensues.

Table 6.1 Pain localization and its visceral origin

Site	Differential diagnosis
Right upper quadrant	Acute cholecystitis
	Acute pancreatitis (bilateral pain)
	Acute congestive hepatomegaly
	Acute hepatitis
	Perforated duodenal ulcer
	Hepatic abscess
	Acute pyelonephritis
	Pneumonia with pleuritic reaction
Left upper quadrant	Ruptured spleen or splenic infarction
	Perforated gastric ulcer
	Acute pancreatitis (bilateral pain)
	Acute myocardial infarction
	Ruptured aortic aneurysm
Central (periumbilical)	Intestinal obstruction
	Food poisoning
	Acute appendicitis
	Dissecting or ruptured aortic aneurysm
	Diverticulitis
	Enteritis
Right lower quadrant	Appendicitis
	Meckel's diverticulitis
	Salpingitis
	Tubo-ovarian abscess
	Twisted ovarian cyst
	Ruptured ectopic pregnancy
	Regional ileitis
	Perforated caecum
	Ureteral stone
	Strangulated inguinal hernia
	Psoas abscess
Left lower quadrant	Acute salpingitis
	Twisted ovarian cyst
	Tubo-ovarian abscess
	Ruptured ectopic pregnancy
	Ureteral stone
	Strangulated left inguinal hernia
	Regional ileitis
	Perforated colon (tumour or foreign body)
	Psoas abscess

INTENSITY OF PAIN

A perforated peptic ulcer characteristically causes very severe pain due to the acid nature of the leaking contents, whereas pain caused by pancreatitis is less intense and is due to enzymes leaking into the peritoneal cavity and retroperitoneal area. Ureteral stones cause severe colicky pain.

TYPE OF ONSET OF PAIN

Sudden and abrupt onset occurs in perforations. Gradual onset occurs in intestinal obstruction, appendicitis, diverticulitis and pancreatitis.

ASSOCIATED VOMITING

Frequent vomiting at the onset points to:

- the gall bladder and the pancreas
- high intestinal obstruction
- food poisoning.

OTHER DIAGNOSTIC POINTS

Age: for example:

- Appendicitis occurs in patients aged 5–40 years;
- Intussusception only occurs under the age of 2 years;
- Cholecystitis is uncommon under the age of 30 years.

Position: the patient naturally assumes the position that he or she finds most comfortable to obtain pain relief. This can be helpful in diagnosing the cause. For example:

- Pancreatitis: the patient characteristically lies on the left side flexing the back, hips and knees;
- Peritonitis: the patient lies still and immobile;
- Appendicitis: the patient lies supine flexing the right hip and knee;
- Renal colic: the patient is anxious and tosses about.

Table 6.2 summarizes the differential diagnosis of severe epigastric pain.

Table 6.2 Differential diagnosis of severe epigastric pain

Diagnostic features	Perforated peptic ulcer	Acute pancreatitis	Acute cholecystitis
Onset	Sudden, sharp	Gradual	Gradual
Location	Epigastric generalizes rapidly	Epigastric slowly spreading (early)	Right upper quadrant only
Radiation	Diffuse	Through to the back	Around to the back and angle of the scapula
Vomiting	Absent to few times	Multiple episode persistent	Few to many times
Alcoholic intake	Variable	Usually heavy preceding attack	Occasional, not heavy
Previous attacks of pain	Ulcer history (45% of cases)	Frequently similar to current episodes	Frequently similar to current episode
Dietary intolerance	Spices, alcohol	Fatty foods	Fatty foods, cabbage
Shock, prostration	Common early	Seen late	Unusual
Tenderness	Diffuse	Epigastric, diffuse	Right upper quadrant
Rebound tenderness	Early (first 4 hours)	Late (after 24 hours)	Rare
Rigidity	Boardlike	Moderate to severe	Unilateral rectus guarding
Peristaltic sounds	Absent	Hypoactive	Normal to hypoactive
Costo-vertebral angle tenderness	Bilateral	Left-sided	Right-sided
Position	Supine	Lying on side, hips flexed	Supine or on side
X-ray	Free (70% have ileus)	Ileus, sentinel loop, colon cutoff sign	Ileus, calculus in right upper quadrant (10%)
		Sonogram (β-mode) positive for pancreatic mass	Sonogram positive for stones in gallbladder
Laboratory	Moderate amylase elevation, elevated haematocrit, high leucocyte count	Marked amylase elevation, modest haematocrit elevation, low calcium and magnesium (after 5 days), glycosuria	Minimal amylase elevation, moderate rise in leucocyte count

Physical examination

VITAL SIGNS

The general appearance of the patient is suggestive of the severity of the condition.

Fever is common with acute abdominal disease. It is important to follow up the pattern of changes in the temperature. With appendicitis, intestinal obstruction or ectopic pregnancy the temperature is around 38.5°C. If perforation occurs, it will rise to 40°C, then fall if there is shock.

An initial temperature of over 39.5°C is more likely with pulmonary and urinary infections, rather than intra-abdominal disease. A temperature of over 40°C at any time indicates abscess, severe systemic infection, infection of the CNS, lung or urinary tract.

An unexplained rising pulse rate in a patient with acute abdominal pain is a critical sign. The same applies to the respiratory rate.

EXAMINATION OF THE ABDOMEN

Start by examining the area of the abdomen furthest from the site of maximal pain. Examine for:

- distension
- rigidity
- area of maximum tenderness and rebound tenderness
- masses
- shifting dullness
- peristaltic sounds (normal activity is 10–20 sounds)
- rectal and vaginal abnormalities.

Distension

When the patient lies flat in bed, the normal abdominal contour is scaphoid or flat, even in the very obese. The presence of distension points to obstruction, ileus or the presence of fluid.

Rigidity

Rigidity can be assessed by placing a pillow under the patient's knees and letting the patient breathe gently and deeply through the mouth. Board-like rigidity points to perforated peptic ulcer. Intense bilateral guarding is suggestive of diffuse peritonitis.

Tenderness

Observation of the patient's facial expression during palpation can

help in localizing the area of maximal tenderness. It is better to avoid eliciting rebound tenderness, which can sometimes be very painful to the patient. Generally, the abdominal findings of peritoneal irritation are constant and reproducible, whereas those of gastroenteritis are variable.

Masses
Search for appendicular mass or abscess, gallbladder, intussusception. An occult incarcerated hernia in the umbilical and inguinal regions and under previous incisions is important.

Shifting dullness
In the presence of distension, percuss to distinguish whether it is due to gas or fluid.

Peristaltic sounds
Normal activity is 10–20 sounds per minute. In the presence of peritonitis, appendicitis or diverticulitis sounds disappear or become markedly hypoactive. The bowel sounds are hyperactive in inflammatory disease of the intestinal tract, early intestinal obstruction and subsiding paralytic ileus. It must be remembered that, with acute inflammations anywhere in the body, severe metabolic disorders (diabetes, uraemia, coma, shock) can cause moderate to severe ileus with loss (but rarely complete loss) of peristaltic sounds.

Rectal or vaginal examination
This must be performed in every patient with acute abdominal pain. It may disclose pelvic abscess, appendicular masses or parametrial inflammation. Testing of stools on the examining finger for occult blood should also be done.
Note: Hourly examination of the patient is mandatory after installing a Ryles nasogastric tube and i.v. line.

Laboratory test

Blood leucocytic count (total and differential). Leucocytosis can be an important clue to abdominal inflammation. Serial white blood cell counts can be very helpful when the diagnosis is uncertain.

Urine analysis, especially sugar and acetone. Ketosis may give signs and symptoms that mimic acute surgical emergencies.

Serum and urine amylase can reveal acute pancreatitis. Values over 400 units/100 ml^3 serum or over 700 units in a single urine specimen are diagnostic.

X-ray studies

- Flat X-ray of the abdomen will show renal and gallstones, subphrenic air.
- Upright X-ray of the abdomen will show fluid levels, masses, subphrenic air.
- Upright X-ray of the chest will exclude pneumonia, diaphragmatic hernia and detect subphrenic air, suggesting perforation.

Other tests

Ultrasonography and rapid CT scanners are helpful means of diagnosis where available.

Diagnostic abdominal paracentesis and lavage paracentesis are useful (see Abdominal injuries below). It is of great importance to know that a negative or dry tap has no significance and can be ignored. It is common to fail to aspirate fluid, even after several needle insertions into the peritoneal cavity and despite the presence of a considerable amount of fluid. Paracentesis should not be done until after an abdominal X-ray has been obtained, since small amounts of air may be introduced, leading to erroneous diagnosis of perforation. The aspirated fluid should be analysed by the laboratory for the following:

- pH, which is acid in perforated gastric ulcer;
- Bile, for perforated duodenal ulcer;
- Amylase, for pancreatitis;
- Blood, erythrocytes and leucocytes (500–700 polymorphs/ml^3), for peritonitis;
- Protein concentration above 3 g/100 ml^3 for exudate of inflammation or neoplastic involvement of the peritoneum.

Operative intervention in the form of exploration still has its place in cases where a diagnosis cannot be reached and where it is thought that further waiting may endanger the patient's life.

6.2 FOOD POISONING

Food poisoning includes a number of disorders presenting with diarrhoea, vomiting and abdominal colic due to gastroenteritis and developing up to 48 hours after the consumption of contaminated food or drink. Food poisoning is commonly bacterial in origin, but may be viral or due to intestinal allergy, e.g. to shellfish or unripe fruits. It may also be due to poisonous chemical

material consumed accidentally or unknowingly, e.g. arsenic. Bacterial food poisoning is usually divided into infective and toxic.

6.2.1 INFECTIVE BACTERIAL FOOD POISONING

The organism most commonly responsible belongs to the *Salmonella* group, S. *typhimurium*. Pets (dogs, cats, turtles) and domestic animals (birds, chickens, turkeys, pigs and goats) are important reservoirs for these organisms.

Contaminated drink or food (raw or uncooked meat, meat and fish by-products, egg and its products, creams, milk, dried or frozen foods) and person-to-person contact are common sources of infection. Food may be contaminated with infected excreta of mice or rats, or infection may be transferred by flies or human carriers employed in the handling of food. The size of the infecting dose of bacteria bears a direct relationship to the speed of onset and the severity of the symptoms.

After an incubation period as brief as 10 hours, fever, chills, nausea and vomiting, headache, colicky abdominal pain and watery diarrhoea with mucus and perhaps blood may begin abruptly.

If the patient is toxic, hepatosplenomegaly is present, but otherwise enlarged liver and spleen are rare. Skin eruptions (rosy red spots, 2–4 mm in diameter) may appear on the chest and abdomen, but can easily be missed.

Treatment

In the majority of cases, food poisoning is a mild self-limiting infection and an uneventful recovery can be expected within 2–3 days. It can be treated at home with oral fluids only. A teaspoonful of salt added to a pint of water and flavoured with fruit juice provides a satisfactory replacement.

Patients who are severely ill, collapsed or dehydrated require i.v. fluids and electrolytes (lactated Ringer's solution) and should be admitted to hospital where they can also receive antibiotics if *Salmonella* bacteraemia is confirmed (ampicillin lg hourly or chloramphenicol 2–5 g every 24 hours. Otherwise, antibiotics and antidiarrhoeals should not be given for acute vomiting and diarrhoea, as they are ineffective and frequently exacerbate symptoms.

6.2.2. TOXIC BACTERIAL FOOD POISONING

Toxic food poisoning is most commonly caused by the enterotoxin

of *Staphylococcus aureus*, which frequently originates in a food handler suffering from a specific lesion. Incubation at a suitable temperature leads to growth of the organism and production of toxin, which is relatively heat-resistant and may not be destroyed by cooking. Some strains of *Clostridium perfringens (Cl. welchii)* are resistant to heat and may contaminate certain foods, particularly meat. Precooking of some foods may not destroy all spores and the keeping of such foods will lead also to the formation of heat-stable toxins, which can give rise to severe gastroenteritis. The severest form of food poisoning is produced by the most potent poison known to man, namely the toxin produced by *Clostridium botulinum*.

The symptoms of toxic bacterial food poisoning are the same as those of the infective type, namely acute abrupt onset of vomiting, diarrhoea and central abdominal colic, although the latter is more prominent with the infective type than with *Staphylococcus aureus* and, in severe cases, prostration and collapse. Unlike the infective type, symptoms develop earlier, within 1 hour. It should be noted that if vomiting starts within 30 minutes of the ingestion of suspect food, it is likely to be due to a chemical poison rather than to an infection (see Chapter 12).

The clinical features of poisoning with *Clostridium botulinum* toxin differ from all other types of bacterial food poisoning. They consist chiefly of severe vomiting, constipation, thirst, the secretion of viscid saliva, and ocular and pharyngeal paresis and aphonia. Mortality is very high.

Treatment

Treatment is symptomatic only, with attention to dehydration. If the poisoning is thought to be due to a chemical or a poisonous food, the patient's stomach should be washed out with tepid water.

Note: Notification of food poisoning to health authorities is compulsory.

6.3 OESOPHAGEAL VARICES

Oesophageal varices develop as collateral pathways when the normal channels for portal blood flow are compromised. Variceal haemorrhage occurs when elevation of the portal venous pressure produces a tear in the varix wall.

The most common cause for an increase in portal pressure in a patient with liver disease is hepatic decompensation, a fact that is reflected in the observation that patients who bleed from oesophageal varices have usually developed ascites prior to the occurrence of haemorrhage.

Diagnosis

An oesophagoscopy is the best diagnostic approach. If the haemorrhage is profuse, coeliac arteriography has proved to be an effective diagnostic approach.

Management

As initial therapy:

1. Initiate blood replacement and gastric lavage with ice water.
2. Consideration should be given to the state of liver function, signs of encephalopathy, renal function, electrolyte and acid–base balance, and portal pressure.
3. Administration of neomycin may be necessary.

If the initial therapy does not succeed in stopping the haemorrhage:

1. Initiate a trial of vasopressin, by arterial catheter into the mesenteric system.
2. Install a Sengstaken-Blakemore tube to reduce the risk of aspiration.

If the therapy outlined above fails, management must be individualized:

1. Install an emergency portacaval shunt.
2. Perform oesophageal tamponade (in severely compromised liver functions).

These lines of management can, of course, only be carried out in a hospitalized patient.

6.4 ABDOMINAL INJURIES

Any patient involved in a road traffic, industrial or sports accident should be considered to have an abdominal injury until proved otherwise. Serious intra-abdominal injury can occur from very minor trauma.

Patients with blunt abdominal trauma are difficult to evaluate. The signs of their injury can be delayed and the consequences of

injury, particularly to solid organs, can be difficult to manage. Signs and symptoms of intra-abdominal injury can be masked by injuries elsewhere. The presence of fractured ribs with secondary splinting makes examination of the abdominal wall difficult. A serious central nervous system injury can also mask abdominal findings. Decisions must be based on repeated examinations, especially during resuscitation, since shock may mask abdominal findings. Frequent examinations should be made by the same physician, until it can be ascertained definitely that significant visceral injury, peritonitis or haemorrhage is not present.

Rupture of a hollow viscus usually produces signs of peritoneal irritation and loss of bowel sounds. These signs may not be present on initial examination, and some patients with small bowel and bladder injuries may show surprisingly minimal early signs and must be re-evaluated frequently.

The patient with injury to a solid viscus, such as the liver or spleen, usually presents with haemorrhage. Peritoneal irritation may occur when blood is present within the abdominal cavity. A trauma victim presenting with unexplained hypovolaemic shock should be assumed to have an intra-abdominal injury.

Organ enlargement, particularly if secondary to other pathological conditions (e.g. lymphoma), makes the organ more susceptible to injury. A distended urinary bladder or pregnant uterus is at increased risk of injury from blunt trauma to the abdomen.

Signs and symptoms

Pain following abdominal trauma may result from abdominal wall injury or injury to the underlying structure. Pain referred to the shoulder is seen with diaphragmatic irritation secondary to splenic or hepatic injury. Patients who have pain and other abdominal signs should not be given narcotics or other analgesics until a decision about the need for an operation has been made.

Localized tenderness or abdominal wall rigidity is a result of peritoneal irritation from blood or hollow viscus contents, and is usually an indication for exploration. Abdominal guarding during palpation makes evaluation of the abdomen difficult, particularly in anxious or uncooperative patients. This is particularly true if there is associated chest, spinal or pelvic injury. Repeated gentle examinations are helpful. If rib fractures are present, intercostal nerve blocks will decrease pain and may help in evaluation of

the abdomen. Absence of tenderness and rigidity is no assurance that intra-abdominal injury is absent.

Abdominal distension is always an ominous sign. If it occurs in a patient with a penetrating wound, there is probably injury to the liver, spleen or a major vessel. In blunt trauma, abdominal distension may be due to ileus secondary to retroperitoneal injury, especially involving the pancreas, or to spinal injury.

The absence of bowel sounds (5 min), particularly in patients with seemingly shallow penetrating wounds, is an indication for exploratory laparotomy.

Inability to resuscitate a hypovolaemic patient with suspected abdominal injury is an indication for rapid operative intervention and direct control of the haemorrhage. If the patient has multiple fractures and a great deal of soft-tissue injury, the resuscitative effort must be vigorous before it can be concluded that continued intra-abdominal haemorrhage is present.

Radiological studies

ROUTINE X-RAY VIEWS

At an appropriate time during the resuscitative effort, flat and upright abdominal and chest X-rays should be obtained. Every effort should be made to obtain an upright chest X-ray, as supine films of the chest are difficult to interpret. Diagnostic features to look for include free air in the peritoneal cavity, retroperitoneal air (especially near the duodenum), elevation of the diaphragm, obliteration of the psoas shadows, displacement of the gastric air bubble, disturbances of normal bowel patterns, and the presence and location of foreign bodies.

ROUTINE ABDOMINAL SONOGRAPHY AND/OR CT SCANNING

Ultrasonography and CT scanning have gained in popularity and have proved very helpful in patients with abdominal injury who have not been operated on and show delayed effects of their intra-abdominal injuries, particularly injuries to the liver and retroperitoneum. They have the advantage that they can be safely repeated to follow up the case. Where available, they should be done on all patients suspected of having an intra-abdominal injury.

OTHER STUDIES

The IVP is useful in demonstrating kidney injury and is mandatory to assure the presence of a functioning contralateral kidney if nephrectomy is necessary. In patients who present with gross blood at the urethral meatus, a urethrogram should be obtained prior to catheterization. Selective angiography may be useful in patients with blunt abdominal trauma whose initial diagnostic tests are inconclusive. Selective coeliac angiography is particularly useful in demonstrating subcapsular splenic injuries. Upper gastrointestinal tract barium studies may be helpful in establishing gastric displacement by an enlarged spleen, and in demonstrating a duodenal intramural haematoma. Unfortunately, on occasion, a barium swallow also demonstrates a retroperitoneal rupture of the duodenum.

Laboratory examination

Although haemoglobin and haematocrit determinations are of little value in the initial appraisal of the patient with blood loss, they can be useful during the observation period to detect continued blood loss.

A leucocyte count in excess of 20 000/mm^3 in the absence of infection is suggestive of significant blood loss, and is particularly useful in supporting an early diagnosis of rupture of the spleen.

Elevation of serum amylase suggests pancreatic injury or bowel rupture. Elevation of transaminase suggests hepatic injury.

Other studies

Abdominal paracentesis is helpful in determining the presence of blood due to intra-abdominal injury. Peritoneal lavage is the preferred technique. A sample of the effluent is analysed for red blood cells: more than 100 000 cells/mm^3 is an indication for exploratory laparotomy. The effluent can also be tested for bile, leucocytes, bacteria and amylase. However, the test is not always diagnostic. Significant intra-abdominal injury can be present in spite of negative results.

If rectal bleeding is seen or blood is present on the examining finger, the possibility of rectal injury should be evaluated by proctosigmoidoscopy. Rectal examination is mandatory.

A nasogastric tube should be inserted in all patients suspected of having abdominal injury. The presence of blood in the aspirate

may mean that an injury to the upper gastrointestinal tract has occurred.

Emergency management

Open wounds caused by bullets, shotgun blasts, large knives or similar means are an indication for abdominal exploration. If the injury is associated with shock or abdominal distension, the abdomen should be explored immediately the condition has been stabilized.

Small open injuries to the anterior abdominal wall in which penetration of the peritoneal cavity is unlikely can be treated expectantly. Anticipate the possibility of peritoneal injury. If there is any sign of peritoneal irritation, such as tenderness, rigidity or absence of bowel sounds, the abdomen should be explored. The safest manner of caring for these patients is to explore the injury directly under local anaesthesia in the operating room. If there is evidence that the peritoneal cavity has been entered, abdominal exploration under general anaesthesia can be carried out. In most instances, it is safe simply to observe patients with stab wounds of the anterior abdominal wall who present without any physical signs.

Patients with blunt trauma of the abdomen are treated according to the symptoms, signs and results of other examinations. A positive paracentesis is an explicit indication for laparotomy. Patients with minimal findings at the time of initial examination, but with the suspicion of significant abdominal trauma still present, should be admitted to hospital for observation. During this observation period, the patient should be examined frequently by the same examiner, X-ray and sonography repeated, and a senior surgeon consulted.

Indications for exploratory laparotomy in blunt abdominal trauma include:

- persistent abdominal wall tenderness or rigidity;
- unexplained, even if minimal, persistent findings on repeated examination of the abdomen;
- appearance of signs of shock or blood loss;
- positive radiological or laboratory findings.

ROUTINE PREOPERATIVE PROCEDURES FOR EXPLORATION

In addition to the general principles applying to all patients

undergoing an operation, the following steps should be taken for patients being explored for possible intra-abdominal injury:

1. Install nasogastric suction.
2. Place an indwelling urinary catheter.
3. Give parenteral antibiotics to patients with signs of gastrointestinal tract injury, severe shock or massive trauma.
4. Insert a chest tube in patients with rib fractures or with even minimal pneumothorax or haemothorax.

Specific injuries

ABDOMINAL WALL

Blunt trauma can cause injury to the abdominal wall without causing intra-abdominal injury. Musculature can be avulsed or major vessels transected. Rigidity, tenderness and a palpable mass can result, for example from a rectus haematoma. Any mass within the anterior abdominal wall remains easily palpable when the patient raises the head, tensing the abdominal muscles, whereas this manoeuvre usually causes an intraperitoneal mass to become less palpable.

SPLEEN

The spleen is the most frequently injured intra-abdominal organ. A ruptured spleen is suspected if there has been trauma to the left side, especially if ribs are fractured.

The clinical findings and evidence of hypovolaemia range from minimal to profound. Pain referred to the left shoulder is common. Other useful findings include leucocytosis, displacement of the gastric air bubble, and the presence of blood on paracentesis. In doubtful cases, selective coeliac arteriograms can be helpful. Delayed rupture of the spleen should be suspected in a patient who has sudden abdominal pain and signs of hypovolaemia occurring within 4 weeks of an injury.

LIVER

The liver is the largest intra-abdominal organ, and the magnitude of parenchymal damage can range from minimal to almost total destruction. Management is left to the surgeon's judgement during exploration, which is mandatory once liver injury is diagnosed.

PANCREAS

Unless there has been a significant rise in serum amylase noted prior to operation, injury to the pancreas is usually detected at the time of exploration.

GALLBLADDER AND BILIARY TRACT

Injuries to the biliary tract are usually caused by penetrating wounds, although the gallbladder can be devascularized by blunt injury. An injured gallbladder must be excised. Injury to the extrahepatic biliary ductal system is usually detected at laparotomy by the presence of bile-stained tissues.

STOMACH

Gastric injuries should be suspected if nasogastric suction reveals the presence of blood.

DUODENUM

Intraperitoneal duodenal injury may be suspected if bile or small-bowel contents are recovered by paracentesis. Retroperitoneal injury to the duodenum is more frequent than intraperitoneal injury.

SMALL INTESTINE

Small-bowel injury should be suspected in any patient with penetrating abdominal injury. In blunt trauma, small-intestine injury usually occurs at or near sites of mesenteric fixation. Signs of peritoneal irritation are usually present, and small-bowel contents are sometimes recovered on paracentesis. Such symptoms may not appear early on, hence the importance of placing the patient under observation.

COLON

Patients with colon injury may present with signs of peritonitis, or an upright X-ray may show free air. A barium enema should **never** be given in patients suspected of having colon injury. Preoperatively, vigorous fluid replacement and systemic antibiotics are required. Such injuries may be missed and only discovered when infective complications appear.

FEMALE REPRODUCTIVE ORGANS

Injuries to the female reproductive organs usually occur in pregnant women and may cause sudden vaginal haemorrhage following blunt trauma. Hysterectomy or removal of injured adnexa may be necessary. Salvage of a pregnancy depends on gestational age and degree of fetal injury.

6.5 UROLOGICAL INJURY

Injury to the genitourinary tract rarely occurs as an isolated lesion. These injuries tend to occur as one aspect of a larger problem in the patient with multiple injuries. Blunt trauma from road traffic accidents and penetrating injuries from bullet and knife wounds account for the majority of cases. The immediate goal in management of these injuries is to prevent or treat hypovolaemic shock and stop major bleeding if the patient's condition remains unstable. A secondary goal is to prevent urine collection and abscess formation by properly draining urinary extravasations.

The role of surgical intervention in the management of renal and urethral injuries has been changing over the past few years. Many urologists now believe that conservative management of these injuries results in less long-term morbidity and functional disability.

Diagnosis

The history may be very useful in helping to establish the diagnosis. Inability to void or severe pain on attempting to void suggests partial or total separation of the urethra. It is important, but sometimes difficult, to get a proper history from these patients. Physical examination should be directed to detection of injuries in three major areas:

- Upper urinary tract injuries are often associated with rib fractures over the involved kidney. An expanding flank mass (found by abdominal palpation) with hypovolaemic shock indicates a major renal injury.
- Lower urinary tract injuries are often associated with blunt trauma to the lower abdomen and with fractures of the bony pelvis. Blood at the urethral meatus is an important sign of lower urinary tract injury.
- A suprapubic mass from a haematoma, or urinary extravasation and haematoma in the perineum or scrotum, are often found.

Rectal examination may reveal a doughy mass in the region of the prostate from a large pelvic haematoma.

Urinanalysis may be helpful in establishing the diagnosis of urinary tract injury. However, trauma to the urinary tract will not result in gross or microscopic haematuria in all patients. This fact should be kept clearly in mind if the physical examination and other studies reveal a urinary tract injury even though the urinanalysis is negative.

Plain abdominal X-rays should be carefully evaluated for loss of psoas shadow, fractures impinging on urinary structures, foreign bodies lying in proximity to the genitourinary systems, kidney outlines, and free air or fluid in the abdomen.

Ultrasonography, being a non-invasive procedure, should be done whenever available. Kidney and bladder collections can be identified clearly in many cases.

An infusion IVP (intravenous pyelogram) should be carried out in all patients suspected of having a genitourinary injury. This should be performed using the i.v. line that is set up when the patient enters the emergency room. In shock, the kidney may not be visualized, but the IVP remains an important radiological study of the urinary tract in trauma. To facilitate visualization of the kidneys, hypovolaemia should be corrected with fluid or blood replacement rather than vasopressors because the latter will diminish renal blood flow and decrease the likelihood of visualization.

Retrograde urethrograms are essential in defining urethral injuries. Retrograde injection of 15–30 ml of sterile contrast medium will define the urethral injury as evidenced by extravasation of contrast material, and should precede catheterization of the urethra.

A urethral catheter is usually required early in the management of the patient with multiple trauma, to assess renal function and the effect of treatment of hypovolaemic shock. However, great care should be taken not to convert a partial tear of the urethra into a complete tear by traumatic catheterization. If there is blood at the urethral meatus or other signs of urethral injury, every attempt should be made to perform a retrograde urethrogram before inserting a catheter. A carefully placed catheter may be all the treatment necessary in many patients with urethral injury with minimal extravasation of urine. If the urinary tract becomes infected after catheterization, an appropriate antibiotic should be started. This is particularly important if extravasation has

occurred into a large pelvic haematoma and there is risk of infecting the haematoma.

A cystogram is essential for diagnosis of a ruptured bladder. Instillation of 50–300 ml of diluted i.v. contrast material should be done through a urethral catheter. Anteroposterior, lateral, oblique and post-emptying X-rays are all necessary to detect extravasation. An extraperitoneal rupture of the bladder will show extravasated contrast material in the perivesical space: with an intraperitoneal rupture, contrast material is seen in the peritoneal cavity, often between loops of small bowel.

Renal arteriograms are needed to assess the extent of renal injury and the possible congenital absence of one kidney if the kidneys are not visualized during intravenous pyelography. Arteriography is also useful for outlining the branches of the renal artery when it is necessary to operate on an injured kidney with urinary extravasation or massive renal bleeding, or both. The extent of other intra-abdominal injuries, such as tears in the spleen and liver, can often be visualized at the same time.

Treatment

EXTERNAL GENITAL INJURIES

Injuries to the penis usually result in the loss of penile skin and, in some cases, in partial amputation of the penis. This should be treated by debridement.

Injuries to the introitus and urethra in the female are usually straddle injuries caused by a fractured pelvis. Lacerations should be sutured and the urethra carefully catheterized to preserve urinary continence.

For a female presenting with bleeding due to circumcision the following procedure should be followed:

1. Insert a catheter.
2. Suture the lacerations.
3. Give antibiotics.

URINARY TRACT INJURIES

Urethral injuries in the male occur in three separate areas: the pendulous urethra; the bulbous urethra below the genitourinary diaphragm; and the supramembranous urethra.

Injuries to the pendulous urethra result in haematoma and urinary extravasation and are usually best treated with an indwelling catheter for 10 days to 2 weeks.

Injuries to the bulbous urethra are most often straddle injuries. Urinary extravasation and haematoma may be present in the perineum and scrotum, and on occasion may be seen on the abdominal and chest walls, as the fluid dissects upward beneath Scarpa's fascia. Treatment consists of gentle placement of an indwelling catheter or placement of a suprapubic tube into the bladder if the urethral catheter will not pass with ease.

Injuries to the supramembranous urethra usually occur as a tear or complete separation at the level of the apex of the prostate and the genitourinary diaphragm. These injuries are almost always associated with pelvic fractures. Large pelvic haematomas develop that are evident on rectal and suprapubic palpation.

Bladder injuries occur as extraperitoneal and intraperitoneal tears of the bladder wall. Intraperitoneal rupture must be closed surgically, preferably in three layers. Extraperitoneal rupture can be managed by an indwelling catheter only if the amount of extravasation seen on the cystogram is small. Large tears should be sutured as well as drained.

Injury to **the kidney** may occur as blunt trauma or as a penetrating injury. Blunt trauma results in contusion of the kidney and, occasionally, in fracture of the parenchyma. Avulsion of the renal artery and thrombosis may also occur with blunt trauma. Penetrating injuries usually cause bleeding with formation of a retroperitoneal haematoma. Penetrating injuries may also damage the urinary collecting system and cause urinary extravasation. As previously noted, the IVP and arteriogram are both important in defining the location and extent of renal injury.

The method of treatment of renal injury depends on three important factors: severity of renal bleeding, as evidenced by stability of blood pressure and size of retroperitoneal haematoma; amount of extravasation of contrast material during the IVP; and severity of major renal parenchymal and arterial injury as demonstrated by IVP and arteriograms.

Patients with stable blood pressure, stable size of flank mass, and little or no extravasation of contrast material on IVP should be managed conservatively.

Once a urinary tract injury has been diagnosed, always consult a urologist.

7

Central nervous system emergencies and head injuries

Summary of contents

CENTRAL NERVOUS SYSTEM EMERGENCIES 164

Status epilepticus 164
Coma 165
Syncope 174
Transient ischaemic attacks 174

PSYCHIATRIC EMERGENCIES 177

TOXIC CONFUSIONAL STATE DUE TO ACUTE ALCOHOL WITHDRAWAL 181

THE UNCONTROLLED AND POTENTIALLY HOSTILE PATIENT 183

HEAD INJURIES 185

Brain injury 186
Concussion 186
Contusion 186
Cerebral irritation 186
Cerebral compression 186

Management 187

7.1 CENTRAL NERVOUS SYSTEM EMERGENCIES

7.1.1 STATUS EPILEPTICUS

Status epilepticus is defined as a state of recurring generalized or lateralized convulsions without an intervening period of recovery;

the term is also applied to children suffering from multiple recurring attacks within a short period of time. Petit mal status epilepticus is characterized by a prolonged blurring of the state of consciousness, lasting from minutes to hours, during which time there may be repetitive movements of the mouth and eyelids. It is a true medical emergency, and every effort should be made to control the seizures as soon as possible.

Aetiology

In the absence of a proper history, the following most likely causes of seizures, depending on age, should be considered:

NEWBORN TO 2 YEARS OLD

- Intracranial birth injuries
- Congenital defects
- Sepsis and meningitis
- Metabolic disorders (such as hypoglycaemia, hypocalcaemia and hyponatraemia)

2–5 YEARS OLD

- Infection
- Fever
- Trauma
- Metabolic disorders
- Meningitis
- Idiopathic conditions

5–12 YEARS OLD

- Genetic or idiopathic conditions
- Chronic brain damage (traumatic or other cause)
- Infection
- Meningitis

12 YEARS OLD AND OVER

- Genetic or idiopathic conditions
- Chronic brain damage or old trauma
- Acute head trauma
- Metabolic disorders

- Neoplasm
- Meningitis

Status epilepticus occurs in 5–10% of children with epilepsy, and may result in postepileptic paralysis or organic psychosis.

Management

Admit the patient to the intensive care unit of the hospital. Take the following steps in the emergency room:

1. Maintain a clear airway by removing foreign bodies or suctioning the secretions.
2. Give oxygen to prevent hypoxic damage to the CNS.
3. Begin i.v. hydration.
4. If the patient's seizures are in any way violent, place a soft resilient cushion under his or her head and body. Use only minimal restraint.
5. Begin suppressive therapy. Control of convulsions is important and requires the immediate use of i.v. anticonvulsants:

- diazepam
- amobarbital sodium
- paraldehyde

If the medications listed above fail to give control, general anaesthesia is indicated. However, this requires intubation for maintenance of the airway and should **not** be attempted without the proper facilities.

7.1.2 COMA

The word coma is used to describe a depressed level of awareness from which the individual cannot be aroused to a usual level of responsiveness. The comatose patient is unable to perceive or respond adequately to internal or external stimuli.

Pathophysiology

In a comatose patient there is no evident appropriate response to exogenous or endogenous stimuli, response which is normally based on the capacity of brainstem reticular formation to maintain arousal in the integrating function of the cerebral cortex. Coma may result from lesions in the midbrain and upper pons, whereas

bilateral cortical damage may interfere directly with integrative capabilities, with altered awareness. Depression of both the cerebral cortex and ascending reticular activity by toxic–metabolic derangements may cause altered arousal function and impairment of integrative function.

TRANSTENTORIAL HERNIATION

Transtentorial herniation (TTH) is a pathological extrusion of brain tissue from one compartment to another within the skull, caused by increased pressure and resulting in the compression of vital centres. Although TTH poses a grave immediate hazard, it is potentially reversible early in its course. It leads to death if not treated effectively.

In the emergency room the physician has to proceed on the assumption that coma and neurologic deficits referrable to the brainstem are due to herniation, and therapy to decrease intracranial tension (ICT) must be instituted immediately.

Initial assessment

Initial assessment of the comatose patient should include a detailed history, complete physical examination, and diagnostic procedures. However, initial management, and sometimes empirical therapy, should be instituted before history taking or complete examination are performed.

HISTORY

Abrupt or insidious onset

A slowly expanding mass gives a history of progressive confusion or hemiparesis. Hypertensive haemorrhage or subarachnoid haemorrhage are associated with abrupt onset of coma.

Recent trauma

Massive trauma may result in immediate coma. Epidural or subdural haematoma will result in coma after a short period.

Cardiovascular state

Cardiac or pulmonary disorders are possible aetiologies of coma, e.g. atheromatous arterial disease, hypertension, myocardial infarction, arrhythmias.

Metabolic disorders
Diabetes mellitus, renal failure, adrenal insufficiency and metastatic carcinoma all increase the likelihood of severe metabolic derangements and, eventually, coma.

Known seizure disorder
Known seizure disorders, e.g. epilepsy or transient ischaemic attacks (TIAs), may be followed by coma (postictal coma).

Anticoagulation and clotting disorders
Patients on oral anticoagulants or with clotting disorders, e.g. thrombocytopenia, are at great risk of intracranial bleeding.

Likelihood of intoxication or attempted suicide
Specific inquiries should be made into psychological background, and drug history should be sought.

INITIAL THERAPY AND PHYSICAL EXAMINATION

Certain supportive measures and some empirical therapy must be initiated immediately the examination begins. The procedure may vary from patient to patient, but the neck must first be stabilized to avoid exacerbating any cervical injury and the patient placed in the coma position. As soon as an airway is assured, blood pressure and heart rate, and external evidence of blood loss should be checked to eliminate hypovolaemic shock. Take venous blood samples for diagnostic studies, establish an i.v. line, inject glucose and thiamine. Proceed carefully to cardiopulmonary auscultation and then to neurological examination.

A detailed physical examination of a comatose patient is essential: the following scheme is an initial approach. It should be noted that the presence and distribution of neurological deficits referrable to the brainstem are of critical importance. Special attention must be paid to the respiratory pattern, eye movements and oculocephalic and oculovestibular responses.

1. Establish a clear airway. Ensure the head is turned to one side to prevent inhalation pneumonia.
2. Observe the pattern of respiration and spontaneous movements.
3. Stabilize the neck and inspect for evidence of head trauma.

4. Check arterial blood pressure and heart rate. Take blood samples. Establish an i.v. line. Give glucose 5% and thiamine. Do not infuse more than 100 ml/h until the possibility of cerebral oedema is eliminated.
5. Look for evidence of other trauma and bruises.
6. Monitor ECG.
7. Install a nasogastric tube and drain gastric contents.
8. Install a Foley catheter and monitor the output.
9. Maintain normal body temperature.
10. Look for a reaction to visual threat.
11. Look for a reaction to painful stimuli to each limb, the chest and the face.
12. Observe pupillary reaction to light.
13. Perform funduscopic examination and inspection of the tympanic membrane.
14. Check gag and corneal reflexes.
15. Check deep tendon reflexes and plantar response.
16. Examine the range of spontaneous eye movement.
17. Test for oculocephalic and oculovestibular reflexes.
18. Test for meningeal irritation.

Reaction to noise and visual threat

Any response to external stimuli serves as evidence that the pathway subserving the sensory modality is functioning, e.g. a facial reaction to auditory stimuli indicates that much of the pons is functioning. A feigning blow to the eye from the midtemporal visual field gives ipsilateral blinking and indicates that the retina, optic nerve, contralateral optic radiation, occipital cortex, corticobulbar tracts, ipsilateral facial motor nucleus and facial nerves are all intact.

Response to painful stimuli

Withdrawal of a limb is the normal response to painful stimuli. This response may be obliterated by spinal shock or severe toxic–metabolic shock. Asymmetry of responsiveness is due to injury somewhere in the corticospinal motor system. Painful stimuli applied to the face, e.g. compressing the supraorbital area with the thumb, is an indicator of vagus nerve function. The response to these stimuli gives a clue about whether the patient is decorticate or decerebrate.

Decorticate posturing is flexion of the fingers, wrist and elbow, adduction of the upper limbs, and rigid extension of the hip and knee with plantar flexion. It indicates lesion in the corticospinal tract at or above the level of the thalamus.

Decerebrate posturing is extension and pronation of the arm with internal rotation at the shoulders, rigid leg extension and plantar flexion due to damage to the rostral half of the pons or to the diencephalon.

Pupillary reflexes

Flashing a bright light into the eyes normally leads to bilateral pupillary constriction. When light is flashed into both eyes but only one pupil constricts, the efferent pathway of the unreactive eye may be damaged. Both sympathetic and parasympathetic pathways are tonically active, and much of the brainstem must be intact to function normally.

Failure of one eye to constrict in response to light indicates TTH of the temporal lobe. Monitoring of shock

Abrupt onset of coma with pinpoint reactive pupils is a sign of pontine haemorrhage.

Fixed, completely unresponsive, halfway dilated pupils indicate a lesion in the midbrain involving the oculomotor nuclei.

A unilateral Horner syndrome of recent onset is ominous because it is often associated with the compression of the hypothalamus during an early phase of diencephalic TTH.

A history of drug abuse should be borne in mind during interpretation of the pupillary response.

Funduscopic examination

The following funduscopic signs are critically important:

- Papilloedema indicates elevated intracranial tension of at least 6 hours' duration.
- Preretinal haemorrhage indicates subarachnoid haemorrhage.
- Arterial tortuosity and narrowing indicates atherosclerosis, cardiovascular disease and raised arterial blood pressure.
- Diabetic retinopathy and neovascularization indicate long-standing diabetes mellitus.
- Central retinal occlusion is due to severe carotid atherosclerosis.
- Dilated blood vessels with perivenous haemorrhage indicate a hyperviscosity syndrome.

Tympanic membrane

The tympanic membrane should be inspected for signs of haemorrhage (skull fracture) or infection (brain abscess).

Corneal and gag reflexes

Corneal and gag reflexes are good indicators of brainstem

function. If the cornea is touched lightly with a cotton wisp it causes blinking of the eyes, which denotes intact pathways of the sensory root of the vagus nerve to the facial motor nucleus. If there is no response massive brainstem damage is indicated, because this reflex is usually preserved even when respiration is depressed to the point of apnoea. On the other hand, the gag reflex is often lost early on in brainstem injury. If the response is hypoactive, then the corticobulbar tract is injured.

Deep tendon reflexes

These are monosynaptic reflexes at the level of the spinal cord, but the amplitude of the motor response is modulated by the corticospinal tracts. Deep tendon reflexes may be active extremis and symmetrical in cases of hypocalcaemia. If Babinski's sign is negative, the corticospinal tract is injured.

Spontaneous eye movements

A lesion in the right frontal lobe can result in sustained involuntary conjugate lateral deviation of the eyes to the right, because the left cerebral cortex is functioning unopposed. A lesion in the lateral gaze centre in the pons prevents gaze to the same side, giving conjugate deviation to the unaffected side. Unless a structural injury has occurred, the eyes of an unconscious patient are usually directed slightly ahead or are slightly divergent. Pontine haemorrhage as a cause of coma can lead to 'ocular bobbing' in which the eyes rest in midposition, intermittently jerk down, and then move slowly back to midposition again.

Oculocephalic and oculovestibular reflexes

Usually, the most useful assessment of brainstem function is based on examination of evoked eye movements. Testing of oculocephalic or 'doll's head' reflexes is straightforward. With the patient's nose and eyes pointing straight up, the examiner briskly rotates the head so that the nose is over the right shoulder. During this passive movement, if oculocephalic reflexes are intact the gaze continues to be directed towards the ceiling. Thus the right eye adducts to the nose (third cranial nerve) and the left eye abducts to the shoulder (sixth cranial nerve). Almost the same system is evaluated in the oculovestibular reflex. The usual response to flooding the external auditory meatus with ice-cold water in a moderately comatose patient is nystagmus, with the fast component towards the opposite ear, while a deeply comatose patient manifests tonic deviation to the irrigated side within 2–3 minutes.

Testing for meningeal irritation

When the meninges are irritated by blood or inflammatory exudate, reflexes emerge to keep the spinal column as short as possible and thus minimize traction on the inflamed meninges. Forced deviation of the neck in the presence of meningeal irritation will sometimes cause the hip to flex, and the neck may extend if the knee is extended.

DIAGNOSTIC INVESTIGATIONS

The following investigations should be carried out immediately for all comatose patients:

- Blood tests: grouping, Na^+, K^+, Ca^{2+}, blood urea nitrogen, glucose, complete blood count, platelet count and prothrombin time.
- ECG: the information obtained is helpful to evaluate various toxic–metabolic causes of coma.
- CT: this is the single most useful test in the evaluation of a comatose patient.
- Skull X-ray: to look for evidence of fracture and verify midline brain structures. Lateral displacement of the pineal body indicates mass above the tentorium. Clinoid resorption indicates sustained increase in intracranial tension.
- Cerebral angiography: to observe calibre and distribution of cerebral vessels.
- EEG: in general, brainwave activity is an indication of cortical function and may be helpful in providing confirmatory evidence of local dysfunction, e.g. compression. It is usually done after CT, X-ray and angiography.

Lumbar puncture permits analysis of CSF and is indispensable in the diagnosis of meningitis. However, it can be fatal in a comatose patient because lowering CSF pressure by puncturing leads to herniation of the cerebellar tonsils and brainstem compression.

TREATMENT OF INCREASED INTRACRANIAL TENSION

1. Install i.v. 20% mannitol infusion 500 ml over 30 minutes to arrest or reverse herniation. More can follow, but first be sure that the patient passes urine.
2. If respiration is already compromised, the patient should be intubated and hyperventilated to $Paco_2$ 20 mmHg.
3. Give dexamethasone 10 mg i.v. to start with, then 4 mg i.v. every 6 hours to reduce brain oedema.

Lumbar puncture to decrease ICT is not recommended, as it may lead to herniation of the cerebellar tonsils through the foramen magnum, or TTH causing acute brainstem compression and cardiopulmonary arrest.

Aetiology

After good history taking and thorough examination combined with diagnostic investigations, the comatose patient should fall into one of four categories (each of which has a set of likely aetiologies):

- Coma with focal neurological deficits and evidence of trauma
- Coma with focal neurological deficits and no evidence of trauma
- Coma without focal neurological deficits
- Coma with meningeal irritation.

COMA WITH FOCAL NEUROLOGICAL DEFICITS AND TRAUMA

Major trauma followed by immediate loss of consciousness may be due to concussion, brain contusion and/or intracranial haematomas. A contused brain gives visual field cuts, pyramidal tract affection, and ophthalmoplegia from brainstem trauma.

The treatment of contusion is the same as for brain oedema. Severe head trauma causing haemorrhage resulting in compression and brain distortion gives TTH. Trauma sufficient to cause an epidural haematoma may often cause immediate loss of consciousness, but a typical history is a person under 35 years of age who has been struck a forceful blow, awakes from a transient spell of unconsciousness, walks away and within 2–4 hours collapses again. Usually the middle meningeal artery has been severed from the beginning. All ages are susceptible to epidural or subdural haematomas, but the combination of brain atrophy and adhesion of the dura to the skull in the elderly makes them vulnerable.

For a proper diagnostic approach a CT scan should be carried out without delay to define the site and size of brain haemorrhage. Skull and spine X-rays will detect associated fractures. If skull X-rays are normal and a CT scan is not available, cerebral angiography should be undertaken. If both CT scan and angiography fail to reveal intracranial haemorrhage, despite a clear history of trauma, proceed to lumbar puncture, ECG and toxic screening to evaluate the possibilities of meningitis, seizure activity or intoxication.

Initial treatment
If a traumatized patient develops signs of brainstem damage, assume there is herniation. Start mannitol infusion and intubate for hyperventilation. Failure of osmotic therapy and hyperventilation to arrest neurological deterioration forces the last resort of using empirical burrholes to evacuate haematomas. This is justified to evacuate presumed subdural or epidural haematoma if the trauma occurred more than 24 hours before the onset of stupor or coma.

COMA WITH FOCAL NEUROLOGICAL DEFICITS WITHOUT TRAUMA

This includes the widest range of aetiologic possibilities. The principal diagnoses are:

- Hypertensive haemorrhage
- Intracranial mass lesion
- Brainstem ischaemia or infarction
- Hemispheric infarction
- Encephalitis
- Unrecognized trauma.

If the examination discloses deficits referrable to the brainstem, begin empirical therapy for TTH. A CT scan and skull X-ray should be obtained.

Further diagnostic steps and initial therapy in presence of a mass lesion
It is fair to assume that a mass sufficient to cause coma will also cause TTH or cerebellar herniation if a lumbar puncture is performed. The safest approach is to initiate mannitol therapy and hyperventilation, and to perform lumbar puncture in the operating room after drilling a burrhole – this precaution facilitates ventricular drainage.

Diagnosis in presence of metabolic derangements
The mechanism of coma with focal deficits and metabolic derangements is not thoroughly understood. There may be certain areas in some individuals that are predisposed to poor function and that may exhibit a disproportionately severe deficit after a diffuse toxic shock. Such a predisposition may stem from sites of previous cortical infarcts, healed contusions, seizure foci or very old subdural haematoma.

COMA WITHOUT FOCAL NEUROLOGICAL DEFICITS

Metabolic derangement or intoxication is most often the cause of coma without focal signs. Examples include hypoglycaemia, renal failure, ketoacidosis, hypoxia, adrenal insufficiency, hypocalcaemia or hypercalcaemia, hyperviscosity, hypothermia or hyperthermia.

If no clear evidence of intoxication is obvious in 2–4 hours, skull X-rays should be obtained after a CT scan. Unless the results contraindicate lumbar puncture, it should be the next test since meningitis is still a possibility. If CSF is under normal pressure and has normal constituents, the aetiology of coma is almost certainly metabolic, toxic or postictal. Take samples of urine, blood and gastric aspirates for toxic evaluation.

COMA WITH MENINGEAL IRRITATION

Meningitis and subarachnoid haemorrhage are the principal causes of this category. Lumbar puncture and cellular examination of CSF are the best diagnostic procedures. If CSF is normal, subsequent evaluation of possible metabolic, toxic or postictal causes is justified.

7.1.3 SYNCOPE

Syncope, or fainting, is a sudden temporary loss of consciousness. It is most usually caused by cerebral hypoxia secondary to inadequate cerebral blood flow.

Simple syncope, or vasovagal syncope, is the most common type of fainting and may occur in perfectly healthy individuals. It is usually precipitated by some stress (e.g. pain, fright, the sight of blood). Simple syncope is more apt to occur when the patient is sitting or standing, and consciousness rapidly returns when the patient becomes, intentionally or otherwise, horizontal. The simple faint may occur without warning or may be preceded by a brief period of symptoms, including pallor, weakness, cold sweat, nausea, abdominal discomfort and blurred vision. There is tachycardia and the pulse usually slows to 50 or less.

7.1.4 TRANSIENT ISCHAEMIC ATTACKS

Diagnosis

Transient ischaemic attacks (TIAs) are episodes of transient neurological deficit. They may be recurrent, may last only a few minutes

or longer, and are due to a temporary reduction in blood supply to part of the brain. The importance of recognizing TIAs is that they are followed by major stroke with a frequency of about 5% per annum, particularly if the TIA is in the carotid territory.

Causes

- Emboli arising from atheroma of the vertebral and carotid arteries, or their branches, or from the heart are the single most important cause of TIA.
- In the setting of borderline local cerebral perfusion, transient reduction in overall cerebral blood flow may cause significant temporary local ischaemia. This can occur on the basis of:
  a fall in perfusion pressure due to: hypotension e.g. hypotensive drugs; decreased cardiac output (e.g. arrhythmias); increased viscosity due to a PCV of above 50%; paraproteinaemia.
- Transient reduction in local blood flow, which may be the result of any of the following:
  Hypertension. Focal neurological deficit may occur as part of hypertensive encephalopathy.
  Migraine. Complicated migraine may occasionally cause hemiplegia characterized more by dysaesthesia than weakness, or a third or sixth nerve palsy (ophthalmoplegic migraine). It is usually possible to elicit a history of previous attacks of classic migraine.
  Mechanical effects on flow:
    Neck movements may cause occlusion of the vertebral arteries, with ensuing posterior cerebral and brainstem ischaemia. Failure of autoregulation of the posterior cerebral circulation may cause the structures so supplied to be vulnerable to changes in the systemic circulation. This may play a role in vertebral basilar insufficiency.
    Subclavian steal. In this condition, movement of the arms diverts blood from the vertebral arteries, causing symptoms of transient brainstem ischaemia.
- Lack of nutrients, which presumably causes focal symptoms on the same basis as above.
- Anaemia. Haemoglobin of less than 7 g/100 ml may be the sole cause of TIA.
- Hypoglycaemia. This may rarely present itself with a focal neurological deficit.

In a proportion of cases no cause can be found, presumably because of lysis of the vascular obstruction or because the vessel involved is too small to be identified.

TIAs should be differentiated from focal epilepsy and Todd's paralysis. In focal epilepsy, the patient often complains of positive symptoms (e.g. paraesthesia, spontaneous movements); these are uncommon in TIAs. Todd's paralysis is the transient focal weakness that occurs after an epileptic seizure.

Attacks resembling TIAs may be the initial symptoms of cerebral tumours. These are presumably caused by alteration of circulation in the adjacent brain. Examination must therefore include careful auscultation of the heart and neck, measurement of lying and standing arterial pressure and the pressure in each arm, and assessment of peripheral vasculature. A 24-hour continuous ECG and tests for plasma glucose, lipid profile, cholesterol, full blood picture and ESR may establish an underlying cause, which should be dealt with accordingly. A prolapsing leaflet of the mitral valve may give rise to a loud midsystolic click or be silent, and can be confirmed with echocardiology.

Management

There are several uncontroversial lines of therapy:

- Control of hypertension. The diastolic pressure should be slowly reduced to less than 100 mmHg. Thiazide diuretics and propranolol are useful hypotensive agents that minimize the chances of postural hypotension. Reduction of arterial pressure may be all that is required to control TIA.
- Control of blood glucose;
- Reduction of PCV to below 45% by repeated small (200 ml) venesection;
- Reduction of hypercholesterolaemia or hyperlipidaemia, and the control of other risk factors, such as smoking;
- Prophylactic anticoagulants following emboli arising from the heart;
- The control of any cardiac arrythmia.

There are three other modes of therapy, each of which has its proponents:

- Endarterectomy
- Prophylactic anticoagulants
- Inhibition of platelet function with aspirin 150–300 mg daily.

The addition of other antiplatelet agents does not help.

It is possible that TIA due to atheromatous disease of the cerebral blood vessels has several causes, and that different subgroups of patients are affected favourably or adversely by these methods of treatment. Well controlled trials of each against no treatment are rare, and those against each other are in progress. One accepted course of practice is to perform four-vessel arteriography (preferably with a digital vascular imager) in patients who are otherwise surgically acceptable and in whom no other cause was found (see above), and to operate on significant stenosis or deeply ulcerated plaques detected in the carotid system.

Anticoagulants may be used in patients with intrinsic carotid disease where no operable lesion is identified, who have no medical contraindication, who are judged to take medicines reliably, and who can be closely supervised. Aspirin may be used in the remainder. It should be remembered, however, that the major cause of death in patients with TIAs is cardiovascular disease, and that treatment directed solely to the cerebral circulation may be irrelevant to the patient as a whole.

7.2 PSYCHIATRIC EMERGENCIES

Any psychiatric condition or circumstance in a patient which calls for immediate attention is to be considered a psychiatric emergency. These include the following:

- A mental state or condition due to which the patient becomes a source of danger to himself or others;
- Any mental condition which causes extreme anxiety for the patient's relatives;
- Any condition which may create disturbance in the community or family to an intolerable or unmanageable degree;
- Extreme or unbearable stress;
- Acute side effects of medications.

There are certain general procedures to be followed in a psychiatric emergency. History taking, however brief, is essential. Specific questions should be asked about the presence of any precipitating factors. A physical examination, including taking the vital signs, particularly blood pressure, should be carried out. With the exception of a severely excited or violent patient, the interview should be given in private, avoiding restraints as much as possible. The patient's experience should be accepted as a reality. Respect should

be expressed for the patient and the patient should obtain reassurance that the examiner is interested in his or her welfare.

The following are the most common psychiatric emergencies: attempted suicide, excitement due to functional psychosis or organic brain disorder, stupor, dystonic reactions, lithium toxicity and hysteria.

7.2.1 ATTEMPTED SUICIDE

All suicidal threats, gestures, attempts and risks should be taken seriously, as there are no fixed criteria to differentiate between genuine and spurious attempts. An unusual overdosage of drugs or intake of poison is almost always suicidal. Admission of suicidal intent by a patient should be accepted, but never their denials. It is not harmful and is useful to discuss the suicide question with the patient or the family. Real suicidal attempts include many conditions, among them:

- Suicidal attempt with a farewell or other note
- Repeated attempts
- Choosing lethal methods and pre-planning
- Attempts in the elderly.

Management

Do not leave the patient alone. Ensure the cooperation of family or friends. Refer to a psychiatrist and hospitalize the patient if family or friends are not confident or competent. Treat the underlying psychiatric disorder.

7.2.2 EXCITEMENT

Excitement may be due to a functional psychiatric illness or to an organic brain disorder caused by various CNS conditions.

Diagnosis

Acute organic mental disorder or delirium consists of fluctuation in the level of consciousness, inability to concentrate and impairment of memory, in addition to restlessness, agitation, sleep disturbance, slurred speech, irritability and fear.

It usually takes a few days for functional psychosis to reach the level of severe excitement. Other symptoms such as behaviour

abnormalities and sleep disturbance are usually present prior to excitement or violence.

The clinical picture of acute psychoses may be very similar to some of the conditions described previously. In addition, sudden medical or surgical illness may provoke an acute psychosis in a sufficiently susceptible patient. Confusion or other alterations in the state of consciousness are not seen.

A history of previous mental illness may therefore be present. A helpful point of distinction between an acute psychosis and a toxic confusional state is that in the former the sensorium is clear, although the content of thought is disordered.

The following possibilities should be taken into consideration:

- Puerperal psychosis;
- Acute schizophrenia: this usually presents a characteristic mixture of disordered thoughts and feelings, with hallucinations and disorders of conduct; it closely resembles the picture of amphetamine psychosis;
- Acute depression: delusions and hallucinations are usually of a self-deprecatory nature; hypochondriasis, suicidal ruminations and a tendency to depersonalization may be evident;
- Acute mania: elation characteristically combines with easily provoked irritability; the patient talks rapidly, jumping from one subject to another;
- Acute hysterical episodes: overtones of acting and self-dramatization may be apparent; even at their most violent, patients rarely injure themselves.

Management

Management of psychotic patients involves the following:

1. If at all possible, make some kind of contact with the patient, if only to establish yourself as a harmless and possibly helpful comrade.
2. Initiate the treatment of specific psychiatric syndromes.
3. Psychiatric consultation should be sought as soon as possible.

If the patient is extremely agitated, an attendant should stay with him or her. It is best to use someone who has not restrained the patient earlier. Physical restraint is not routinely recommended; however, one should not hesitate to take all the necessary precautions in dealing with a violent patient. Never argue with patients regarding their hallucinations or delusions. Always face the patient and stay out of arm's reach.

Sedation can best be achieved (except in head injury) by giving 50 mg chlorpromazine i.m. This dose can be repeated at half-hour intervals to a maximum of 200 mg, keeping in mind the fall in blood pressure as a side effect. If the fall in blood pressure makes chlorpromazine too risky, 5 mg haloperidol can be given i.m. and repeated twice, keeping in mind the possibility of severe extrapyramidal or dystonic side effects, management of which will come later.

Do not use any medication in patients with head injury. Always observe or refer.

In acute organic mental conditions always determine the underlying physical condition and start treatment as soon as possible. For behavioural disturbances chlorpromazine 50–150 mg orally can be used.

7.2.3 STUPOR

Non-responsiveness to surroundings, total absence of self-care, neglect of physiological needs and total motor inactivity are the symptoms of stupor. It is caused either by depression or schizophrenia. Hospitalization is necessary.

7.2.4 DYSTONIC REACTIONS

Dystonic reactions occur as side effects of antipsychotic drugs. The most common are muscle spasms, torticollis, difficulty in swallowing and oculogyric crisis (eyes fixed in the position of upper gaze).

Management

50 mg promethazine i.m. or 2 mg trihexyphenidyl orally three times daily. If the symptoms do not subside, refer to a psychiatrist.

7.2.5 LITHIUM TOXICITY

If the periodic serum lithium level is over 1.5 mmol/l, stop the drug and immediately refer the patient to a psychiatrist. Harsh tremor, severe confusion and severe vomiting or diarrhoea should be taken seriously in patients on lithium therapy. Stop the drug and refer the patient.

7.2.6 HYSTERIA

Hysteria is not considered an emergency, but the patient's relatives or whoever brings the patient in believe it is. The symptoms are dramatic and can be of a motor or a sensory nature. Reassurance, providing conditions for the patient to talk and release emotions and looking for underlying causes are important management steps. Never deceive the patient or inject a placebo. Minor tranquillizers can be given.

7.3 TOXIC CONFUSIONAL STATE DUE TO ACUTE ALCOHOL WITHDRAWAL

The alcoholic syndrome usually consists of two phases. The first is problem drinking, during which alcohol is used frequently to relieve tensions or other emotional difficulties. The second phase is the state of true addiction. The alcoholic is more prone to injuries and to the following illnesses:

- Impaired clotting mechanisms, and therefore susceptibility to subdural haematoma and bleeding within the dural sac covering the brain;
- Liver damage, which may result in cirrhosis;
- Pancreatitis;
- A variety of central nervous system disorders.

The signs of acute intoxication are drowsiness, disordered speech and gait, and erratic behaviour. Acute withdrawal from alcohol causes a characteristic toxic confusional state (delirium tremens) which, if uncontrolled, may be fatal.

Diagnosis

Delirium tremens usually occurs in a patient who has been withdrawn suddenly from alcohol after a binge lasting at least 2 weeks, and often considerably longer. The characteristic symptoms are tremulousness, apprehension, disorientation as to time and place, and visual, tactile and auditory hallucinations. In addition, insomnia, nausea and vomiting and motor incoordination may be present. These symptoms usually begin within hours of withdrawal, and are maximal from about 24 to 48 hours. Their occurrence should be anticipated in persons known to be heavy drinkers.

Excessive intake of alcohol may also give rise to cirrhosis, cardiomyopathy and various neurological syndromes such as

peripheral neuropathy due to vitamin deficiency, chronic cerebellar disease and Wernicke's encephalopathy. Thus, delirium tremens may be superimposed on an already debilitated patient.

There is a substantial and rather unpredictable variability in the severity of the symptoms of withdrawal. About 80% of patients develop mild symptoms, 14% moderate symptoms and 6% progress to full-blown delirium tremens.

Management

GENERAL

It is very important to establish contact with the patient, who is frightened, disoriented and frequently aggressive.

Thiamine 50 mg i.v. and 50 mg i.m. should always be given before starting a dextrose infusion, thereby avoiding the possibility of precipitating Wernicke's encephalopathy in a susceptible patient.

SPECIFIC

The aim of treatment is the induction of light sleep sufficient to control symptoms, while leaving vital functions unimpaired. Drugs to achieve this end are given orally, but may have to be given i.v. Chlormethiazole is the drug of choice. The does required to achieve a light sleep ranges between 4 and 10 g/day. It needs to be reviewed daily, the highest dose generally being needed 24–48 hours after alcohol withdrawal.

Patients generally do not need this drug after the 7th day. If oral administration is impossible, chlormethiazole may be given i.v. Give a loading dose of 30–50 ml of a 0.8% solution over 3–5 minutes to induce sleep, and continue an infusion of this concentration at 0.5–1.0 ml/min, adjusting the rate to the minimum dose required to keep the patient just sleeping lightly. Usually, 500–1000 ml are needed in the first 6–12 hours. If it is used in this way for a maximum of 12–18 hours, chlormethiazole is a safe drug. Side effects, which are dose-related, are respiratory depression, hypotension and supraventricular tachycardia culminating in respiratory arrest. For this reason do not continue the infusion for longer than 18 hours without first measuring chlormethiazole levels.

Chlordiazepoxide, either orally or i.v. in sufficient dosage to induce light sleep, may be used as an alternative to chlormethiazole.

Start with 40 mg 4-hourly and increase to 100 mg 2-hourly if necessary.

Although the tremulousness, fever, tachycardia and hallucinations subside over 3–4 days, there may be an interval of 1–2 weeks before full return to the patient's previous mental state. This interval is characterized by a lack of concentration, intermittent disorientation and agitated confusion. The latter is best treated with haloperidol 10 mg i.m. hourly as necessary (to a maximum of 60 mg/24 h). This may precipitate dystonic reactions, which can be relieved by benztropine 2 mg i.m.

Promazine derivatives should **not** be used, because of their hepatotoxic effect, and opiates should be avoided as they may cause respiratory depression in patients with liver damage.

The possibility of cirrhosis, heart failure and neurological disease induced by alcohol should be considered during examination of the patient, as these may need treating also. To this end, the following investigations should be made as soon as possible: chest X-ray, ECG, liver function tests and serum proteins, full blood picture and ESR, serum folate, electrolytes and blood urea.

Remember: All this is only first aid. Psychiatric consultation should be obtained as early as possible.

7.4 THE UNCONTROLLED AND POTENTIALLY HOSTILE PATIENT

Sudden onset of mental deterioration must be considered an emergency. It constitutes one of the severest tests of clinical skills.

Diagnosis

The following conditions must always be borne in mind:

- Cerebral hypoxia (poor perfusion or poor oxygenation);
- Infection – any, but particularly meningitis, encephalitis, pneumonia (especially pneumococcal) and septicaemia;
- Any pain or discomfort (commonly urinary retention) in a patient already seriously ill;
- Drugs – the following most frequently: barbiturates (especially in the elderly), amphetamines, monoamine oxidase inhibitors, atropine, corticosteroids, antiparkinsonism drugs and ephedrine. Almost every drug has been implicated at some time;
- An intracranial space-occupying lesion, e.g. tumour, abscess or haematoma (extradural, subdural or intracerebral);

- Hypoglycaemia or, more rarely, hyperglycaemia;
- Alcohol – either its excess or its sudden withdrawal;
- Complex partial seizures, such as may occur in temporal lobe epilepsy;
- Myxoedema;
- Thyrotoxicosis;
- Systemic lupus erythematosus;
- Deficiency of thiamine (Wernicke's encephalopathy – external ophthalmoplegia, ataxia and confusion), nicotinamide and vitamin B_{12};
- Hyponatraemia or hypernatraemia;
- Hypokalaemia;
- Hepatic pre-coma;
- Hypocalcaemia and hypercalcaemia;
- Acute porphyria.

Two or more of these may occur together. Any may be exacerbated by anaemia, hypotension or pre-existing chronic dementia.

Management

This involves consideration of the above causes. As a routine the following investigations and tests should be carried out:

- Haemoglobin and PCV
- Electrolytes and urea
- Blood sugar
- Blood calcium
- Blood gases
- Skull X-ray
- Liver function tests, including prothrombin time
- Blood culture.

Consider a lumbar puncture. Take a careful drug history.

Confusion is always worse at night. Disorientation may be helped by an easily visible clock, a familiar nurse and a light.

Never attempt to sedate an uncontrolled patient without due consideration of the cause; it may make the situation worse and it may be fatal. If sedation is vital or is deemed unharmful, give chlorpromazine (50–100 mg i.m.) initially, or phenobarbitone 100 mg i.m. or diazepam 10 mg i.v.

If the above are ineffective, give either chlormethiazole or a cocktail of haloperidol 20 mg, procyclidine 20 mg and promethazine 100 mg, all i.m. and drawn up in the same syringe. This rarely fails to bring peace to patients and their attendants.

Never give paraldehyde to confused but conscious patients as the pain provides considerable force and direction for the structure of their delusions. It goes without saying that verbal or pharmacological attempts to calm the patient must not be attended by any hint of aggression. This not only betrays lack of insight on the part of the physician, it may also be the facet of the physician's relationship to be grasped by the patient and is therefore disastrous.

7.5 HEAD INJURIES

Any type of scalp wound may occur. A fissured fracture of the skull may be simple (skin closed) or compound (skin opened). A depressed fracture may be simple or compound with or without leak of blood and/or cerebrospinal fluid. Skull fractures result from violence (whether direct or indirect) to the vault, face, chin or spine.

Clinical picture

The main results of a head injury are:

- Escape of the cranial contents: blood, brain matter, cerebrospinal fluid
- Injury to the cranial nerves
- Brain injury (concussion, contusion, laceration).

The signs and symptoms vary with the site of injury, as detailed below.

ANTERIOR CRANIAL FOSSA

- Epistaxis
- Extravasation of blood in the eye
- Rhinorrhoea (escape of cerebrospinal fluid from the nose)
- Escape of brain matter into the nose
- Injury to nerves: olfactory, optic, oculomotor, trochlear, abducent and first division of the trigeminal
- Concussion

MIDDLE FOSSA

- Escape of cerebrospinal fluid and blood from the ear
- Epistaxis
- Surgical emphysema of the scalp around the ear

- Injury to the nerves: facial, auditory, second and third division of the trigeminal
- Concussion

POSTERIOR FOSSA

- Extravasation of blood in the suboccipital region
- Injury to the glossopharyngeal, vagus and accessory nerves
- Irritation of upper cervical nerves leading to neck rigidity
- Patient is usually deeply comatosed

BRAIN INJURY

Brain injury may complicate injuries to the skull and may present with one of the following syndromes:

Concussion

This is momentary loss of consciousness and depression of the vital functions, leading to relaxed muscles and closed eyes; the pupils may be contracted and reactive and, in severe cases, dilated and motionless. The patient is pale and cold, with shallow rapid respiration and reflexes absent, but conciousness usually returns within a short time.

Contusion

Unconsciousness is prolonged; if the patient recovers he or she is confused, irritable or delirious, and shows focal signs. Severe concussion is usually followed by headache and photophobia. Lumbar puncture shows clear CSF with low tension. In severe cases coma persists, with dilated inactive pupils.

Cerebral irritation

The patient presents with general irritative phenomena, with focal signs. The patient assumes a position of flexion and lies curled on the side with the body bent forward, knees drawn up and arms flexed. The eyes are closed with photophobia and the pupils are equal and contracted. The skin is pale, the pulse weak and slow. Breathing is quiet and irregular and temperature is slightly raised.

Cerebral compression

This is due to increased intracranial pressure as a result of haemorrhage, oedema, infection or depressed bone. Usually there is a lucid interval. Cerebral compression is classified as early, late and terminal.

- Early cases present with headache, vertigo and mental dullness, vomiting, high blood pressure, slow full pulse, slow and deep stertorous breathing, and diminished temperature.
- In late cases the coma is deeper, coning may occur with Cheyne–Stokes breathing, rapid pulse and high temperature.
- In terminal cases there is hyperthermia, rapid irregular breathing and no pulse.

Management

COMBATING ANOXIA

Anoxia may cause more damage than the injury. A clear airway is essential.

1. Set up suction of the oropharynx.
2. Prevent the tongue from falling backward as follows:
   - Pull the tongue forward with a tongue forceps;
   - Insert an airway of adequate size;
   - Pull the jaw forward (to open the larynx).
3. Install an endotracheal tube if necessary.
4. Give oxygen therapy when needed.

POSITION OF THE PATIENT

The patient should be placed on the side with the head raised (to avoid venous congestion). Avoid Trendelenburg's position. Take care when manipulating the head because associated cervical spine injuries are common.

TREATMENT OF SHOCK

Shock is usually due to an associated injury elsewhere (fracture or internal bleeding). Hypovolaemic shock causes cerebral anoxia and so must be corrected by infusions. It is usual for isolated head injuries to be associated with shock.

ASSOCIATED INJURIES

When a head injury (implicating the brain) is associated with injuries in other parts of the body, injuries which may cause anoxia or shock should be dealt with immediately and as a priority, e.g.:

- Open or flail chest injuries, by pack or fixation;
- Maxillofacial injuries, by establishing a clear airway;

- Fractures, by splinting;
- Internal haemorrhage, by thoracotomy or laparotomy.

In all conditions an epidural clot (middle meningeal haemorrhage) should be dealt with immediately.

CONTROL OF EXTERNAL BLEEDING OF SCALP WOUNDS

External bleeding of scalp wounds should be stemmed as follows:

1. Apply a pressure dressing and crepe bandage as a first-aid measure if the patient is to be transferred to hospital for definitive treatment.
2. Apply finger compression of the scalp against the skull 2 cm from the wound margin while preparing for wound suture.
3. Apply haemostats to all bleeding vessels and bring the edges of the wound together.
4. Suture the wound.

SUMMARY OF EMERGENCY MANAGEMENT

1. Establish and maintain a clear airway.
2. Control haemorrhage.
3. Treat shock.
4. Examine the patient quickly but thoroughly to ascertain the type and degree of all injuries.
5. Splint long-bone fractures.
6. Evaluate the type and severity of nervous system injury.
7. Do not move the patient for any reason until the extent of all injuries is known, and until the immediate threats to life, e.g. respiratory obstruction, bleeding, etc. have been controlled and the patient's condition stabilized.

Where head injuries are involved the neurosurgeon should be consulted at an early stage.

8

Endocrine emergencies

Summary of contents

DIABETIC KETOACIDOSIS 189

HYPOGLYCAEMIA 190

HYPERGLYCAEMIC HYPEROSMOLAR NON-KETOTIC COMA 192

THYROTOXIC CRISIS 193

8.1 DIABETIC KETOACIDOSIS

Ketoacidosis can develop in any patient with diabetes mellitus, and it can be a rare complication in the non-diabetic patient receiving intravenous diazoxide, high-dose diphenylhydantoin (Dilantin), or high-dose steroids. Factors precipitating ketoacidosis are:

- Omission of insulin or oral hypoglycaemic medication
- Long-standing lack of control
- Acute infection
- Acute cardiovascular events
- Acute emotional stress.

Severe insulin deficiency is a prerequisite for the development of severe diabetic ketoacidosis. Insulin deficiency can lead to the following metabolic changes:

- Increased lipolysis, resulting in an elevation of plasma-free fatty acids;

- Increased catabolism, leading to amino acidaemia and nitrogen loss;
- Decreased peripheral utilization of glucose.

In summary, lack of insulin leads to:

- Acidosis
- Dehydration with hypovolaemia and hyperosmolarity
- Sodium depletion
- Total body potassium depletion
- Negative nitrogen balance.

A distinctive smell of apples is emitted on the patient's breath.

Complications

Complications related to therapy include:

- Hypoglycaemia
- Hypokalaemia
- Cerebral oedema.

Complications unrelated to therapy include:

- Hypotension
- Shock
- Renal failure.

In older diabetics, congestive heart failure and/or acute myocardial infarction are common. Oliguria and progressive azotaemia, despite adequate therapy, are most often related to underlying chronic renal disease, acute urinary tract infection, obstructive uropathy, and/or varying degrees and severity of acute tubular necrosis.

Management

The aim of management is to correct:

- fluid loss
- electrolyte losses (potassium and sodium)
- hyperglycaemia
- acidosis.

Management must also deal with the complications that may ensue.

8.2 HYPOGLYCAEMIA

Hypoglycaemia is a state in which the rate of glucose removal from the blood exceeds sufficient replacement to keep a patient

normoglycaemic. The exact glucose level required to remain asymptomatic varies according to:

- the rapidity of the fall in glucose level
- individual and sex variation
- age.

The primary glucoregulatory organs are:

- the liver, which responds with increased glycogenolysis and gluconeogenesis;
- the pancreas, which releases glucagon;
- the adrenal glands, which release adrenaline;
- the pituitary gland, which releases growth hormone and adrenocorticotrophic hormone (ACTH).

Clinical picture

The clinical picture varies depending on the person, the cause and the level of the blood glucose. The following conditions may present:

- Sympathetic responses, with an increase in pulse rate and blood pressure, and accompanying diaphoresis and hyperventilation;
- Parasympathetic responses, including hunger, nausea, eructation and occasionally bradycardia and mild hypotension;
- Cerebral responses, including less spontaneous conversation, lack of concentration, lethargy, lassitude and frequent yawning.
- Seizures and/or coma, finally, if the process is not interrupted by an increase in blood glucose levels.

Diagnosis

- In fasting hypoglycaemia the diagnosis should be confirmed by an 18–24-hour fast and/or by a low blood sugar level at the onset of signs and symptoms.
- In reactive hypoglycaemia a 5-hour oral glucose tolerance test is the procedure of choice.
- Dextrostix can be used in an emergency.
- **Never** give insulin as a 'diagnostic test'.

Management

Whenever a hypoglycaemic seizure occurs or threatens to occur, e.g. after an overdosage of insulin, a tablespoonful of honey may be of help. Elevating the blood glucose level by i.v. therapy is

essential in an emergency. Proceed with treatment of the sequelae and causes of hypoglycaemia accordingly.

8.3 HYPERGLYCAEMIC HYPEROSMOLAR NON-KETOTIC COMA

Precipitating causes

The most common cause of hyperglycaemic hyperosmolar non-ketotic coma (HHNK) is untreated or uncontrolled adult-onset diabetes mellitus. Less commonly, the following may be the cause: high-dose corticosteroid therapy, intravenous diphenylhydantoin, immunosuppressive agents, thiazide diuretics, glycerol therapy for cerebral oedema, propranolol, ethacrynic acid, mafenide burn ointment, diazoxide, Gram-negative pneumonia, pancreatitis, acute pyelonephritis, hepatitis, acute myocardial infarction, subdural haematoma, arterial thrombosis, gastrointestinal tract haemorrhage, pemphigus, systemic lupus erythematosus, eczematoid dermatitis, status epilepticus, peritoneal dialysis with high glucose or sorbitol dialysate, or (rarely) uncontrolled insulin-dependent diabetes mellitus.

Clinical picture

- Initially, there is polyuria and polydipsia.
- After a few days marked dehydration, with dry mucous membranes and doughy skin, will be present.
- The mental state may range from mild confusion to hallucination to coma.
- Focal neurological and motor function abnormalities may include hemisensory defects, hemiparesis, homonymous hemianopia, coarse flapping of the upper extremities, unilateral or bilateral hyper-reflexia to areflexia, tremors, fasciculations, nuchal rigidity and opisthotonos. Seizures may be present in about 15% of patients.

Haematological and chemical laboratory values in hyperglycaemia and dehydration are as follows:

- Glucose levels range from 400 mg/100 ml to 2800 mg/100 ml, with the average being just over 1000 mg/100 ml. Urine shows 4^+ glycosuria and no ketones.
- The white blood cell count, haematocrit and haemoglobin are usually elevated.
- Serum urea nitrogen and serum proteins are elevated.

- Sodium levels are increased.
- Potassium levels are decreased or normal.
- The effective osmotic pressure across the cell is usually between 330 and 460 mmol.

The most popular explanation for the fact that patients with HHNK do not develop ketoacidosis is that the amount of insulin present is sufficient to inhibit lipid mobilization, but insufficient to promote effective glucose transport into the cell.

Management

Therapy should be tailored to the findings in the individual patient. The following conditions should be corrected:

- Dehydration, hyperosmolarity and sodium and potassium depletion
- Hyperglycaemia.

The patient should be given heparin for 2–3 days to avoid venous and arterial thrombosis, and the underlying or precipitating cause should be treated. After the acute episode, diabetes may be controlled by diet alone or with small doses of hypoglycaemic agents.

8.4 THYROTOXIC CRISIS

Diagnosis

Signs of breathlessness, anxiety, tremor, severe eyelid retraction and uncontrolled atrial fibrillation are virtually diagnostic of thyrotoxic crisis. The thyroid gland is usually enlarged and obviously hyperactive, and the patient hyperpyrexial. However, patients can occasionally present with:

- a rapidly progressive weakness leading to drowsiness and coma;
- an acute psychosis;
- abdominal pain and vomiting, simulating an acute abdominal crisis.

Thyrotoxic crisis is usually precipitated by an infection, surgery, diabetic ketosis or by prematurely stopping antithyroid treatment. It may occasionally occur following ^{131}I therapy for thyrotoxicosis if the gland has not been suppressed beforehand.

Management

Take blood for a full blood picture, electrolytes, blood glucose and serum thyroxine, and save serum for triiodothyronine estimation should this be required later.

HYPERTHYROIDISM

- Potassium iodide 600 mg i.v. over 1 hour and then 2 g orally per day; this is reduced when the hyperthyroidism comes under control. Its beneficial effect lasts not longer than 2 weeks.
- Propylthiouracil, 1000 mg/day, or carbimazole 100 mg/day by mouth (or stomach tube if necessary) in three divided doses. It is best to give the propylthiouracil 1 hour before giving the potassium iodide, as this will ensure that the blockade of organification of iodine is established before the potassium iodide is given. Many authors deny the benefit of thiouracil derivatives in emergency.

ANXIETY

The patient should, if possible, be nursed alone in a quiet semidark room. Give chlorpromazine 100 mg i.v. This also helps treat hyperpyrexia. If anxiety is severe, an acute psychosis may supervene which, although sometimes resistant to chlorpromazine, usually responds to propranolol. The usual dose is 40 mg orally t.d.s., but it may be given i.v., 0.5–2 mg 6-hourly, if the patient is too ill to swallow. This may, however, precipitate severe hypotension and heart failure, and propranolol should not be used if there is pulmonary or peripheral oedema unless there is associated atrial fibrillation. Start with the lower dose, increasing as necessary. Should heart failure supervene, atropine 0.4–1.0 mg i.v. should be given.

LEFT VENTRICULAR FAILURE

This is caused by uncontrolled atrial fibrillation, which is treated along the usual lines with diuretics and oxygen. In addition, propranolol, in the dosage described above, rapidly reduces the ventricular rate and restores sinus rhythm, thereby controlling the failure. Digoxin has no influence on the ventricular rate in this situation, but is given to increase the force of myocardial contraction. In atrial flutter, propranolol is theoretically dangerous.

HYPERPYREXIA

Some degree of fever is always present and does not necessarily indicate infection. Use fans and a tepid sponge, together with chlorpromazine as above. Aspirin increases the metabolic rate and displaces thyroxine from prealbumin, and should not be used. If the above measures are ineffective, propranolol given as above may cause a dramatic improvement.

DEHYDRATION

This may occur from hyperventilation and sweating as well as insufficient fluid intake. CVP recordings are especially valuable in this situation, as hypovolaemia may be complicated by heart failure. Give dextrose 20% or 33% in water i.v. as extra calories are needed to supply increased metabolic demands. In addition, cautious replacement of sodium losses will also be necessary. Do not attempt to raise the serum sodium by giving hypertonic saline, as this may precipitate pulmonary oedema. Repeat the electrolytes after 12 hours.

ADRENAL INSUFFICIENCY

Hypotension and vomiting may be due to adrenocortical insufficiency, which is revealed by the metabolic stress. Take a blood sample for plasma cortisol and give 100 mg of hydrocortisone i.v. without waiting for the result, followed by 50 mg 6-hourly.

HYPOXIA

Occasional patients have a severe associated myopathy. This may give rise to ventilatory failure, so monitor the blood gases and be prepared to institute IPPR as necessary.

THROMBOEMBOLIC COMPLICATIONS

These appear to be common, and are serious. Give heparin.

Thyrotoxic crisis may be fatal, and in severe cases it may be necessary to anaesthetize, immobilize and ventilate the patient in an attempt to reduce metabolic requirements. The use of haemoperfusion over a polyacrylamide gel column has been advocated in intractable thyrotoxic crisis. This technique looks promising, and is worth considering. Effective therapy of the precipitating cause is a major determinant of the ultimate outcome.

9

Orthopaedic emergencies

Summary of contents

FRACTURES 196

Types of fracture 197
Diagnosis 199
First aid 200
Splinting 201

JOINT INJURIES 205

DISLOCATIONS 206

FRACTURES OF VERTEBRAL BODIES AND SPINAL INJURIES 207

HAND INJURIES 210

Diagnosis 210
Initial management 211
Management of specific injuries 212

HAND INFECTIONS 213

Management 214

FISHHOOK REMOVAL 215

9.1 FRACTURES

A fracture is a sudden interruption in the continuity of a bone, usually as the result of severe violence (traumatic fracture) or, rarely, due to abnormal fragility of the bone (pathological fracture) or, still more rarely, to fatigue.

Traumatic fractures (Fig. 9.1) are produced by three types of injury:

- Direct violence applied at the point of break. This usually gives rise to a transverse and sometimes a comminuted or compound fracture.
- Indirect violence which breaks the bone at its weakest point, by torsion or leverage, some distance away from the site of trauma; it is usually of the oblique or transverse type.
- Muscular violence applied by the patient via a forced and uncoordinated muscle movement which affects the bony prominences attached to the strong muscles.

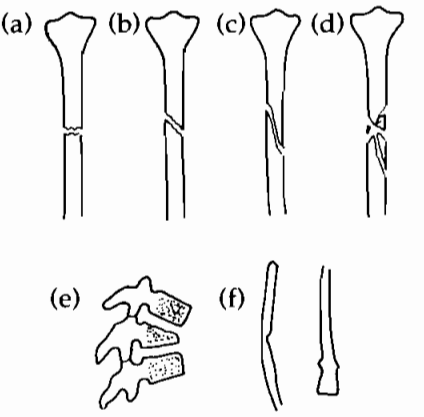


Fig. 9.1 Common patterns of fracture. (a) Transverse; (b) oblique; (c) spiral; (d) comminuted; (e) compression; (f) greenstick.

Types of fracture

CLOSED FRACTURES

Closed or simple fractures (Fig. 9.2), where the skin is intact, are classified into:

- Complete fractures, where the continuity of the bone is completely interrupted;
- Incomplete fractures, where there is no displacement because the fracture is not complete. These include:
  cracks or fissures (in flat bones and joint surfaces)
  greenstick fractures (in children) where the bone breaks on one side and bends on the other

subperiosteal fractures where the bone fractures and the periosteum remains intact, holding the fragment together. This is common in children because the periosteum on the growing bone is thick.

OPEN FRACTURES

In open or compound fractures (Fig. 9.3) the skin or mucous membrane is torn and the fragments are exposed. There is great risk of infection in an open fracture, especially one resulting from direct injury.

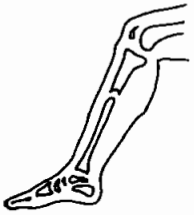


Fig. 9.2 Closed fracture.

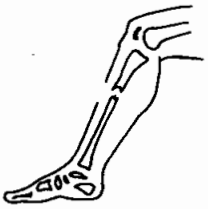


Fig. 9.3 Open fracture.

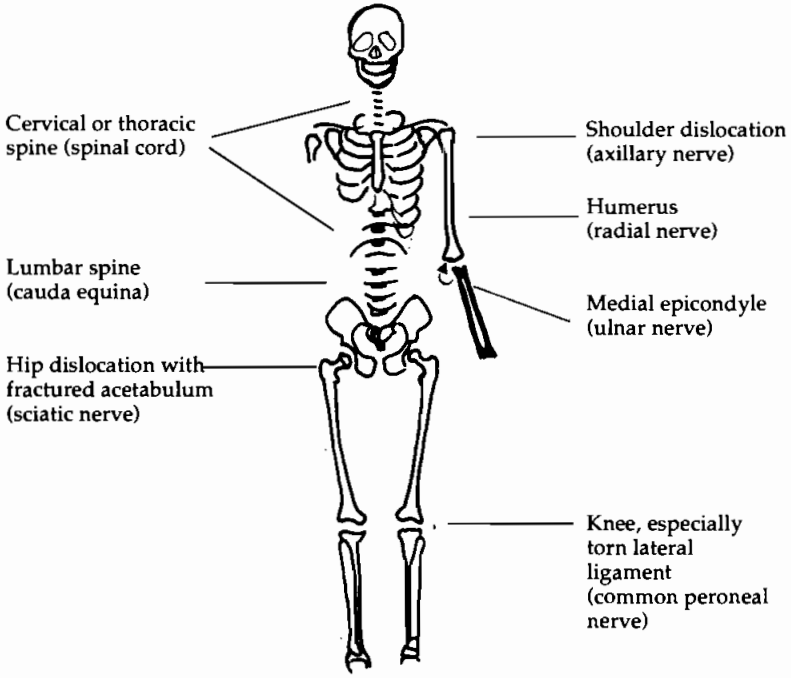


Fig. 9.4 Sites where fractures or dislocations are most commonly complicated by nerve injuries.

COMPLICATED FRACTURES

A complicated fracture is used to describe an open or closed fracture associated with injury to important nerves (Fig. 9.4), vessels (Fig. 9.5) or viscera.

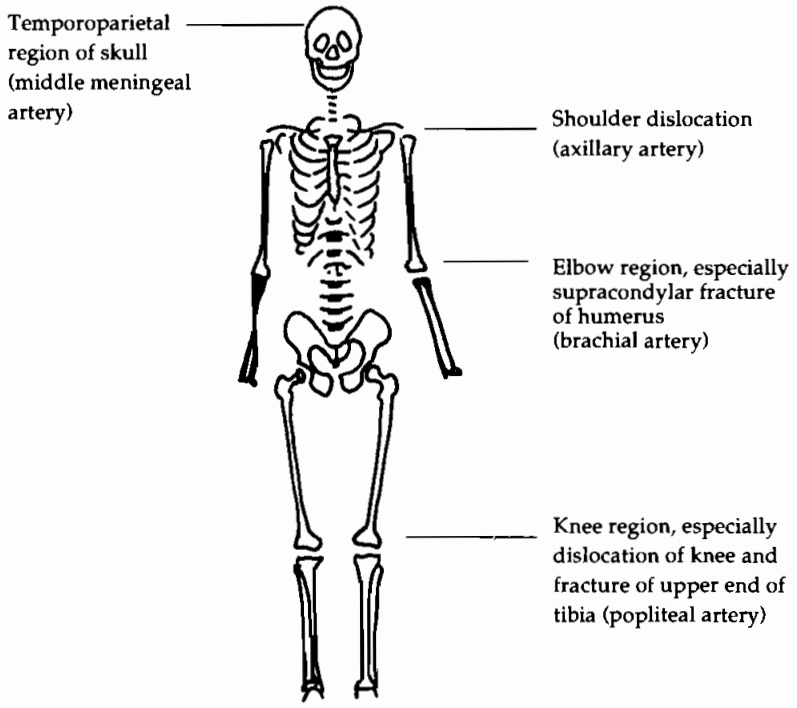


Fig. 9.5 Some sites where fractures or dislocations are liable to be complicated by damage to important blood vessels.

Diagnosis

There is a history of trauma with sharp pain increased by movement. Clinically, the cardinal signs of fracture are:

- Swelling and ecchymosis;
- Deformity – separation and displacement may be:
  angular (unequal action of powerful muscles);
  lateral with loss of apposition;
  over-riding, with shortening of the limb;
  distracted or separated due to excessive muscle pull (avulsion);

impacted (abnormal mobility cannot be elicited);
depressed (in a body cavity such as the skull);
rotated;

- Abnormal mobility (absent in incomplete and impacted fracture);
- Crepitus (painful);
- Loss of function;
- Local tenderness (acute, localized to a part of the bone).

Emergency management

Attention must be paid to:

- first-aid treatment
- resuscitation
- clinical assessment
- presence of complications.

FIRST AID

1. Make the patient comfortable while waiting for the ambulance.
2. Ensure a clear airway.
3. Cover any wounds with clean dressings.
4. Immobilize any fractured limb or part.
5. Control any bleeding.

Immobilization relieves pain, prevents shock and avoids complications. When moving a patient with a fractured limb, pain is diminished if traction is applied to the limb while it is being moved. When a fracture of the spine is suspected special care is necessary to avoid injury to the spinal cord; the patient should be lifted bodily onto a firm surface, taking care to avoid both flexion and extension (see Fractures of vertebral bodies and spinal injuries later).

In the upper limbs support may be provided by bandaging the arm to the chest, or, in the case of the forearm by improvising a sling. In the lower limbs temporary immobilization of the long bones is conveniently arranged by bandaging the two limbs together, so that the sound limb forms a splint for the injured one. For hip, thigh and knee fractures, use a traction splint where available, otherwise make a wooden splint from sticks, boards etc. (see Splinting below).

In compound fractures bleeding should be stopped and sterile dressings applied. Bones protruding through the skin should not

be reduced at the site of accident; a sterile dressing should be applied to prevent contamination and infection.

Antishock measures should be taken, e.g. morphine, infusions, while the patient is being transferred to hospital for definitive treatment.

SPLINTING

Splints may be classified into standard and improvised.

Standard splints

- A coaptation splint consists of a padded board or a rigid piece of material appropriately bandaged to the limb (Fig. 9.6).
- A traction splint (Thomas splint) is a half-ring splint used to provide continuous traction to a broken lower limb (Fig. 9.7).
- An air splint is a form of coaptation splint and consists of a plastic, airtight cylindrical bag with an air intake valve. An air splint is closed over the limb by a zipper and inflated by blowing air through the mouth tube. The air valve is closed by twisting the tube. A mechanical pump should never be used to inflate the splint because of the danger of excessive inflation leading to circulatory constriction.

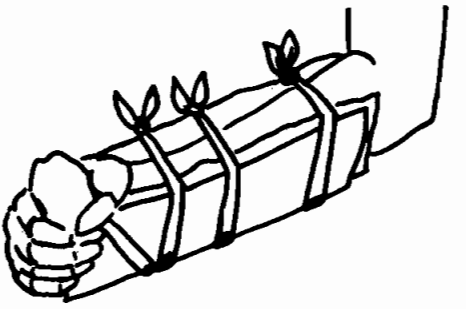
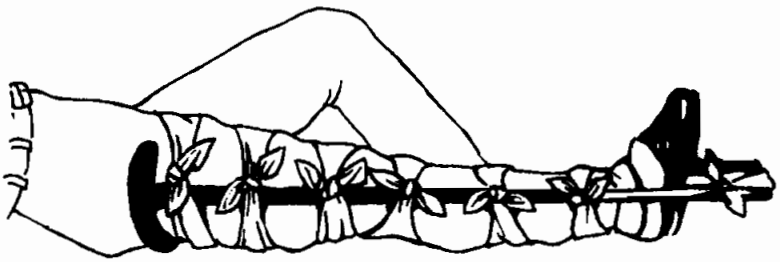


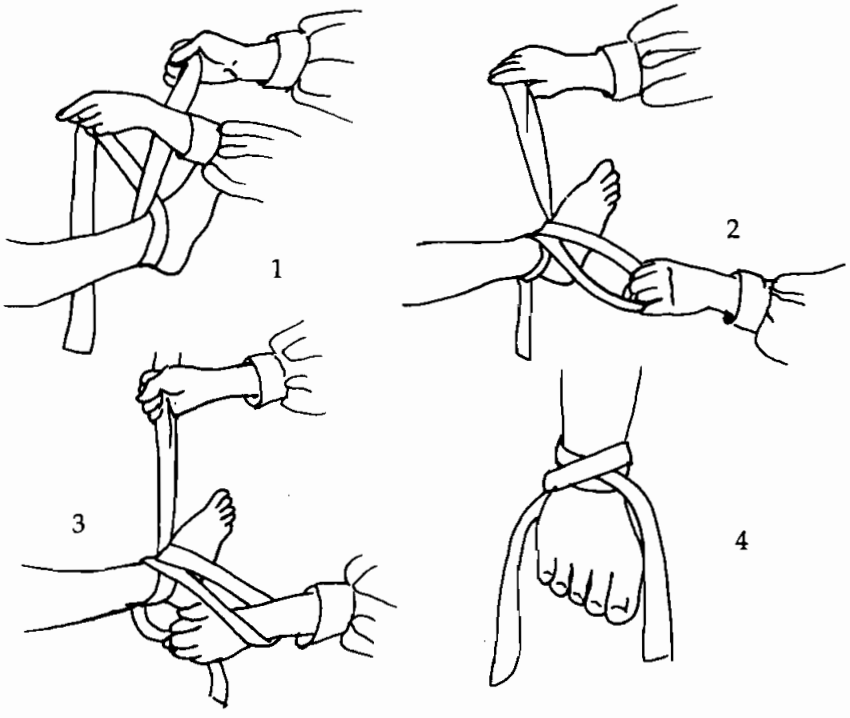
Fig. 9.6 Coaptation splint.

Improvised splints

In emergency situations, particularly outside the hospital where standard splints are not available, splints improvised from materials available should be used. Splinting at different sites may be performed as follows:



(a)



(b)

Fig. 9.7 Transportation of a patient with fracture of the thigh or leg. (a) Traction splint; (b) tying a Collins hitch.

Shoulder, arm and elbow

Standard splinting

The arm should be held close to the chest with the elbow at a right-

angle and a sling should be applied (Fig. 9.8a). If the elbow has assumed a straight position because of an injury to the elbow, the entire limb (arm, elbow and forearm) should be bandaged to the body without disturbing the position of the elbow. If available, a short padded-board splint should be bandaged to the front and back of the arm.

If a wire ladder is available, it should be bent to match the angle at the elbow. After being properly padded, it can be applied to the arm, elbow, forearm and hand with a bandage; the arm can then be placed in a sling or swathe.

Improvised splinting
A sling can be made by turning up the front portion of the patient's shirt or blouse and pinning it to itself. Bandaging the limb to the chest is more comfortable for the patient (Fig.9.8b).

Forearm, wrist and hand

Standard splinting
The forearm is placed in a sling; the elbow should be kept at a right-angle.

Improvised splinting
If boards of the proper length are not available, a magazine or heavy newspaper may be used around the forearm and hand and held in place with a bandage or adhesive tape. A sling is used to hold the forearm.

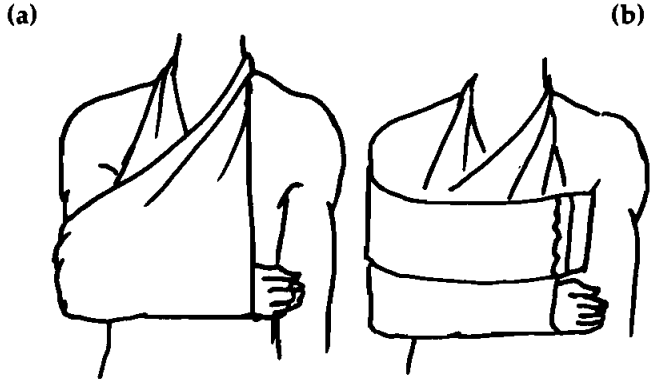


Fig. 9.8 Application of sling and swathe. (a) and (b) are explained in the text.

Hip, thigh and knee

Standard splinting
A traction splint is the most effective.

Improvised splinting
Coaptation splints with padded boards can be used. A long board is placed on the lateral side of the injured limb and trunk, extending from the lower thorax to the foot. Shorter boards are placed on the inner and back sides of the limb. All three boards are then bandaged; the long board should also be bandaged to the trunk. If no kind of splint is available, the injured limb may be bandaged to the uninjured limb; folded towels or small blankets and sheets are placed between them at pressure points, such as the knee and ankle (Fig. 9.9). The feet should be secured with a bandage.

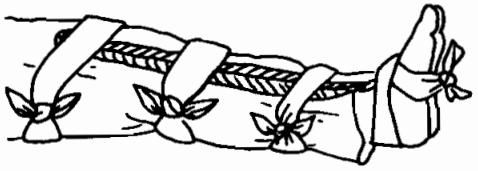


Fig. 9.9 Improvised splinting of a lower limb.

Leg

Standard splinting
A well-padded posterior gutter splint made of metal is useful for fractures of the leg. This should extend from the middle of the thigh to the ankle. The limb is held in the splint by bandages.

Improvised splinting
Padded-board splints are placed on each side of the limb and kept in place by bandages. When boards are unavailable a pillow splint is also highly effective, particularly for fractures of the lower third of the leg. The pillow should be long enough to extend from above the knee to a few inches beyond the heel. The pillow is bandaged to the limb and around the heel; it is then turned upward and pinned to itself so that it provides some support to the foot. Again, if materials are not available at all, the injured limb may be bandaged to the uninjured limb.

Ankle and foot

Standard splinting
A well-padded short posterior gutter splint of aluminium is used for fractures of the ankle and foot.

Improvised splinting
A pillow splint is quite effective. It should extend from the upper portion of the calf to the heel. The pillow is bandaged to the leg, and the portion projecting beyond the foot is folded and pinned to itself so that it supports the foot in a position close to a right angle.

OPEN FRACTURES

An open fracture is usually associated with severe haemorrhage and shock. It is therefore very important to control the haemorrhage, provide adequate breathing, and start management of shock before splinting or dressing the wound.

1. An open fracture should be covered with a sterile, saline-soaked dressing and a pressure bandage that should not be so tight that it acts as a tourniquet. The protruding fragment of bone should not be pushed back into the wound.
2. The fractured part may be splinted. An air splint is preferred to other splints where it is available.
3. Tetanus toxoid should be given, depending on the immunization status of the patient.
4. A patient with an open fracture should be treated with antibiotics prophylactically. Once a suspected fracture is confirmed, or vital signs have become stable in patients with an open fracture, an orthopaedic surgeon should be consulted for further management.

9.2 JOINT INJURIES

9.2.1 SPRAINS

Sprains are due to indirect trauma, causing overstretching and partial tear of the ligaments of the joint. Clinically a sprain causes pain, localized tenderness, swelling and ecchymosis. Movement is painful but abnormal movement is absent and the joint is stable. An effusion is common.

Treatment

Apply a pressure bandage or elastoplast bandage for 10–15 days. If pain persists, injection of procaine may be needed.

9.2.2 TRAUMATIC SYNOVITIS

Traumatic synovitis may follow direct trauma, sprain or the pres-

ence of a foreign body. Clinically the joint is distended and the patient assumes the most comfortable position. Movements are painful, with no muscle spasm, and there is no marked limitation of movement.

Treatment

1. Remove any foreign body.
2. Apply a firm bandage and immobilize the limb with a splint to minimize further effusion.
3. Aspirate if effusion is marked, to prevent overstretch and to relax the ligaments.
4. Perform muscle drill to maintain muscle tone and active movements to prevent adhesions.

9.2.3 HAEMARTHROSIS

Haemarthrosis follows severe injuries or fractures, causing severe pain and discomfort.

Treatment

Aspirate completely to avoid fibrosis and to prevent formation of adhesions. Apply a firm bandage and splint, and start muscle drill early.

9.2.4 PENETRATING WOUNDS

Penetrating wounds are very serious and liable to infection.

First-aid treatment

1. Sterilize the wound with an alcoholic antiseptic.
2. Immobilize the joint with a splint in the position of function.
3. Give antibiotics
4. Give tetanus toxoid.

9.3 DISLOCATIONS

A dislocation is the displacement of the joint; it may be complete or incomplete. Dislocation is caused by:

- trauma
- congenitally weak or deficient ligaments
- neuromuscular disorder or weakness.

Dislocation as a result of severe trauma is associated with haemorrhage

and soft-tissue injury. Arterial or nerve injury may complicate dislocation of the joint.

Clinical picture

There is pain at the site of injury after a history of trauma, for example, a fall on an outstretched arm or a car accident. A child may sustain a dislocation if the arms are pulled rapidly. There may be a deformity in the affected joint, loss of function, and muscle spasms around the joint.

Management

1. Determine the status of the arterial pulsations and neurological deficit distal to the site of injury.
2. Splint the extremity in the position in which it is found, to protect it from further injury and to minimize the pain.
3. Refer to an orthopaedic surgeon.

9.4 FRACTURES OF VERTEBRAL BODIES AND SPINAL INJURIES

The primary protection for the spinal cord is the spinal column; therefore, any injury to the spinal column can potentially result in injury to the spinal cord. If the cord is damaged, the effects are frequently permanent and disabling. Proper initial care can decrease the likehood of spinal cord injury but incorrect handling can result in damage to the cord.

The most frequent aetiology is car accidents, followed by falls, sports-related accidents (especially water sports) and gunshot wounds.

At the scene of an accident or in the emergency room, the main objectives are to recognize a potential spinal cord injury and to manage the patient in such a way as to minimize or prevent injury to the spinal cord. The primary task is to diagnose the nature of the spinal injury, and establish the condition of the spinal cord and the presence of associated injuries.

Spinal cord injuries are usually caused by forces exerted on the spine indirectly through other parts of the body, which may result in flexion, extension, rotation, axial loading or any combination of these. Injuries are more common in an area in which a highly mobile segment joins a less mobile one. Therefore, the most common sites are the lower cervical spine and the thoracolumbar junction (T10–L1).

Injuries to the spine occur in the form of pure dislocation, fracture dislocation (flexion forces), posterior displacement (exten-

sion forces), or wedge compression (axial loading). Injuries to the spinal cord are either complete, with no sensation and total paralysis below the level of injury, or incomplete, where there is sparing of some sensory and motor function below the level of injury.

Clinical picture

Shock is severe and there is acute pain with the least movement. One of the spinal processes will be prominent. If the spinal cord is injured, paraplegia is likely.

Emergency management

EVALUATION OF THE PATIENT AT THE ACCIDENT SITE

Any patient who has sustained a head injury, is unconscious, has fallen from a height, or has had a diving accident, should be assumed to have a spinal injury. Information about how the accident happened, the position of the patient and of parts of the body, and any external evidence, e.g. bruises, contusions or lacerations, may indicate injury to the spine.

The patient should be handled in such a way that he or she is not subjected to any abrupt movement or manipulation, and should be completely immobilized and secured onto a long board before transportation. Avoid unnecessary movements, especially flexion of the spine, which might precipitate or increase cord damage.

Again, pain or stiffness in the neck or spine, tenderness of the spine, or a complaint of inability to move the limbs or loss of sensation points to a spinal injury. If the patient is unconscious, diaphragmatic breathing or paralytic ileus and acute urinary retention may be the only signs of cord damage that may appear later on during the coma. Evaluation should include a search for signs of:

- compromised respiration and airway obstruction, or chest injuries from cord injuries above T10;
- external bleeding;
- limb deformity;
- bleeding from the ears, nose or mouth;
- unequal dilatation of the pupils.

The evaluation should also include a brief and quick check of the motor and sensory functions. This involves at least observing the patient's ability to move the hands and feet, and testing for the level of sensory loss by testing for touch and pain sensation and documenting this level on a diagram, or even by marking the level directly on the patient for further follow-up.

IMMEDIATE LIFE SUPPORT MEASURES

Any complete injury above C3 necessitates application of total artificial respiration by an Ambu bag, whereas an injury between C4 and C5 (below the origin of the phrenic nerve) requires respiratory assistance because lack of innervation of the intercostals causes paradoxical movement of the chest wall, decreasing the tidal volume and ventilatory capacity.

Oxygen should be given to all patients and suction of secretions, blood and vomitus from the pharynx performed because such patients are unable to cough effectively.

If respiration is affected do not give morphine. Otherwise give morphine to alleviate pain and anxiety. Start an i.v. lifeline. Other life-saving measures should be carried out simultaneously, especially stoppage of bleeding, treatment of concomitant lesions, and antishock measures (sympathectomy).

TRANSPORTATION

Patients who are properly immobilized with a spine board can be transported safely in the supine position. If a spine board of any kind is not available the patient should be wrapped in a blanket. Three people are needed to lift a patient with a suspected injury to the spine: one at the head and shoulder, the second at the back and the third at the pelvis. They should lift the patient on to the blanket or board in a single movement (Fig. 9.10).

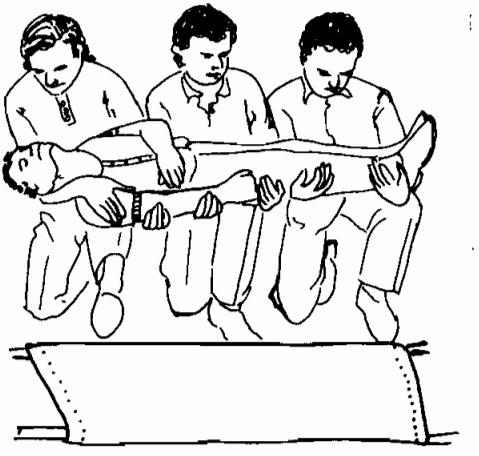


Fig. 9.10 Lifting a patient with a suspected injury to the spine.

Dorsal and lumbar fractures should be transported face downwards and cervical injuries should be transported on the back with the head supported between sandbags or other firm supports.

Transportation from the site of accident to the nearest hospital should be as expeditious as possible. However, unless the patient is in a critical condition due to non-spinal injuries or respiratory involvement, high-speed transfer is unnecessary. Indeed, the inevitable bouncing and jolting may further injure the patient's spine and spinal cord.

INITIAL THERAPY

Initial therapy is directed towards respiration, hypotension, bradycardia and the bladder. Hypotension and bradycardia are the result of lesions to the cervical or high thoracic spinal cord. These result in immediate functional sympathectomy. Therefore, hypotension and bradycardia are dealt with accordingly. The urinary bladder should always be catheterized with a Foley catheter.

9.5 HAND INJURIES

Following injury, a hand may be seriously disabled. This is true even if the treatment is appropriate.

Diagnosis

HISTORY

- Time and place of accident
- Mechanism of injury
- Amount of first aid given
- Right and left hand dominance
- Occupation
- Age
- General health

EXAMINATION

The patient should be supine with the arm extended. If the injury is open, the examination should be carried out using sterile techniques, including cap, mask and gloves. The initial examination should determine the structures injured. Any further procedures should be carried out in the operating theatre with the necessary anaesthesia, assistance, lighting and instruments, including a tourniquet, which is frequently necessary. Test for sensory functions as follows:

1. Using a sharp needle, test for sensation in areas supplied by all nerves that may have been injured.
2. Record with a diagram all areas of decreased or absent sensation.

Motor branch of median nerve

1. Ask the patient to abduct the thumb from the palm as much as possible.
2. Examine the thenar eminence.

Motor branch of ulnar nerve

1. Test the interossei: ask the patient to spread apart the extended fingers.
2. Test the adductor muscles: ask the patient to hold a piece of paper between the thumb and index finger, and try to pull the paper.

Bone and joint

1. Suspect injury if the appearance is distorted.
2. Palpate gently for movement at the suspected fracture site.
3. Obtain X-rays (posteroanterior, lateral and oblique).
4. Bear in mind the possibility of wrist dislocation and fracture.

Initial management

The task of the first person to see the patient is to examine the hand, control bleeding, clean the wound, apply a dressing, splint the hand in a safe position, elevate it and obtain a consultation.
The injured hand should not be subjected to repeated examinations. If the initial examiner suspects an injury, he or she should ask for consultation from a surgeon, who will carry out the necessary repairs.

Simple lacerations involving the skin can be repaired in the emergency room. Other injuries involving bones, tendons, nerves and vessels require the facilities of an operating room. Remove any clothing, rings and watches from the injured hand. The entire hand and forearm should be thoroughly washed with soap and water. Dirt and debris should be removed. Repair of simple lacerations requires a local anaesthetic.

All nerve blocks should be done in the operating theatre. Do not use solutions containing epinephrine as this may cause vasoconstriction and predispose to gangrene of the fingers.

DRESSING, SPLINTS AND ELEVATION

Wounds of the wrist or proximal fingers, palm or dorsum of the hand require a complete hand dressing. For splinting, the injured hand should be placed in the position of function, which is:

- Wrist extension 15°
- Metacarpophalangeal joints flexed 45°
- Interphalangeal joints slightly flexed at 15–25°
- Thumb abducted and rotated into an opposing position.

Elevation of the extremity minimizes swelling and discomfort.

TOURNIQUET

A small tourniquet may be used for finger lacerations. It should not be applied for more than 20 minutes. If a tourniquet is applied around the arm, a blood pressure cuff is preferred, inflated to 250 mmHg. This should not be used for more than 20–30 minutes.

REPAIR

Use of sterile techniques is essential. Primary closure is unwise if the wound contains foreign material. Antibiotics should be given. If skin closure cannot be achieved, coverage may require a skin graft or flap.

Management of specific injuries

FINGERTIP INJURY

This is the most common type of injury. If the tip is amputated, the length is maintained by use of a graft or a flap if the bone is protruding. Replacement of the amputated tip is seldom successful, but the injury should be cleaned.

NAILBED INJURY

Remove the loose nail. Repair the nail matrix accurately with 6/0 or 7/0 non-absorbable catgut. The nail can be used as a splint for the cut matrix.

NERVE INJURIES

These should usually be repaired primarily. Refer promptly to a consultant. Digital nerves are repaired if the laceration is proximal to the distal interphalangeal joint; operating microscopes are used.

TENDON INJURIES

These should be repaired primarily in the operating theatre. Flexor tendons should be repaired by a hand surgeon. If a surgeon is not available, the wound should be cleaned and the skin closed. Refer the patient as soon as possible for delayed tendon repair.

AMPUTATION INJURIES

Fingers or hands that have been completely amputated or are partly attached should be cleaned and reserved for replantation or revascularization. The stump wound should be cleaned and a compression dressing applied. The patient should then be transported to hospital with the limb elevated. A tourniquet should only be used for controlling bleeding initially, and should be removed after the dressing has been applied. The amputated part should be cleaned, placed in a sterile sponge, and then in a sealed plastic bag. The bag should be placed in ice until replantation, which should be done as soon as possible by an experienced team.

FRACTURES

Fractures must be reduced accurately. Unstable or open fractures require open reduction. Stable injuries should be immobilized for 14 days. Fractures should be splinted for 4 weeks.

BURNED HAND

Clean the hand with bland soap and water and apply flamazine ointment. Dress the hand with an occlusive dressing. It is essential to immobilize it in a safe position. Deep burns require grafting by a specialist (see Chapter 3).

BITES

Bites can usually be closed primarily after thorough cleaning (see Chapter 12).

9.6 HAND INFECTIONS

Pressure caused by oedema and pus in a closed space leads to ischaemic necrosis of tendons, nerves, bones and joints.

PHYSICAL SIGNS

- Swelling, which may be localized or generalized
- Oedema of the dorsum of the hand
- Discoloration, tenderness on palpation
- Local heat
- Pain on movement

Look for a recent minor cut or penetrating wound. Record the patient's temperature.

TREATMENT

1. Immobilize the infected hand to avoid the spread of bacteria.
2. Elevate it to minimize swelling.
3. Give antibiotics.
4. If there is pus drain it immediately and obtain a smear for culture and sensitivity.
5. Give tetanus toxoid.

Management

EARLY INFECTION (CELLULITIS)

Before pus is formed, there is a tender red swollen area in the hand. Do not wait for fluctuation to be sure that there is pus collection. Treatment consists of immobilization in the safe position, elevation and antibiotics.

ESTABLISHED INFECTION

1. Give antibiotics prior to incision and drainage.
2. Make a small incision over the site of maximum fluctuation.
3. Drain any pus immediately.
4. Carefully probe the cavity.
5. Leave a drain in the abscess cavity.

Most infections should be treated in the operating room under anaesthesia. The use of a surface anaesthetic such as ethyl chloride is not recommended as it is not effective and the frozen skin is difficult to cut. Immobilization, elevation and antibiotics should be continued after drainage.

SPECIFIC INFECTIONS

Pulp abscess

A pulp abscess is pus in the pulp space of the volar aspect of the fingertip. It follows a minor penetrating wound and may impair the blood supply of the distal phalangeal bone. It should be drained through a lateral incision, or in a skin crease over the site of fluctuation or skin necrosis.

Paronychia

Paronychia is infection in the skin at the sides of the fingernail, caused by pricking. When pus is present under the nail, the part of the nail that is not attached to the nailbed must be removed and the pus drained. This requires a digital block.

Subcutaneous abscess

Subcutaneous abscess usually follows minor penetrating wounds of the volar surface. The abscess is usually a point on the lateral surface of the finger and the incision should be made directly over the abscess.

Acute tenosynovitis

Acute tenosynovitis is an infection inside the tendon sheath. The most common cause is a penetrating trauma, so the history is important. Destruction of the tendon may occur and result in a useless finger. The diagnostic signs are a finger held in slight flexion, uniform fusiform swelling, a significant increase in pain with full extension of the finger, and tenderness over the course of the flexor tendon on palpation. If the condition is seen early, treatment with immobilization, elevation and antibiotics may prevent the development of pus. Refer to a hand surgeon.

Palmar abscesses

Palmar abscesses are usually located deep to the palmar fascia and usually occur in conjunction with a suppurative tenosynovitis. The hand is very swollen, with pitting oedema on the dorsum of the hand. Treatment requires careful exploration of the abscess cavity to ensure adequate drainage. The use of antibiotics, whether conventional or according to culture and antibiotic sensitivity, gives satisfactory results.

9.7 FISHHOOK REMOVAL

Occasionally, the sharp barbed end of a fishhook may accidentally lodge in the finger or ankle region of a child. It can be removed easily by one of the following techniques.

When the barbed end of a fishhook has passed through the skin, further advance the hook through the soft tissue until it projects outside the skin surface. Cut the shank close to the skin, grasp the distal fragment with forceps at the exposed tip and pull it out. Local anaesthetic may be required for this procedure.

If the barbed end of a fishhook lies deep in the skin and cannot be pushed out through the same skin surface, insert a no.11 blade proximal to the barb, after giving a local anaesthetic. Cut the skin and subcutaneous tissue, freeing the barb and permitting easy withdrawal of the hook. Alternatively, loop a piece of heavy silk two or three times around the index or ring finger of one hand, and pass the other end under the curve of the shank. Depress the barb, and disengage it with the other hand. Withdraw the fishhook with a quick jerk. This technique may also require local anaesthesia.

After removing the hook, dress the wound and give tetanus toxoid according to the immunization status of the patient.

10

Fever

Summary of contents

PATHOPHYSIOLOGY 217

MANAGEMENT 218

ACUTE PYREXIAL ILLNESS 219

HEAT STROKE 225

MALARIA 226

TYPHOID 228

SEPTICAEMIA 230

ISOLATION PROCEDURES 231

Pathophysiology

The thermoregulatory neurons in the hypothalamus are sensitive to endogenous pyrogens and control body temperature. In a healthy person there is a daily variation around the normal temperature of 37°C, the evening temperature being 0.5–1°C higher than the morning temperature. An endogenous pyrogen is a small molecular weight protein released by monocytes and many of the fixed phagocytic cells of the body, such as Kupffer cells. The release of endogenous pyrogens is activated by many infective agents, by lymphokines formed by antigenic sensitization of lymphocytes and by soluble complexes. Some tumours produce endogenous-like proteins.

The sudden onset of a fever below 40.5°C is not in itself harmful. Temperature only requires lowering if it:

- produces a tachycardia sufficient to cause cardiac embarrassment (usually only in patients with pre-existing cardiac disease);
- produces febrile convulsions (usually in children);
- makes the patient extremely uncomfortable.

The main concern with a fever of this level is therefore diagnostic; an approach to this problem is outlined below.

A fever above 41.1°C may, of itself, cause an encephalopathy, and therefore requires treatment. This extreme hyperpyrexia is usually caused by the following:

- Heat stroke, which is defined as a rapid rise in temperature to above 40°C following exposure to intense heat. The patient's skin is hot and dry as there is generalized anhydrosis, and initial confusion may progress to coma. It is thought to be due to a control failure of the sweating mechanism.
- Primary neurological lesion of the pons or hypothalamus, involving the thermoregulatory centre.
- Only rarely, infection. Malignant tertian malaria (*Plasmodium falciparum*) is the most important of these, and should always be excluded in hyperpyrexial patients.

Every 1°C rise in temperature increases the pulse rate by about 10–15 beats/min and the respiratory rate by about 3–5/min.

Management

If the temperature is below 40.5°C and treatment is thought necessary:

1. Commence tepid sponging, cooling with a fan and, if available, use cooling blankets.
2. Give aspirin 300–600 mg 4-hourly. Do **not** give aspirin to a patient under 14 years of age. Alternatively, paracetamol, 500 mg 4-hourly may be used.

If the temperature is above 41.1°C this constitutes a medical emergency. The aim is to reduce the temperature to 38.3°C within an hour. The following steps should be taken:

1. Immerse the patient in a bath containing cool water and ice chips.

2. Take the temperature at 5-minute intervals.
3. When the temperature has dropped to 38.3°C, nurse the patient on a bed in a cool room. The temperature usually then continues to fall to around 37°C (98.6°F).
4. If, after the initial cooling, the temperature starts to rise again, it can usually be controlled by the methods outlined above.
5. Where restlessness is a problem, chlorpromazine 25–50 mg i.v. can be given in addition to the physical methods of treatment.
6. During cooling, set up an i.v. line with dextrose 5% in water as the patient may become shocked or dehydrated.

10.1 ACUTE PYREXIAL ILLNESS

Diagnosis

Worldwide, viral illness is the commonest cause of pyrexial illness. This may give rise to pyrexia alone, or may be accompanied by non-specific symptoms such as headache, abdominal pain, vomiting, sore throat and nasal discharge. Such viral illnesses are self-limiting, usually settling within 3–4 days and are not, at present, amenable to specific therapy.

Fever may also be the initial symptom of other, more serious, infections and, if associated with rigors, bacteraemia or parasitaemia is likely. The range and diversity of these vary considerably from one geographical location to another and physicians should be aware of the local circumstances. Trypanosomiasis, which causes sleeping sickness in central Africa, and various of the haemorrhagic fevers specific to certain localities, are examples of locally relevant pyrexial illnesses.

Notwithstanding particular local circumstances, acute febrile illnesses of consequence (temperature above 39.5°C and often rigors) have associated clinical features which allow the doctor to make an informed decision about diagnosis and treatment. The clinical features may point to a specific system involved with the infection.

- Headache, photophobia, stiff neck and positive Kernig's sign suggest meningitis or meningoencephalitis.
- Pleuritic pain, cough and disproportionate dyspnoea suggest pneumonia.
- An acutely painful joint suggests a bacterial arthritis.

- Lower abdominal tenderness associated with an offensive vaginal discharge in a sexually active woman suggests pelvic inflammatory disease. The gonococcus (a Gram-negative diplococcus) must be excluded, but multiple organisms, including anaerobes, may be involved. Therapy should include metronidazole and a broad-spectrum antibiotic active against the prevailing gonococcus. Septic abortion has a similar presentation, with the addition of an enlarged tender uterus and dilated os. The likely organisms are again multiple, and include anaerobes. These patients are treated with penicillin, metronidazole and a broad-spectrum antibiotic such as gentamicin or chloramphenicol.
- A sore throat, with enlarged exudative tonsils, enlarged lymph nodes, difficulty in swallowing and prostration, suggest a bacterial tonsillitis (usually a β-haemolytic streptococcus, and therefore sensitive to penicillin). An associated erythematous rash, especially on the face and flexor surfaces of the arms and legs, which desquamates after fading, is characteristic of scarlet fever. Diphtheria is now uncommon. If difficulty in breathing is associated with a sore throat, consider acute epiglottitis. This is usually caused by the Gram-negative rod *Haemophilus influenzae* and responds to chloramphenicol.
- Local skin sepsis, either a cellulitis (most commonly due to a β-haemolytic streptococcus) or a boil (most commonly due to *Staphylococcus aureus*), may progress to septicaemia. Penicillin will be active against the streptococcus, but the staphylococcus may be penicillinase-producing and if this is suspected use flucloxacillin as well.
- Acute tenderness over a bone, with associated local swelling, suggests osteomyelitis, the treatment of which is initial drainage and flucloxacillin, on the assumption that the causal organism is *Staphylococcus aureus* and may produce penicillinase.
- Bloody diarrhoea associated with high fever is often infective, and implies not only local invasion of the colon but also bloodstream invasion by the organism. In these circumstances, a broad-spectrum antibiotic active against the likely pathogens– *Shigella* (ampicillin 50 mg/kg/day in four divided doses), *Campylobacter* (erythromycin 500 mg q.d.s.) and, less commonly, the non-typhoid salmonellas (ampicillin as above) – should be started. Chloramphenicol 1 g 6-hourly, orally or i.v., is effective against all the above. *Entamoeba histolytica* should first be excluded by examining a fresh stool. If present, metronidazole 400 mg t.d.s. should be started.

- Pain in the loin, frequency and dysuria suggest a urinary tract infection. If this is community-acquired, the infecting organism is likely to be an *Escherichia coli* (a Gram-negative rod) sensitive to sulfonamide (such as sulfamethizole 200 mg five times each day) or ampicillin 500 mg q.d.s.
- A painful enlarged liver, usually without jaundice but with signs of tenderness at the base of the right chest, suggests a liver abscess, which may be amoebic. Amoebic abscesses respond well to metronidazole 800 mg t.d.s., and aspiration is now considered to be unnecessary. There is not always a history of preceding diarrhoea. In 50% of bacterial liver abscesses there is no obvious primary site: there are usually a number of different bacteria involved, some of which are anaerobes. Bacterial abscesses should be aspirated and metronidazole and ampicillin given, depending on the results of culture.
- Jaundice. Viral hepatitis is unlikely to cause significant pyrexia. Yellow fever, malaria, leptospirosis and Q fever are specific infections commonly associated with jaundice and pyrexia. Septicaemias may also cause a non-specific toxic hepatitis.
- Abdominal pain. The acute infective diarrhoeas often cause pain, as may cholecystitis and diverticulitis, when there will also be localized tenderness and often a mass.
- Earache, a red drum or a discharging ear, suggests an acute otitis media, usually caused in adults by the Gram-positive *Streptococcus pneumoniae*, and therefore responsive to penicillin.
- A herpes simplex eruption is commonly associated with malaria, bacterial meningitis, pneumococcal pneumonia, leptospirosis and severe viral infection. It is uncommon in other acute febrile illness.
- AIDS. If the patient has had an elevated temperature for more than 2 weeks and no other cause can be found, the possibility of AIDS must be considered (see Chapter 14).

In addition, the clinical picture may suggest a possible cause through association. Contact with animals, either recreational, occupational or inadvertent, should be determined.

Anthrax, caused by *Bacillus anthracis*, a Gram-positive rod, is common among the cattle-rearing peoples of east and central Africa, and in those intimately involved in the handling of hounds. There may be a characteristic malignant pustule and surrounding oedema but the respiratory variety occurs without the pustule. Penicillin G 4 million iu 6-hourly is the treatment of choice.

Brucellosis is a septicaemic illness caused by a Gram-negative rod conveyed to humans through intimate contact with infected goats and cows, or through consuming infected milk or cheese. The signs and symptoms include fever, sweating, joint pains, and an enlarged spleen. Leucopenia is characteristic. It responds to tetracycline or chloramphenicol.

Leptospirosis The Leptospira, finely coiled motile spirochaetes, are concentrated in the urine of rodents. Humans, when in contact with infected non-salt water, are at risk, and so the disease particularly affects sewer workers, farmers, and workers in abattoirs. Penicillin G 4–10 million iu a day may be helpful.

Psittacosis, caused by the obligate intracellular organism *Chlamydia psittaci*, and usually transmitted to humans by the parrot family, gives rise to an atypical pneumonia responsive to tetracycline 500 mg q.d.s.

Q fever is an illness caused by the rickettsial organism *Coxiella burneti*. Humans are infected by inhalation of the organism after exposure to infected goats, cattle or sheep. Farmers and those involved in animal husbandry are at risk. Unpasteurized milk, though often contaminated with *Coxiella burneti*, does not seem to transmit the disease. Q fever responds to tetracycline 500 mg q.d.s.

If there is evidence that the patient is from an overcrowded, unhygienic deprived community, particularly one recently overtaken by any social calamity, the following possibilities should be taken into consideration:

Plague *Yersinia pestis* is the Gram-negative bacillus causing plague. The disease is transmitted to humans when there is close cohabitation between infected fleas, rats, humans and their domestic animals. The septicaemic illness is associated with swollen, tender and sometimes discharging local lymph nodes (bubo). The disease responds briskly to tetracycline 500 mg q.d.s. and streptomycin.

Epidemic louse-borne typhus This is caused by body lice infected with *Rickettsia prowazeki* living in intimate contact with humans. Here the initial septicaemic illness is followed by diffuse purpuric rash; tetracycline 500 mg q.d.s. is the most effective treatment.

Tick typhus A group of rickettsial diseases transmitted to humans by the bite of an infected tick afflicts hunters as well as the underprivileged. Some varieties produce an initial eschar at the site of the bite, and all are then followed by a headache, fever and petechial rash. They respond briskly to tetracycline 500 mg q.d.s.

Recent travel may be relevant, particularly if it has been to recognized infectious areas. The patient may be aware of recent exposure to infection, or a close contact may have a similar illness, which may even have been diagnosed. Prior surgery or trauma should alert the physician to the possibility of associated infection.

A menstruating woman using tampons may have the toxic shock syndrome caused by a staphylococcal exotoxin. Antibiotics do not appear to help, but it is reasonable to give flucloxacillin.

Diarrhoea, sometimes bloody, following a course of antibiotics suggests *Clostridium difficile* infection. Sigmoidoscopic appearances are characteristic.

Previous immunization may be of relevance, if only to exclude those illnesses from which the patient ought to have been protected.

The physician may, however, be faced with a patient with a severe pyrexial illness without any clear localizing or associated features. Worldwide, the two major problems here are typhoid and malaria (see later), and these should be considered in every seriously ill pyrexial patient. Virally induced influenza-like syndromes can simulate these two diseases, but viraemias do not usually give rise to rigors or the same degree of prostration as do these more serious conditions.

Many of the conditions mentioned above can give a septicaemic picture, but the presence of discriminatory signs and symptoms should help to decide between the various causes.

There are non-infectious causes of acute pyrexia. Some have already been mentioned (e.g. heat stroke) and are usually easily excluded. In addition:

- Adverse reactions to drugs can produce a fever; again, the circumstances usually indicate the diagnosis.
- Tumours, immunological disorders, chronic infections such as tuberculosis and infective endocarditis, and many other inflammatory diseases such as sarcoid, can produce fever. These conditions do not usually present acutely, assuming major importance only in the investigation of pyrexia of unknown origin.

The causes of pyrexia in immunocompromised patients is beyond the scope of this book.

Investigations

The aim is to identify the specific causal organism as quickly

and simply as possible; only in this way will the physician be able to offer rational therapy.

1. Take a specimen of urine, sputum, stool, pus, or a swab from any relevant site for culture before beginning any antibiotics. Immediate Gram-staining of this material should be carried out. This will often provide a vivid demonstration of the correctness of the diagnosis.
2. Take blood cultures. Anaerobic cultures should be set up, and the bacteriologist should be asked to provide a preliminary result in 12–24 hours.
3. Examine the urine for cells, blood and protein, the presence of which will suggest a urinary tract infection.
4. Obtain a chest X-ray.
5. Do a full blood count and stain a blood film, which may show the following:
   - malarial parasites or trypanosomes;
   - alymphocytosis with a typical mononuclear cells, suggestive of mononucleosis;
   - an absolute neutrophilia, suggesting a bacterial infection; typhoid and brucellosis are exceptions, as both are associated with a low blood count (below $5 \times 10^9/mm^3$); viral infections do not raise the white cell count;
   - a normochromic normocytic anaemia which may suggest haemolysis (common in malaria) or a more chronic process.
6. If there is any suggestion of meningism, perform a lumbar puncture.
7. Liver function tests are seldom immediately helpful. If focal intra-abdominal pathology is suspected, an abdominal ultrasound is the easiest and quickest way of confirming this; in any case it is a very useful investigation in seeking the cause of occult sepsis.
8. Take blood for serological investigations. These only assume importance retrospectively, as a four-fold rise in titre must be demonstrated to confirm a particular infection.

Treatment

Clinical examination and preliminary investigations will nearly always suggest the most likely diagnosis, which can then be treated appropriately.

Difficulty arises when, despite the physician's best endeavours, he or she does not have a clear idea of the likely cause of the pyrexia. There are several options:

1. Treat with broad-spectrum antibiotics, as for bacterial shock. This is a reasonable course of action if the patient is poorly perfused, is toxic with rigors, or has evidence of organ failure.
2. Adopt a wait-and-see policy, examining the patient repeatedly and being guided by clinical developments and the results of the investigations. This is the course usually adopted.
3. In anyone who has been to a malarial area, especially if there is doubt about the validity of the blood smear, give chloroquine or quinine (see later).
4. In anyone from an area where typhoid is shown to be a prominent problem, give chloramphenicol alone (see later).

In practice, the course adopted will depend on the physician's perception of how ill the patient is, and the facilities available, modified as necessary in the light of clinical developments and the result of investigations.

10.2 HEAT STROKE

The following factors predispose a subject to heat stroke:

- Elevated ambient temperature and humidity
- Lack of acclimatization
- Obesity
- Strenuous exercise
- Dehydration
- Fever
- Cardiovascular or central nervous system disease
- Delirium tremens
- Diabetes mellitus
- Skin disease (miliaria, scleroderma),
- Alcohol ingestion
- Phenothiazine, diuretic, anticholinergic or antihistaminic therapy
- Amphetamine abuse
- Anaesthesia.

Clinical picture

In children and young adults with a history of strenuous exercise in a warm environment the onset is sudden, with confusion, weakness or collapse; sweating is usually present; initially, the temperature may be only mildly elevated but continues to rise.

In older individuals heat stroke often occurs after a prolonged period of unusually hot weather, producing prodromal symptoms

of anorexia, nausea, vomiting and weakness, which may be present for several days before collapse. Occupation may also be a factor.

Seizures, hypotension and hypokalaemia may be present. Serum enzyme levels are elevated, reflecting liver and muscle injury; urinalysis may reveal myoglobulinuria. ECG changes involve non-specific S–T wave changes and prolongation of the Q–T interval.

Diagnosis

The diagnosis of heat stroke is usually evident but the physician must also consider meningitis, intracranial haemorrhage, typhoid fever, Rocky Mountain spotted fever, adrenal insufficiency, thyroid storm, delirium tremens, malaria, pyogenic infections and drug abuse.

Management

Measures to lower the body temperature and restore the blood volume and constituents should be carried out quickly. The principal aim in therapy is elimination of the toxic effects of heat and the accompanying increased oxygen requirement by rapid lowering of the rectal temperature to 38.8°C. Chlorpromazine can be used to prevent shivering and vasoconstriction. Hypotension may require pressor agents, but these should be used with caution. Steroids are not helpful and aspirin should be avoided.

10.3 MALARIA

Malaria due to any of the *Plasmodium* species is, worldwide, one of the commonest causes of an acute pyrexial illness. It often administers the final blow to an individual already primarily debilitated and anaemic from a combination of previous infections and malnutrition. Moreover, *Plasmodium falciparum* can give rise to complications in previously healthy individuals as follows:

Cerebral malaria Definitions vary, but this is usually taken to mean a severe encephalopathy with or without focal neurological signs in a patient with acute falciparum infection. The pathogenesis is probably a clogging of small intracerebral blood vessels by parasitized and haemolysed blood cells.

Blackwater fever (malaria haemoglobinuria) This involves massive intravascular haemolysis leading to jaundice and/or acute renal failure. Again, the pathogenesis is obscure; it may occur in

the absence of parasitaemia and is thus presumed to be an immunological response to the parasite.

Diagnosis

Diagnosis of malarial infection depends primarily on seeing parasites in a blood smear. Because the intensity of parasitaemia can vary from hour to hour, several serial blood smears should be examined before abandoning the search. It is common for a second smear to be positive when no parasites were seen in the first one.

There is usually a normochromic normocytic anaemia, low white blood count, and the platelet count is frequently reduced to around $100 \times 10^9/l$. This last is presumably due to platelet consumption, but a full-blown disseminated intravascular coagulation (DIC) is very uncommon.

Hypoglycaemia may contribute to the coma of malaria, particularly in those treated with quinine. This should always be looked for, and treated with i.v. dextrose 10% in water infusions.

Malaria can be confused with many other acute pyrexial illnesses, as outlined in the section above.

Management

The mainstay has been oral chloroquine 0.6 g given immediately, followed by 0.3 g 6 hours later, and 0.3 g daily for 3 days. The i.m. route is an alternative if there is persistent vomiting. However, the emergence of chloroquine-resistant strains has, in a variety of localities, forced an alternative regimen of quinine, pyrimethamine and sulfonamide. Quinine dihydrochloride is given with a loading infusion of 20 mg/kg in 500 ml of dextrose 5% infused over 4 hours. This is followed by 10 mg/kg doses every 8 hours given as an infusion of 250 ml of dextrose 5% over 4 hours. Quinidine may be used as an alternative. It too is given as a loading dose (15 mg base/kg), followed by infusions of 7.5 mg base/kg every 8 hours. The infusion may be discontinued when the patient can take by mouth. A side effect of both drugs is hypoglycaemia secondary to induced insulin release. This should be looked for regularly, if necessary being controlled by the use of dextrose 10% rather than 5%. The course should be continued for 10 days.

In addition, and starting concurrently, pyrimethamine 25 mg b.d. is given for 3 days, plus a sulfonamide such as sulfadiazine 2 g initially, followed by 0.5 g 6-hourly for 5 days.

Anaemia should be treated on its merits by blood transfusion. In desperately ill patients with over 10% of red blood cells parasitized, exchange transfusion should be considered.

As mentioned above, the pathogenesis of cerebral malaria is obscure. However, cerebral oedema does not play a significant part. Dexamethasone seems to prolong coma in survivors, as well as exposing them to the risk of steroid therapy. It has no positive effect on mortality. Having given antimalarials, the treatment of cerebral malaria is as for the comatose patient. Renal failure in blackwater fever is treated along the usual lines.

Pulmonary oedema due to leaky capillaries (ARDS) occurs in a few patients with severe falciparum malaria. The physician must be alert to this possibility. Management of such cases is as for pulmonary oedema (see Chapter 4).

10.4 TYPHOID

Diagnosis

Typhoid, due in 95% of cases to infection with the Gram-negative motile rod Salmonella typhi, is a leading cause of acute pyrexial illness in developing countries. The initial symptoms (first phase of the disease) are non-specific and resemble influenza: stepwise increase in fever, deafness, headache, malaise, anorexia, cough, sore throat and musculoskeletal aches; 10% of patients have associated gastroenteritis. At this stage 90% of patients will have positive blood cultures. Positive stool cultures (in 80% of patients) and urine cultures (in 25%) occur during the second phase of illness.

The second phase, that of the established disease, begins 2–3 weeks after ingestion of the organism. About 7 days into the second phase, the widely known but seldom seen rose-red spots occur on the chest and abdomen. These patients have bronchitis, lymphadenopathy, splenomegaly, occasionally hepatomegaly, a relative bradycardia, constipation and typhoid stupor. Stupor describes the characteristic mental state and may progress to frank confusion and coma, which is characteristic of the toxic encephalopathy related to typhoid. Intestinal perforation can occur in this phase of the disease.

Occasionally, patients present with the late complications of the disease, either metastatic spread to any site, or immune complex deposition, which is responsible for glomerulonephritis, myocarditis, pneumonia and cholecystitis.

Although typhoid usually presents as an acute febrile illness, it may also present as an emergency in one of three forms:

- Deteriorating consciousness progressing to coma;
- Intestinal perforation. Pain in the right lower quadrant is the most common initial sign, followed by signs of localized or generalized peritonitis. Abdominal X–ray should reveal free air.
- Intestinal haemorrhage. Macroscopic bleeding occurs not infrequently (massive haemorrhage is fortunately rare) and is signalled by a sudden fall in arterial pressure and temperature.

Clinical picture

Most but not all patients will achieve at least a fourfold rise in antibodies to O antigen (the Widal test), but this is an unreliable diagnostic pointer, particularly in communities in which salmonella infections are endemic. Ninety per cent (90%) of patients have a normal or low white blood count, and most have a normochromic normocytic anaemia. Jaundice is uncommon, but mild abnormalities of liver enzymes are present in 40% of patients.

In the early stages, typhoid may be confused with many other acute pyrexial illnesses. An approach to the differential diagnosis is outlined in the section above on acute pyrexial illness.

Management

If typhoid is suspected, the patient should be isolated and barrier-nursed. Take blood for a full blood count, electrolytes, urea and liver function tests. Take three blood cultures and send urine and faecal specimens for bacteriology.

The drug of choice is chloramphenicol. Give 50 mg/kg/day in divided doses 6-hourly, orally if possible, i.v. if not, for 2 weeks. The side effects of prolonged treatment with chloramphenicol must be borne in mind.

Coma in typhoid has been assumed to be due to a combination of toxaemia, inanition and fluid electrolyte derangement. Treatment therefore depends on chemotherapy and ensuring appropriate volume replacement.

If a large perforation of the intestine is suspected, a laparotomy should be carried out. These patients are obviously not ideal surgical candidates. Careful attention to volume replacement with a CVP line will maximize their chances. It may be possible to temporize with bowel rest and volume replacement with small perforations, since these may self-seal.

Intestinal haemorrhage is treated with blood replacement in the usual way.

10.5 SEPTICAEMIA

Septicaemia is a severe form of infection characterized by invasion and multiplication of large numbers of bacteria in the bloodstream.

Clinical signs

- Fever is usually high, spiking and accompanied by chills.
- Tachycardia accompanies or precedes fever and is proportional to it.
- The total leukocytic count may not show much abnormality in sepsis; the differential count is more reliable.
- Petechial-like lesions may be seen in the skin or the conjunctiva due to *Staphylococcus*, *Pseudomonas*, *Escherichia coli* and *Clostridium*.
- Shock is frequent in Gram-negative septicaemia but occurs less often with Gram-positive sepsis.
- Metastatic abscesses, especially involving bone, brain or spleen, are not unusual after septicaemia.

Any injured tissue is easily infected during septicaemia.

Diagnosis

Diagnosis is aided by a high index of suspicion on the part of the examining physician. In cases of suspected septicaemia a blood culture is the diagnostic procedure.

Causes

The cause of septicaemia is an infection somewhere in the body that invades the bloodstream. The type of bacteria causing the sepsis can usually be identified from the source of the infection as follows:

- Wound or intra-abdominal infection: coliforms, *Bacteroides* or *Staphylococcus*
- Burns: *Pseudomonas*, *Serratia* or *Staphylococcus*
- i.v. site: *Serratia*, *Klebsiella*, *Bacteroides* or *Staphylococcus*
- Lung: pneumococcus, *Streptococcus*, *Staphylococcus*, *Klebsiella* or *Pseudomonas*
- Urinary tract: usually *Escherichia coli* or *Proteus*
- CNS: pneumococcus or meningococcus.

Management

1. Establish an aetiological diagnosis.
   - Septicaemia rarely develops early in the postoperative period unless the operation was in or through infected tissues, or the patient had a pre-existing infection.
   - Examine the patient for clues as to the source of infection. Is there pain or tenderness in the surgical wound or an i.v. infusion site? Does the patient have purulent sputum, cough, pleuritic pain, rales or dullness? Is there diarrhoea? Is there dysuria or flank pain? Is there pain in the shoulder and an immobile diaphragm, suggesting a subphrenic abscess? Is there a pelvic or prostatic mass on rectal examination? Is there headache or nuchal rigidity?
   - Carry out appropriate laboratory studies: blood count, urinalysis, Gram stain of any discharge or of sputum or urine, chest X-ray and fluoroscopy for diaphragm movement.
2. Take appropriate cultures. Blood (50 ml from single or multiple sites), urine, sputum, wound or other drainage, stool if diarrhoea is present and cerebrospinal fluid if there is headache or nuchal rigidity. Always obtain cultures prior to starting antibiotics.
3. Antibiotics should be started immediately after the physical examination has been completed and cultures have been taken. Antibiotics are given in high doses by the i.v. route and, when possible, should be bactericidal in action and as specific as possible. The choice of antibiotic is based on the probable source of infection, the most likely bacteria found in that area, and information gained from Gram stains of material obtained from the infected area. If the infection is not responding readily to the agents being used, the antibiotics should be changed in accordance with the results of cultures and sensitivity studies when these become available.
4. Drainage. When a collection of pus is sealed off, forming an abscess, it is difficult for antibiotics to penetrate the area. An abscess should be drained as soon as its presence and location are determined and the patient's overall condition permits. Drainage is done only after large loading doses of antibiotics have been given.

10.6 ISOLATION PROCEDURES

Isolation procedures are a prime source of frustration, wasted time and wasted facilities in most hospitals. The unnecessary

use of strict isolation procedures is harmful, since a barrier is placed between the patient and the nurses and physicians. The isolation barrier tends to interfere with observation and care of the patient, and is damaging to patient morale. However, failure to use appropriate isolation procedures is also harmful, since a patient with a communicable infection may then become a threat to all patients and staff in the hospital. The same applies to the uncontrolled hostile patient.

Reasonable isolation procedures have as their objective the interruption of the pathway of transmission of communicable infections, whether from an infected patient to others or from the environment to a highly susceptible patient. Isolation should be discontinued as soon as the infection hazard is minimal.

To isolate a patient, all that is required is a room containing a sink and a closed soap dispenser. **Outside** the room place a table or cart containing gowns, masks, gloves, dressings and any other materials needed repeatedly. **Inside** the room place a commode for the patient's use if the room has no bathroom, linen, and plastic bags in which to discard disposable items.

Hand washing before and after attending each patient is professionally proper behaviour. As a matter of isolation technique, however, thorough hand washing is carried out either on leaving or on entering the isolation room, depending on the objectives being sought by isolation. Hands are washed on leaving in most isolation situations, but are washed on entering the room of a patient in protective isolation.

It is pointless to isolate patients with minor wound infections caused by common focal organisms, since these organisms are ubiquitous in hospitals. Such wounds must always be covered with an adequate dry dressing.

10.6.1 STRICT ISOLATION

Indications

- Infections at any site with staphylococci, group A streptococci, meningococci
- Open-cavity tuberculosis
- Clostridial myonecrosis (gas gangrene)
- 'Traditional' communicable infections such as smallpox, diphtheria and hepatitis

Technique

1. Gown. Put on outside the room and discard when leaving.
2. Mask. Put on when entering the room and discard when leaving.
3. Gloves. Wear if in contact with the patient.
4. Hand washing. When leaving the room.
5. Linen, equipment. Discard when possible. Otherwise place linen in marked bags and autoclave before routine laundering or use an inner plastic bag that is soluble in hot water. Dressings should be put in impermeable bags for incineration. Disinfect equipment before removing from the room.
6. Terminal cleaning. Air the room for 2 hours with the windows open and the door closed. Furniture, floors and soiled walls should then be washed with a germicidal solution.

10.6.2 WOUND ISOLATION

Indications

Grossly infected or copiously draining wounds infected with organisms other than those requiring strict isolation.

Technique

1. Gown. Wear if in direct contact with the patient.
2. Mask. Wear if in close contact with the patient.
3. Gloves. Wear if in direct contact with the patient during dressing changes.
4. Hand washing. On leaving the room.
5. Linen, equipment. Discard when possible. Otherwise place linen in marked bags for routine laundering and contaminated dressings in an impermeable bag for incineration; disinfect equipment before removing from the room.

10.6.3 STOOL PRECAUTIONS

Indications

Patients with enteric infections in which variable organisms are passed in faeces (amoebiasis, salmonellosis, shigellosis and similar infections).

Technique

1. Gown. Wear if in direct contact with the patient.
2. Mask. Not necessary.
3. Gloves. Wear if in direct contact with the patient, or when handling material contaminated with faeces.
4. Hand washing. On leaving the room.
5. Linen, equipment. Discard when possible. Otherwise place linen in marked bags for routine laundering and disinfect equipment before removing from the room wrapped for autoclaving.
6. Stools are passed or discarded directly into the sewage system. If the patient is using a bedpan, or if a laboratory stool specimen is removed from the room, wrap and treat the containers as contaminated.

11

Paediatric emergencies

Summary of contents

ACUTE RESPIRATORY EMERGENCIES 236
Foreign body obstruction of the airway 236
Acute asthmatic attack 237
Status asthmaticus 238
Bronchiolitis 239
Croup 239
Epiglottitis 240

SEIZURES 240

CARDIOPULMONARY RESUSCITATION 241

INTRAVENOUS TECHNIQUES 243

ENDOTRACHEAL INTUBATION 245

FEVER 246

OTITIS MEDIA 248

PNEUMONIA 249

GASTROENTERITIS AND DEHYDRATION 250

HEAD INJURIES 251

SURGICAL EMERGENCIES 252

Examination

The infant under 6 months old will probably not object to being placed on a bed and having all his clothes taken off, so long as

one provides entertaining distractions such as cooing and other pleasant noises. The 6–24-month-old also may not object to having all his clothes removed, but he will not like it one bit if he is taken away from mother and put on a bed or stretcher. It is usually most conducive to peace to examine the child while he sits on mother's lap.

The 2–3-year-old is usually difficult, no matter how charming the examiner may be. He does not like to have his clothing removed. He does not want to be touched, especially by a stranger. He has not the slightest desire to play with the doctor and there is often nothing that anyone, including mother, can do to convince him that the doctor is really a splendid person.

The school-age child is likely to be cooperative. He appreciates being treated with respect and also appreciates explanations of what you are doing.

SPECIAL CONSIDERATIONS

Certain principles apply in dealing with children of any age who are sick or injured:

- Be calm, patient and gentle.
- Be honest. Do not tell the child that something will not hurt if it will hurt.
- Where there are multiple casualties, if at all possible try not to separate the child from his parents, even if they are injured as well. Under such circumstances the child's normal anxieties about separation will only be heightened by worries that something terrible has happened to one or both of his parents.

11.1 ACUTE RESPIRATORY EMERGENCIES

11.1.1 FOREIGN BODY OBSTRUCTION OF THE AIRWAY

Upper airway obstruction in the infant or child may be due to aspiration of small objects, such as peanuts, coins or small toys, into the air passages, with resulting complete or partial airway obstruction.

Types of obstruction

PARTIAL OBSTRUCTION

The child may still be capable of good air exchange. If so, the child will be able to cough forcefully and will have good colour, although there may be wheezing in between the coughs.

COMPLETE AIRWAY OBSTRUCTION

The airway is completely obstructed if there is no air exchange at all. The treatment of complete obstruction in the child utilizes a combination of back blows and chest thrusts.

Management

Try to remove the obstruction first by putting your finger into the child's mouth. If this is unsuccesful, proceed as follows: if the victim is an infant, straddle him over your arm with his head lower than his trunk and supported with your hand around his jaw and chest. Use the heel of your other hand to deliver four back blows between the shoulder blades. After delivering the back blows, place your free hand on the infant's back, with your fingers supporting his head and neck, so that he is sandwiched between your two hands. Then turn him over and place him supine on your thigh, with his head lower than his trunk. While he is in that position, give four chest thrusts (performed just as you would perform external chest compressions on an infant). Then lift the infant's tongue and lower jaw forward, by placing your thumb inside the mouth, over the tongue, and open his mouth. If you see a foreign body, hook it out with your finger.

If the infant has not started breathing after these manoeuvres, open the airways and attempt artificial respiration (see Cardiopulmonary resuscitation below). If the chest does not rise it means the airway is still obstructed, and the back blows and chest thrusts must be repeated.

When the victim is a child who is too large to straddle over your forearm, kneel on the floor and drape him across your thighs, keeping his head lower than his chest. Once again, the sequence is four back blows followed by four chest thrusts, followed by inspection of the mouth.

11.1.2 ACUTE ASTHMATIC ATTACK

Asthma is a common condition among children, many of whom have associated allergies.

Clinical picture

The acute asthmatic attack is characterized by:

- Spasm

- Constriction of the bronchi
- Oedema and congestion of the lining membranes
- Hypersecretion of tenacious mucus plugs.

The chest, as a consequence, becomes hyperinflated and hyperresonant to percussion. Ventilation is progressively impaired. The patient develops increasing hypoxaemia, hypercarbia, acidosis and dehydration.

Treatment

1. Give bronchodilators: for a mild attack, epinephrine is usually sufficient (0.01 ml/kg), sometimes supplemented with an aminophylline infusion.
2. Give fluids to treat dehydration and loosen thick mucus collections.
3. Oxygen may be required to treat hypoxaemia.
4. Increased ventilation, usually with mechanical assistance, may be necessary to reverse hypercarbia and respiratory acidosis.
5. Treat the acidosis with sodium bicarbonate.

11.1.3 STATUS ASTHMATICUS

Physical examination

- General appearance: in what position is the child sitting or lying? In how much distress does he appear to be?
- State of consciousness: sleeplessness, stupor and coma are very grave signs in the asthmatic, usually indicating severe degrees of hypercarbia, hypoxaemia and acidosis.
- Vital signs: as the attack worsens, the pulse grows faster and weaker, and the blood pressure may fall.
- Skin and mucous membranes: look for evidence of dehydration.
- Look at the chest to assess respiratory excursion, which will be increased in the mild asthmatic attack and may be entirely absent in the severe attack.

Treatment

1. Adminster humidified oxygen, if possible by IPPR.
2. Start an i.v. lifeline with dextrose 5% in water or dextrose 5% in 0.25% normal saline if at all possible.
3. Give epinephrine 1:1000 by subcutaneous injection in a dose of 0.01 mg/kg.

4. Use an aerosolized bronchodilator through a nebulizer.
5. Give sodium bicarbonate: 1 mmol/kg, to be given i.v. over 5 minutes if needed.
6. Aminophylline: 2–4 mg/kg diluted in at least 10 ml of dextrose 5% in water to be given i.v.
7. Hydrocortisone: 5 mg/kg.
8. Transfer the child to hospital to recieve the proper specialized care.

11.1.4 BRONCHIOLITIS

Bronchiolitis is an inflammation of the bronchioles (small bronchi) caused by a viral infection and seen in children under 2 years of age. It produces essentially the same clinical picture as asthma, with prominent expiratory wheezing.

Treatment

1. Give humidified oxygen by mask.
2. The child may be more comfortable in a semisitting position, with the neck slightly hyperextended.
3. Have epinephrine 1:1000 ready if bronchospasm is severe, or if there is doubt as to whether the child has bronchiolitis or asthma.
4. Have a laryngoscope and endotracheal tube of the appropriate size ready.
5. Seek the help of a paediatrician or transfer the child to hospital.

11.1.5 CROUP

Croup (laryngotracheobronchitis) is a viral infection of the upper airways occurring in children between 6 months and 4 years of age. The infection leads to airway obstruction by causing oedema beneath the glottis.

Clinical picture

- The child with croup is hoarse, with a high-pitched stridor and a so-called 'seal bark'.
- There is a peculiar whooping sound on inhalation.
- Signs of hypoxia may be evident in restlessness, a rising pulse rate, and eventually cyanosis.
- Examination of the throat should be very gentle.

Treatment

1. Give humidified oxygen by mask.
2. In severe cases, start an i.v. lifeline with dextrose 5% in water.
3. Let the child assume the position in which he is most comfortable.

11.1.6 EPIGLOTTITIS

Epiglottitis is caused by a bacterial infection of the epiglottis, leading to a swollen, 'cherry-red' epiglottis, which may obstruct the airway.

Clinical picture

- The patient is usually, but not always, over 4 years old.
- There is pain on swallowing.
- Drooling is frequent.
- The patient may have an extremely high fever.
- Examination of the throat should be very gentle.

Treatment

1. Give humidified oxygen by mask.
2. Start an i.v. lifeline with dextrose 5% in water by microdrip.
3. Let the child assume the position in whch he is most comfortable.
4. Give a broad-spectrum antibiotic.

11.2 SEIZURES

Causes

The causes of seizure include head trauma, meningitis, hypoxia and hypoglycaemia. There may also be fever. As always, the history is of considerable importance.

- Has the child had seizures before?
- How many seizures has the child had today?
- Does the child have a history of head truama, diabetes, recent headache or stiff neck?
- Is the child taking any medication?

Treatment of the child with multiple seizures

1. Place the child on the floor or a bed, away from other furniture.

2. Clear and maintain an airway. Do not attempt to jam a bite-block between a child's clenched teeth.
3. Give oxygen.
4. Start an i.v. lifeline with dextrose 5% in water.
5. If the child is febrile, sponge him with tepid water to lower the temperature.
6. If the seizures do not stop, give diazepam in a dose of 0.3 mg/kg (to a maximum of 10 mg) and send the child to hospital immediately.

11.3 CARDIOPULMONARY RESUSCITATION

In infants and children cardiac arrest is much more likely to occur secondary to respiratory arrest and resulting hypoxaemia. Thus, children merit meticulous attention to maintenance of a clear airway and adequacy of ventilation; if these are assured, there may be no need for further resuscitation measures.

CPR is very dramatic and sometimes very rewarding, but it cannot save nearly the number of young lives that could be saved by adequate preventive measures.

11.3.1 AIRWAY

The airway should be opened. If the child is struggling to breathe but his colour is pink, the airway is probably adequate and immediate transport is indicated. If however, the child is blue or is not making breathing efforts at all, first open the airway. There are two alternative techniques:

- The head-tilt–neck lift: place one hand under the child's neck and the other on his forehead, then lift the neck slightly while exerting gentle pressure backward on the forehead.
- The head-tilt–chin lift: this may be helpful in drawing the tongue further forward away from the posterior wall of the pharynx.

11.3.2 BREATHING

As soon as the airway is opened, immediately check whether the child is breathing. Place your ear over the child's mouth and nose and look towards his abdomen. If the child is not breathing, or if breathing is not adequate, begin mouth-to-mouth resuscitation and/or provide artificial ventilation (IPPR).

To apply rescue breathing to an infant or small child, cover the infant's nose and mouth with your mouth; for a larger child, the standard mouth-to-mouth technique is applicable.

Adjuncts such as a bag–valve–mask of paediatric size may be used, but great care must be taken not to deliver too high a tidal volume.

GASTRIC DISTENSION

When artificial ventilation is excessive the child's stomach may become distended and push the diaphragm upward, thereby reducing lung volume. In that case, turn the child's whole body to the side and apply gentle pressure over the abdomen.

11.3.3 CIRCULATION

Once the child's airway has been opened and four quick breaths have been delivered, it is necessary to determine whether breathing alone has stopped or whether cardiac arrest is present as well. Therefore, check for a pulse. In infants, it is recommended that the brachial pulse be checked. This is located on the inside of the upper arm, midway between the elbow and the shoulder. To palpate for the brachial pulse, place your thumb on the outside of the infant's arm, between his elbow and shoulder, and lay the tips of your index and middle fingers along the volar (inside) surface of the arm, pressing lightly towards the bone. If there is a pulse, cardiac activity is present and the problem is only one of respiratory arrest, so continue rescue breathing – 20 times per minute for the infant, or 15 times per minute for the child. If the pulse is absent, then rescue breathing must be combined with external chest compressions to circulate the oxygenated blood.

The technique of chest compressions differs somewhat in the infant and small child because of differences in anatomy between children and adults. In the young child, the heart occupies a larger proportion of the chest and the ventricles lie higher in the chest than in an adult.

The proper area of compression for the infant is the midsternum. Two fingers are usually quite adequate to perform external chest compressions on an infant. Place the two fingers on the midsternum and compress 1–2.5 cm.

The child's heart is lower in the chest than that of the infant. Therefore, the compression point is over the lower half of the sternum. To apply compressions, use the heel of one hand with

the fingers kept well off the chest wall, and depress the sternum 2.5–4 cm. The compression rate in children is 80 per minute, with a breath interposed after every fifth compression.

11.3.4 DEFINITIVE THERAPY

1. Give oxygen as soon as it is available.
2. If ventricular fibrillation is present on the monitor, administer shock, starting with 50 joules, placing one paddle over the right chest at the junction of the clavicle and the sternum and the other over the apex of the heart.
3. Drugs:
   - Sodium bicarbonate: 1–2 mmol/kg i.v. stat. bolus, to correct acidosis.
   - Epinephrine : 1:1000:0.1 ml/kg i.v. stat. bolus, for asystole and fine ventricular fibrillation.
   - Atropine: 0.01 mg/kg i.v. stat. bolus, for bradycardia.
   - Lidocaine; 0.5–1.0 mg/kg i.v. stat. bolus for ventricular tachycardia, frequent premature ventricular contractions (PVCs), or recurrent episodes of ventricular fibrillation after countershock.
   - 10% calcium chloride: maximum dose of 1 ml/5 kg slowly i.v., in some cases of asystole or electromechanical dissociation.

11.4 INTRAVENOUS TECHNIQUES

Choice of the site and method of intravenous cannulation in the child will depend on the age of the patient as well as local preference.

11.4.1 SCALP VEIN INTRAVENOUS CANNULATION

The steps in establishing a scalp vein i.v. are as follows:

1. Gather the necessary equipment:
   - a 21 or 23 gauge scalp vein needle (butterfly)
   - adhesive tape
   - a 5 ml syringe filled with sterile saline.
2. Prepare the site on the scalp with an alcohol swab, being careful not to let any alcohol run into the child's eyes.
3. Attach the butterfly needle to the syringe and flush with saline. Detach the syringe to check the backflow of blood when the needle is inserted into the vein.

4. Palpate the target vein with one hand and grasp the plastic 'wings' of the needle with the other. Hold the needle tangentially with the bevel up, and pierce the skin about 0.5 cm distal to the point of entry into the vein.
5. Once there is good blood return and the tubing has been cleared, tape the needle in place.

11.4.2 HAND VEIN CANNULATION

Hand veins are not always easy to find in small children, especially chubby infants. The steps are as follows:

1. Restrain the arm on an armboard in such a way that the target vein is readily accessible.
2. Place a small tourniquet proximal to the puncture site.
3. Prepare the puncture site with an alcohol or povidone–iodine swab.
4. Attach a saline-filled, 5 ml syringe to the butterfly needle, and flush.
5. Pierce the skin tangentially about 0.5 cm distal to the anticipated point of entry into the vein. Advance the needle carefully into the vein until blood return is evident.

11.4.3 EXTERNAL JUGULAR VEIN CANNULATION

For cannulating the external jugular vein the over-the-needle catheter is usually to be preferred; a 22 gauge Medicut is well suited to this approach.

1. Wrap the child securely in a sheet or blanket, so that the arms and legs are restrained.
2. Place the child on a table so that both shoulders are touching the surface, and rotate the head 90°. An assistant should hold the child in position so that the head is extended 45° over the end of the table.
3. Palpate the vein along its whole length in order to obtain a feel for its direction. The i.v. catheter should be inserted so that flow will proceed caudad, that is, from the neck toward the feet.
4. Prepare the puncture site with an alcohol or povidone–iodine swab.
5. With one hand, immobilize the vein to keep it from rolling away from the needle.

6. Pierce the skin tangentially about 0.5 cm distal to the anticipated site of entry into the vein. Gently advance the needle under the skin until free blood return is obtained.
7. Should the external jugular vein be inadvertently perforated, swelling and discoloration will be immediately obvious. If this occurs, remove the needle and catheter and apply pressure to the puncture site for 3–5 minutes with the child in a sitting position.

11.5 ENDOTRACHEAL INTUBATION

The equipment and approach used for endotracheal intubation in infants and children varies according to age.

1. Gather the necessary equipment:
   - laryngoscope and blade: the blade should be straight, usually a Miller or Wis–Hipple is ideal;
   - endotracheal tube: endotracheal tubes for infants and small children do not have cuffs. The size of the tube used also depends on the size of the child. Table 11.1 indicates the size of tube to be used;
   - suction catheters that will pass the tube easily;
   - tape for securing the tube;
   - water-soluble lubricant.
2. Preoxygenate the child by bag–valve–mask oxygen supplementation.
3. Position the child on a firm surface with the neck flexed and the head elevated on a folded blanket. The neck should not be hyperextended, as this will make visualization of the cords more difficult.
4. Hold the laryngoscope handle and blade in the left hand and insert it into the right side of the child's mouth. As the blade is carefully advanced, the oropharynx and then the hypopharynx come into view. The tip of the blade is advanced into the vallecula, and the handle pulled gently upward at an angle of 45° to the floor, to elevate the tongue and jaw and bring the glottis into view. The little finger of the hand holding the laryngoscope is used to support the mandible and apply light pressure to the larynx.
5. When the cords have been visualized, watch them until they open spontaneously. Then take the appropriate endotracheal tube, hold it so that its curve is in the horizontal plane, and

insert it from the right side of the mouth. Slip the tip of the tube about 5–10 mm below the vocal cords, and rotate the curve of the tube into the proper plane as it advances. Once the tube is in the trachea, hold it firmly in place by gripping it between the thumb and index finger against the hard palate. This keeps your hand and the tube fixed in relation to the trachea, and patient movement will not cause the tube to be jerked out. The position of the tube must be checked by watching for chest movement and by auscultation of both lung fields during ventilation through the tube.

Table 11.1 Size of endotracheal tube in children

Age of child	Internal diameters of tube (mm)
Premature newborn	3.0
Normal newborn	3.5
Large newborn	4.0
1 month	4.5
1 year	5.0
2–3 years	5.5
4–5 years	6.0
6–9 years	6.5
10–12 years	7.0
13 years and over	7.5

11.6 FEVER

Elevated temperature in a young child may be a sign of a potentially serious illness. When localizing symptoms and signs are present, the nature of the evaluation can be concentrated on these findings. In febrile children without evidence of a specific source of infection, the clinician may rely on the rational use of laboratory tests to identify the aetiology of the fever.

The age of the child is an important factor in assessing the risk of serious bacterial infection:

- An infant under 3 months of age has an immature immune response. Fever is a dangerous sign of possible bacterial infection, e.g. sepsis, pneumonia and meningitis from Gram-negative organisms and group B, β haemolytic streptococci.

- Children aged 3–24 months predominantly suffer from bacteraemia and deep tissue invasion from *Strep. pneumoniae* and *H. influenzae* type B.
- In children over 24 months of age bacterial illness usually takes the form of a recognizable local infection, and rarely involves occult disease.

Diagnosis

HISTORY

The following information should be obtained:

- Deviation in feeding and sleeping patterns;
- Lethargy, poor feeding and irritability, which may be the primary symptoms of serious infectious illness;
- Time, course and severity of the above symptoms;
- Objective definition of the scope of observed alteration, including amount of fluid ingested and number of hours asleep;
- Symptoms related to specific processes;
- Nasal congestion, cough, rash, swelling, flaccidity and other localizing symptoms;
- A good percentage of infants and young children exhibiting a temperature between 39 and 40.1°C have a positive blood culture.

EXAMINATION

- General appearance: look for lethargy, anxiousness, weak cry.
- Vital signs: a significantly increased respiratory rate may be an important sign of respiratory infection, e.g. pneumonia or respiratory compensation for metabolic acidosis. Marked tachycardia not attributable to the fever or even bradycardia suggest severe stress.
- Look for meningism, swollen joints, rales, petechiae, purpura or diffuse rash.

Laboratory tests

Tests should include a complete blood picture; a white blood cell count in particular and sedimentation rate are sensitive indicators of serious bacterial illness.

It is important to obtain a chest X-ray to rule out pneumonia. If meningitis, septicaemia or urinary tract infection are suspected

then samples for CSF, blood culture and urine examination are essential. A temperature of 41°C or greater and/or a WBC of 20 000 or greater in an infant are indications for lumbar puncture.

Management

Exclude otitis media, which is common in children under 2 years of age, by careful examination and otoscopic examination of the ear. If the examination reveals no specific source of infection in a child under 2 years, then management is as follows until a paediatrician can be consulted.

- Give either amoxycillin 30 mg/kg/day and/or ampicillin 200 mg/kg/day, or penicillin G 100 000 iu/kg/day or gentamicin 5–7 mg/kg/day.

The aim is to eradicate the two most prominent offending organisms in this age group, *Strep. pneumoniae* and *H. influenzae*. Antipyretics and cold compresses are important safeguards against the development of febrile seizures. Fluids, whether by mouth or parenteral, are essential.

11.7 OTITIS MEDIA

Otitis media is the most common bacterial infection in children under 2 years of age. It is an important cause of generalized symptoms and fever in this age group. If not recognized by the general practitioner it can progress to cause serious impairment of hearing, and even bacteraemia and brain abscess. The most common responsible organisms are, again, *Strep. pneumoniae* and *H. influenzae*.

Diagnosis

Painful ear and discharge are the obvious symptoms. Symptoms may also be systemic in nature, e.g. irritability, poor feeding, vomiting, fever. An otoscopic examination can reveal changes in contour and colour of the tympanic membrane, or even perforation, with escape of discharge.

Management

1. Give broad-spectrum antibiotics:
   - erythromycin 30 mg/kg/day
   - ampicillin 100 mg/kg/day
   - sulfonamides in older children.

2. Give antipyretics.

Refer to an ear, nose and throat specialist, as the child may need tympanocentesis.

11.8 PNEUMONIA

Pneumonia in infancy and childhood is a serious illness. It may present with only non-specific complaints, but on examination tachypnoea is a consistent sign. Again, there may be nasal flaring, grunting, subcostal retraction, cyanosis, decreased breath sounds and rales.

Diagnosis

About 50% of infants below 2 years of age cannot be diagnosed as having pneumonia by history or even by auscultation and percussion of the chest. Therefore it is important that a child presenting with fever, non-specific symptoms and no obvious source of infection be considered as having pneumonia. An elevated white blood cell count and sedimentation rate puts the child at high risk of having an occult pneumonia, and a chest X-ray should be obtained in all such cases.

In older children pneumonia is more likely to present with more specific pulmonary findings such as cough, chest pain, abnormal breath sounds, localized dullness and rales. These symptoms may be accompanied by generalized symptoms including fever, chills, malaise, abdominal pain or headache.

Management

When pneumonia is suggested, a reasonable effort to identify the bacterial aetiologic agent should be made; this includes blood cultures and a Gram stain for sputum, which can be obtained if necessary by tracheal aspiration in infants.

Hospitalization of children with pneumonia is indicated if there is significant respiratory distress.

Under 5 years of age, pneumonia is most commonly caused by viral agents. *Strep. pneumoniae* is the most common bacterial cause and therefore penicillin G 100 000 iu/kg/day or penicillin V 100 mg/kg/day is the therapy of choice. For a pneumonia of unknown aetiology, *H. influenzae* must be considered and ampicillin 100 mg/kg/day is prescribed.

General treatment includes humidified oxygen inhalation, in an oxygen tent if necessary, ample fluid intake and antipyretics.

11.9 GASTROENTERITIS AND DEHYDRATION

Gastroenteric diseases are common among infants and young children, and constitute a frequent reason for visiting the emergency room. The vast majority are caused by viral infection and characterized by severe vomiting and diarrhoea. Dehydration associated with diarrhoeal diseases carries a high rate of child mortality in developing countries.

Diagnosis

Gastroenteritis must be differentiated from other diseases of the gastrointestinal tract, e.g. appendicitis, bacterial enteritis and hepatitis.

Gastroenteritis in children typically produces an initial syndrome of vomiting, anorexia and decreased fluid intake, often followed by diarrhoea and crampy diffuse abdominal pain. Fever is not prominent and most children recover with minimal care provided they do not become dehydrated.

Clinically the concern is for the hydration status and the electrolyte balance. The hydration status can be evaluated from the moisture of the mucous membranes, skin elasticity and urine specific gravity. The laboratory will determine the electrolyte deficits, mainly sodium, potassium and calcium.

Management

Paediatricians classify dehydration into three kinds: hypertonic, isotonic and hypotonic. According to severity these are divided into:

- mild dehydration (less than 5% of body weight)
- moderate dehydration (5–10% of body weight)
- severe dehydration (more than 10% of body weight).

For **mild dehydration**, simple directions to the mother as regards diet, fluid intake and signs to watch for at home will be sufficient. The opportunity should be taken to explain to the mother how to make up an oral rehydration solution at home using a three-finger pinch of salt, a four-finger scoop of sugar and 0.5 l of cold boiled water.

Chemically simple fluids are better tolerated than more complicated formulas, provoking less nausea and vomiting, and are better retained, e.g. soft drinks, jelly and juices. If a patient is febrile, rectal antipyretics should be administered to avoid gastric irritation.

For **moderate dehydration**, which is still an isotonic dehydration and can be managed without i.v. fluid replacement, simple electrolyte formulas (oral rehydration solution) available in the market can be used, provided dilution with cold boiled water is accurate, to avoid the administration of either hypertonic or hypotonic solutions.

Severe dehydration is serious because it may result in shock, with impaired tissue perfusion and a prerenal uraemia. There is a risk of sudden death. In such a child, an attempt should be made to measure the blood pressure and evalute tissue perfusion by estimating the capillary refill time.

The child should be transferred to hospital and should receive an initial i.v. infusion to restore the blood volume. Proper electrolyte balance is restored after accurate blood electrolyte and pH measurements have been taken.

11.10 HEAD INJURIES

Head injury is one of the most common types of injury in infants and young children. It is usually not serious and the majority of children do not require hospitalization.

11.10.1 MINOR HEAD INJURY

After a minor head injury, such as might occur in a fall out of bed, an infant or small child commonly exhibits the following signs and symptoms:

- Crying (crying immediately after injury is a good indication of the preservation of consciousness during the acute period)
- Vomiting (once or more)
- Transient period of lethargy.

Older children may exhibit additional symptoms:

- Headache
- Dizziness
- Fatigue
- Irritability.

An absence of specific signs points to neurological disability.

Management

These children require rest, symptomatic relief of headache, and

judicious restoration of fluid intake. Advise a gradual return to daily activities.

11.10.2 SEVERE HEAD INJURY

A severe head injury may occur following a fall from a significant height, or as a result of a car accident. It constitutes a true medical emergency and requires prompt evaluation, recognition of developing complications, and treatment for a favourable outcome.

Management

Hospitalize in the intensive care unit those patients who have focal or diffuse neurological disturbances, and all patients who have been rendered unconscious for a period of time. The following treatment may be started in the emergency room before the patient is transferred to an admitting unit. Steroids, hyperosmolar agents and assisted ventilation may help to prevent herniation of cerebral tissue at either the tentorial opening or the foramen magnum.

1. Provide adequate ventilation.
2. Steroids in massive doses may minimize the development or limit the extent of cerebral oedema following trauma.
3. Start i.v. fluid therapy. Administer hyperosmolar agents, such as 20% mannitol (2–3 g/kg) or a diuretic such as frusemide (1 g/2 kg) for control of cerebral oedema.
4. Insert an indwelling bladder catheter in a comatose patient, particularly one undergoing diuretic therapy, to prevent urinary retention or bladder distension.
5. Neurosurgical consultation may be required. Supportive care includes administration of daily fluid requirements, and careful skin care. Anticonvulsive drugs should be started in the presence of seizures.

11.11 SURGICAL EMERGENCIES

There are many clinical conditions that lead to surgical emergencies in infants and children. Most of these are due either to congenital abnormalities of the respiratory and gastrointestinal tracts, or to abnormalities of the muscles surrounding the abdominal cavity. There are two main categories:

- conditions leading to respiratory symptoms
- conditions leading to gastrointestinal symptoms.

11.11.1 CONDITIONS LEADING TO RESPIRATORY SYMPTOMS IN THE NEWBORN

The symptoms that accompany these cases may include:

- respiratory distress or air hunger
- generalized cyanosis
- collection of mucus and secretions in the mouth and pharynx.

These cases are emergencies and need immediate interference, which, in most cases, is surgical. They result from:

- congenital anomalies in the oesophagus
- congenital anomalies in the larynx (stenosis)
- congenital emphysema
- congenital diaphragmatic hernia
- congenital choanal atresia (at the back of the nose).

It is important to know the symptoms and signs of the above conditions in order to be able to diagnose them and save the child's life.

Congenital anomalies of the oesophagus

The oesophagus is a muscular tube lined by mucous membrane. It extends from the pharynx to the cardiac end of the stomach. Its function is delivery of food and fluid, together with saliva, to the stomach. Congenital defects of the oesophagus include complete obstruction and oesophageal atresia.

Complete oesophageal obstruction takes the form of two sacs: the upper sac, which is connected to the pharynx, is usually distended; the lower sac, which is connected to the stomach, is usually contracted or atrophic. In many cases there is a fistula, which connects either of the two sacs to the trachea. It is important to remember that most cases of congenital anomaly of the oesophagus are usually accompanied by other anomalies in other parts of the body, e.g. nervous system and urinary tract.

CLINICAL PICTURE

After delivery, the midwife or obstetrician notices the rapid collection of saliva in the mouth, with the appearance of foam. This is usually accompanied by choking and generalized cyanosis. If any of these is noticed, the presence of congenital anomaly of the oesophagus should be suspected and suckling or any other oral feeding should be stopped. To prove the diagnosis, a small

tube is passed through the mouth into the stomach. The tube will be stopped in the upper pouch of the oesophagus and may recoil to appear again in the mouth. It is preferable that this tube be radio-opaque, so that it will appear coiled in the upper pouch on an X-ray picture. A collection of saliva and/or milk will fill the upper pouch and will spill over, to be inhaled by the lung. This leads to chest infection. The lungs may also be affected by gastric secretion if a tracheo-oesophageal fistula is present as well.

MANAGEMENT

1. Stop oral feeding.
2. Apply continuous suction of saliva from the mouth and upper pouch.
3. Give intramuscular broad-spectrum antibiotics.
4. Refer to a paediatric surgeon.

Congenital bronchial emphysema

This results from stenosis of a bronchiole leading to one of the lung lobes. Air will be trapped in this lobe, which will enlarge inside the chest cavity, pressing on the rest of the lung, and will even push the mediastinum, together with the heart, to the contralateral side, pressing the lung on that side.

CLINICAL PICTURE

After birth the symptoms will develop gradually, the most important being difficulty in breathing, with generalized cyanosis. There is no collection of mucus or saliva in the mouth and pharynx, and no foaming.

On examination, there is enlargement on one side of the chest, together with spreading of the ribs and widening of the intercostal spaces. The mediastinum and trachea are shifted to the contralateral side. On percussion, there is evident hyper-resonance on the affected side, compared to the other side.

An X-ray will show the affected lobe in the form of an air sac, which has to be differentiated from congenital diaphragmatic hernia. In the latter case, there is a concave abdomen, due to the escape of most of the abdominal viscera to the chest cavity, together with peristaltic sounds heard on the affected side of the chest.

MANAGEMENT

Once diagnosed, the baby should be referred immediately to a paediatric surgeon.

Congenital diaphragmatic hernia

This is one of the most serious anomalies affecting neonates and it needs rapid surgical intervention. Mortality is about 50%, even after early surgery.

The pathology in such conditions is usually due to defective diaphragmatic development. The defect may be in the postero-lateral part of the diaphragm; this is the most common condition, and occurs usually on the left side. Rarely, it may be in the anterior part, behind the sternum.

As a result of this defect, the abdominal viscera (usually the stomach at first, followed by coils of intestine) will pass into the chest cavity due to the difference in pressure between the chest and abdominal cavities. The net result is compression of the lung and respiratory distress.

CLINICAL PICTURE

Signs appear immediately after birth in the form of respiratory distress and cyanosis. The most important sign is concavity of the abdomen, together with the presence of peristaltic sounds on the affected side of the chest, usually the left side. Plain X-ray shows the presence of coils of intestine inside the chest cavity.

MANAGEMENT

These cases need urgent surgical intervention. In the meantime it is advisable to pass a small stomach tube through the oesophagus, in order to empty the stomach of air and partially relieve the lung compression.

Some babies might need artifical respiration and oxygen administration until they are transferred to the nearest paediatric surgical unit.

Important: All neonates should be transferred in a portable incubator.

11.11.2 CONDITIONS LEADING TO GASTROINTESTINAL SYMPTOMS

Congenital conditions leading to gastrointestinal obstruction

Alimentary tract obstruction in babies is usually due to congenital anomalies. Such conditions are generally characterized by the following signs and symptoms:

- Repeated vomiting. The vomitus is usually yellowish or greenish in colour, due to the presence of bile. Any vomitus having a greenish or yellowish colour in the newborn denotes the presence of an organic obstruction in the gastrointestinal tract.
- Absence of meconium. Normally, the neonate passes meconium within the first 24–48 hours after birth.
- Abdominal distension. The degree of distension differs according to the site of obstruction. The higher the obstruction is, the less the distension will be, and vice versa.
- The presence of signs of dehydration, such as loss of skin elasticity and depression of the anterior fontanelle (usually in late cases).

The most common causes of intestinal obstruction in newborn babies and infants are:

- congenital gastrointestinal obstruction
- imperforate anus
- meconium ileus
- congenital abnormalities of the pancreas
- Hirschsprung's disease
- strangulated abdominal hernia.

CONGENITAL GASTROINTESTINAL OBSTRUCTION

This condition usually takes the form of complete obstruction of the gastrointestinal tract, with the absence of part of the tract. Above the obstruction there will be enlargement, due to collection of intestinal secretions. The part below the obstruction will be atrophic and collapsed. This condition can occur in any part of the gastrointestinal tract from the pylorus to the anus.

Clinical picture

After delivery, vomiting occurs immediately. The vomitus is usually yellowish or greenish. No meconium is passed and the abdomen starts to distend. In cases of congenital pyloric obstruction or

duodenal atresia, distension is limited to the upper part of the abdomen, whereas in atresia of the small intestine distension is more marked, and involves the whole abdomen. The most important sign is distension of the abdomen with evident loops of small intestine under the abdominal wall. Peristaltic movements may be seen through the skin. On examination of the anal canal, no meconium or faeces is found. Instead, some whitish mucus might be present.

Management

1. Perform gastric suction through a stomach tube.
2. Set up i.v. hydration with lactated Ringer's solution.
3. Refer to a paediatric surgeon.

HIRSCHSPRUNG'S DISEASE

This disease results from the congenital absence of neurogenic ganglia in the wall of the colon. In most conditions, the affected part is the rectum and the adjoining part of the descending colon. As a result of this, the affected part of the colon does not share in the peristaltic movements and consequently does not pass the intestinal contents through it. The end result is the collection of stools and gases in the upper part of the colon, which in turn will be distended and hypertrophic.

Clinical picture

In babies, Hirschsprung's disease takes the form of intestinal obstruction with vomiting, abdominal distension, no meconium and, lastly, absolute constipation.

Signs and symptoms may be delayed for some weeks. Then the mother notices the presence of constipation, which does not yield except following the use of rectal prescriptions such as rectal enemas and/or suppositories. Later, abdominal distension becomes very clear and the baby starts to be cachectic and to waste, especially in the lower limbs.

In untreated cases, ulceration of the colon and small intestines takes place. This leads to the occurrence of diarrhoea, which is a bad sign.

Diagnosis

Diagnosis depends upon:

- History, the most important aspect of which is late passage of meconium and signs of intestinal obstruction in the first 4 weeks of life;

- The clinical signs described above;
- An X-ray of the colon with barium, which will show distension of the colon and a narrowed part below the distended segment.

Management
After diagnosing the case, consult a paediatric surgeon.

Acquired conditions resulting in intestinal obstruction

INTUSSUSCEPTION

The most important and most common acquired condition resulting in intestinal obstruction is intussusception. A segment of intestine telescopes, into the lumen of an adjacent portion of intestine, causing intermittent and ultimately permanent obstruction and, if neglected, intestinal necrosis and gangrene. The underlying causes remain unclear, and it is therefore termed idiopathic. In some cases Meckel's diverticulum may also be present.

Clinical picture
Intussusception usually occurs within the first 2 years of life, commonly between the 5th and 10th months with a strong male preponderance. History includes intermittent abdominal pain, which is severe and colicky, causing the child to cry and draw up his legs into a flexed postion. Vomiting and bloody stools are very frequent symptoms. Often blood in the stools is mixed with mucus, producing the classic 'redcurrant jelly' stools which, in rare cases, may be the only presenting symptom. A detailed history from the mother is important. Early diagnosis may depend upon a high level of suspicion on the part of the physician.

Later on the child may present with prostration, pallor, clammy cool perspiration and an altered mental state. On examination, a mass is palpable either abdominally or rectally in the vast majority of cases. A rectal examination is essential because the mass or bloody stool may only be detected in this manner. Diagnosis can be confirmed by barium enema, which shows a lobulated filling defect that extends from the caecum into distal parts of the colon, usually the transverse colon.

Management
Most intussusceptions, especially early ones, can be reduced at the time of diagnostic barium enema by the application of external pressure on the barium column under screen. If not, a paediatric surgeon should be referred to.

12

Poisoning

Summary of contents

DRUG OVERDOSE 260

Identification of the drug(s) and dosage involved 260
Knowledge of pharmacology and toxicity 260
Intensive supportive care 260
Prevention of drug absorption 262
Elimination of the absorbed drugs 263
Antidotes 264
Ancillary measures 265

COMMON FORMS OF POISONING AND INTOXICATION 267

Sedative intoxication 267
Narcotic overdose 267
Amphetamine overdose 268
Salicylate intoxication 269
Ingested poisons 269
Strong acids 270
Cyanide 270
Petroleum products 271
Organophosphate poisoning 271
Carbon monoxide poisoning 274

BITES AND STINGS 275

Human bite wounds 275
Animal bite and sting wounds 276
Snake bites 277
Insect stings 278
Marine animal stings 278

12.1 DRUG OVERDOSE

The vast majority of adult cases of acute drug poisoning are self-administered, although most of these are manipulative or represent a cry for help, rather than genuine attempts at suicide.

Management

The principles of management are as follows:

- Identification of the drug(s) and dosage involved
- Knowledge of pharmacology and toxicity
- Intensive supportive care (priority one)
- Prevention of drug absorption
- Elimination of the absorbed drugs
- Antidotes
- Ancillary measures.

IDENTIFICATION OF THE DRUG(S) AND DOSAGE INVOLVED

The patient history may be notoriously unreliable regarding both the drug taken and the dosage. The clinical findings may be obscured by the ingestion of more than one drug. Specimens of urine, gastric contents and blood should be taken for definitive drug identification. Supportive therapy should not be delayed while awaiting identification of the drug.

KNOWLEDGE OF PHARMACOLOGY AND TOXICITY

The regional poison control centre should be contacted immediately and information obtained on the drug in question, its pharmacology, toxicity and the specific therapeutic measures to be taken.

INTENSIVE SUPPORTIVE CARE

This is the most important aspect of poison management. The following steps should be taken:

Maintenance of respiration

1. Assess adequacy of ventilation and oxygenation by monitoring arterial blood gases.
2. Establish and maintain a clear airway:
   - Clear vomitus or blood out of the mouth and pharynx.

- If not intubated, place the intoxicated patient on the left side with the head down to prevent aspiration.
- Install orotracheal, or preferably nasotracheal, intubation in the following cases:
  respiratory insufficiency
  loss of consciousness
  impaired or absent gag reflex
  convulsions.
- Provide oxygen supplementation or mechanical ventilation as indicated.

Maintenance of circulation

1. Assess the cardiovascular status by monitoring vital signs, ECG, CVP.
2. Establish a reliable i.v. line in all intoxicated patients.

Hypotension is usually responsive to volume expansion with normal saline and correction of hypoxia and acidosis; if there is no response to such measures, dopamine 5–20 μg/kg/min is the vasopressor of choice in most cases of drug overdose.

Hypertension is uncommon, usually transient, and does not usually require treatment.

Dysrhythmias may occur due to autonomic and direct cardiac effects. Antidysrhythmic treatment may be needed.

Care for the central nervous system

Depression of the CNS is a common feature of overdosage, especially with sedatives, hypnotics and narcotics. The stages of CNS depression are as follows:

0: asleep; arousable
I: comatose; withdrawal from painful stimuli
II: comatose; no withdrawal from painful stimuli; most reflexes intact
III: comatose; most reflexes absent; no respiratory or cardiovascular depression
IV: comatose; reflexes absent; respiratory and/or circulatory failure.

Any drug-induced comatose patient should be given the following:

- 50 ml dextrose 5% in water i.v. stat. bolus until the blood glucose level is obtained.
- Naloxone hydrochloride i.v.
- Thiamine i.v.

Supportive care is vital to any case of poisoning with depressed consciousness. Haemoperfusion or haemodialysis is indicated in managing stage IV overdosage.

Convulsions
Convulsions may complicate poisoning and are best treated with diazepam.

Metabolic acidosis may accompany certain drug overdosage, e.g. salicylates, methanol, ethylene glycol. Sodium bicarbonate is given i.v. according to blood gas measurements.

Normothermia is restored by appropriate means.

Contraindications
Analeptic drugs are contraindicated, as they have been associated with an increase in mortality.

PREVENTION OF DRUG ABSORPTION

Forced emesis
Forced emesis is the preferred means of emptying the stomach in conscious patients. Syrup of ipecac 30 ml orally with 200 ml water usually induces vomiting. If there is no response within 20 minutes, repeat the dose. If there is still no response within 20 minutes, carry out gastric lavage to remove both the ingested drug and the ipecac because of its cardiotoxic properties.

Forced emesis is contraindicated in cases of:

- CNS depression
- impaired gag reflex
- status epilepticus
- caustic ingestion
- ingestion of certain hydrocarbons.

Gastric aspiration and lavage
1. With the foot of the bed raised about 0.5 m and the patient lying on the left side, insert a wide-bore rubber stomach tube, gauge 30. A gag may be necessary to prevent biting on the tube.
2. When the tube has been inserted, verify its position in the stomach by aspiration of the stomach contents, or by blowing a little air through it and auscultating over the abdomen, when a bubbling sound will be heard. Aspiration is better achieved by lowering the funnel to which the stomach tube is attached

to a level well below the patient's head. It is advisable to aspirate prior to lavage, as initial lavage will drive some of the stomach contents into the duodenum and promote absorption.

3. When no further material can be aspirated, carry out repeated careful lavage with tepid water, using the same apparatus and no more than 300 ml water for each single washout. Continue lavage until the returning fluid is clear, which may necessitate a total volume of 40 l. Little is achieved by employing lavage fluid other than water, except in some specific instances.

The following may be left in the stomach after lavage: 100 ml milk because of its demulcent properties in corrosive poisoning; the chelating agent desferrioxamine, in acute iron poisoning; a solution of sodium bicarbonate for salicylate poisoning. For all other cases of poisoning no solution should be left in the stomach because of the risk of aspiration pneumonia, which may lead to further impairment of consciousness.

The contraindications of gastric lavage are the same as those for forced emesis, but they can be overcome by prior endotracheal intubation.

Activated charcoal

Activated charcoal is an inert, non-toxic substance which absorbs most drugs and inhibits the absorption of drugs not removed by emesis or lavage. Give 50–100 g orally or by gastric tube. It is contraindicated if an oral antidote is to be given, as it will bind the antidote.

Cathartics

Drastic purgatives decrease the intestinal transit and absorptive times of ingested drugs. They may be given orally or by gastric tube as follows:

- 150–300 ml of magnesium citrate or 70% sorbitol
- 50–150 ml magnesium sulfate 10%.

ELIMINATION OF THE ABSORBED DRUGS

Forced diuresis

Forced diuresis eliminates drugs that are:

- distributed in the plasma

- not bound to plasma proteins
- excreted in urine in active form.

Altering urinary pH increases the ionized fraction, thus decreasing renal tubular resorption by ion trapping.

Forced **alkaline** diuresis is used for certain drugs that are weak acids (e.g. salicylates, phenobarbital). Administer the following:

- 500 ml sodium bicarbonate 1.4%
- 1 l dextrose 5% in water
- 500 ml normal saline
- 20 mmol potassium chloride.

Treatment is over a period of 3 hours and the goal is to achieve urine output around 5 ml/kg/h and pH around 8.

Forced **acid** diuresis is used for drugs that are weak bases (e.g. amphetamines, phencyclidine). Administer the following at a rate of 1 l/h:

- 0.5 l dextrose 5% in water with 1.5 g ammonium chloride
- 0.5 l dextro saline.

Forced diuresis is contraindicated in cases of renal insufficiency, cardiac disease and hypokalaemia.

Dialysis
The indications for dialysis are:

- life-threatening poisoning due to a dialysable drug;
- poor body clearance due to underlying hepatic or renal disease;
- management of renal failure.

Haemoperfusion
Haemoperfusion, with either activated charcoal or resin columns, removes haemoperfusable drugs and toxins by adsorption. Table 12.1 shows the substances that can be removed by dialysis and haemoperfusion.

Exchange transfusion
Exchange transfusion is of limited clinical use in adults.

ANTIDOTES

There are very few effective antidotes that can be used in treating an overdose. Antidotes are indicated for diagnostic manoeuvre and to reverse the drug effects. Intensive supportive care remains the mainstay of poison management. Table 12.2 shows the uses and dosages of the available antidotes.

ANCILLARY MEASURES

Psychiatric evaluation or drug abuse counselling should be obtained for patients who have taken overdoses intentionally, or who are habituated to substance abuse.

Table 12.1 Substances removed by haemodialysis and haemoperfusion

Haemodialysable	Haemoperfusable	
Acetaminophen	Iodide	Acetaminophen
Aluminum	Iron deferoxamine	Amanita toxins
Amanita toxins	Isoniazid	Ammonia
Amikacin	Isopropyl alcohol	Amobarbital
Ammonia	Kanamycin	Bromide
Amobarbital	Lactate	Butabarbital
Amoxycillin	Lead edetate	Carbon tetrachloride
Amphetamines	Lithium	Chloral hydrate
Ampicillin	Magnesium	Creatinine
Aniline	Mannitol	Demeton
Arsenic	Meprobamate	Digoxin
Azathioprine	Methanol	Dimethoate
Barbital	Methaqualone	Ethanol
Borate	Methotrexate	Ethchlorvynol
Bromide	Methyldopa	Glutethimide
Calcium	Methylprednisolone	Meprobamate
Camphor	Methyprylon	Methaqualone
Carbenicillin	Metronidazole	Methotrexate
Carbon tetrachloride	Monoamine oxidase inhibitors	Methyprylon
Cephalosporins (most)	Neomycin	Nitrostigmine
Chloral hydrate	Nitrofurantoin	Paraquat
Chloramphenicol	Nitroprusside	Parathion
Chlorate	Ouabain	Pentobarbital
Chromate	Paraquat	Phenobarbital
Cimetidine	Penicilline	Salicylate
Cisplatin	Phenobarbital	Secobarbital
Citrate	Potassium	Theophylline
Creatinine	Primidone	Thyroxine
Cyclophosphamide	Procainamide	Uric acid
Demeton	Quinidine	
Diazoxide	Quinine	
Dimethoate	Salicylate	
Disopyramide	Sodium	
Ethambutol	Streptomycin	
Ethanol	Sulphonamides	
Ethchlorvynol	Theophylline	
Ethinamate	Thiocyanate	
Ethylene glycol	Ticarcillin	
Flucytosine	Tobramycin	
Fluoride	Urea	
5–Fluorouracil	Uric acid	
Gallamine	Water	
Gentamicin		
Hydrogen ions		

Table 12.2 Antidotes for drug and acute chemical poisoning

Antidote	Adult dosage	Usage
Acetylcysteine	140 mg/kg orally, followed by 70 mg/kg 4-hourly for 17 doses	Acetaminophen
Atropine sulfate	1–5 mg i.v. (i.m.) every 15 min as needed	Cholinergics Organophosphates
Benztropine mesylate	1-2 mg i.v. (i.m., orally) as needed	Extrapyramidal signs
Calcium disodium edetate	1 g i.v. (i.m.) over 1 h 12-hourly	Lead
Cobalt edetate	300 mg i.v. over 1 min	Cyanide
Deferoxamine mesylate	1 g i.m. (i.v. at a rate of 15 mg/kg/h if shock or coma) followed by 0.5 mg 4-hourly	Iron
Dimercaprol	2.5–5.0 mg/kg i.m. 4–6-hourly	Arsenic Gold Mercury Lead
Diphenhydramine hydrochloride	25–50 mg i.v. (i.m., orally) as needed	Extrapyramidal signs
Ethanol	11 ml/kg of absolute (100%) ethanol in 5% dextrose in water i.v. over 15 min, followed by 0.1–0.2 ml/kg/h to maintain a blood level of 100 mg/dl	Ethylene glycol Methanol
Methylene blue	1–2 mg/kg i.v. (0.1–0.2 ml/kg of 1% solution) over 5 min	Methaemoglobinemia
Naloxone hydrochloride	0.4–2.0 mg i.v. (i.m., s.c.) every 30–60 min, as needed	Opiates Diphenoxylate Pentazocine Propoxyphene
Nitrites		Cyanide
Amyl nitrite	Inhalation pearls	
Sodium nitrite	Every 1 min 300 mg i.v. (10 ml of 3% solution) over 3 min	
Oxygen	100%, hyperbaric	Carbon monoxide
Penicillamine	250–500 mg orally 6-hourly	Copper Mercury
Physostigmine salicylate	0.5–2.0 mg i.v. (i.m.) over 2 min every 30–60 min as needed	Anticholinergics
Pralidoxime chloride	1–2 g i.v. (orally) over 15–30 min as needed (usually 8–12-hourly to a maximum of three doses/24 h)	Organophosphates Anticholinesterases

Some common forms of poisoning and intoxication are discussed below. The patient should be handled and cared for in a specialized unit where possible. Intensive supportive care remains the most important principle in the management of acute poisoning.

12.2 COMMON FORMS OF POISONING AND INTOXICATION

12.2.1 SEDATIVE INTOXICATION

Clinical picture

Central nervous system depression occurs in a rostral caudal pattern; initially there is delirium, ataxia and nystagmus, followed by stupor and coma, with spasticity, decerebration and flaccidity. Response to painful stimuli becomes impaired and deep tendon reflexes are lost. The pupillary light reflex is preserved until the late stages of intoxication.

Depression of respiration, venous collapse, hypercapnia and hypotension may follow. Hypothermia may also occur. There may be seizures as a result of hypoglycaemia, hypoxia or simultaneous amphetamine abuse.

Diagnosis

A simple method for assessing blood barbiturate levels should be available.

Management

Management is the same as that for any comatose patient: establish a clear airway and ensure adequate oxygenation and support of circulation.

Charcoal haemoperfusion is probably the treatment of choice. Forced alkaline diuresis is only indicated for substantial overdoses of phenobarbitone and barbitone. Haemodialysis is the most effective method of removing all barbiturates. Attention to avoiding the complications ensuing from the comatose state is probably the most important aspect of the therapy.

12.2.2 NARCOTIC OVERDOSE

The narcotic drugs include heroin, morphine, Dilaudid, methadone, codeine, Demerol and propoxyphene (Darvon).

Signs and symptoms

- Respiratory depression, initially manifested by slow, deep breathing, but leading rapidly to apnoea
- Hypotension
- Stupor
- Coma
- Pinpoint pupils

Treatment

1. Maintain a clear airway.
2. Give oxygen.
3. Start an i.v. lifeline with dextrose 5% in water.
4. For a patient in a coma of unknown cause, the first medication should always be 50 ml of dextrose 5% in water i.v. stat. bolus until the blood glucose level is obtained.
5. If the patient fails to respond to improved ventilation and glucose injection, and if there is reason to suspect narcotic overdose, give naloxone.
6. If the patient remains comatose after naloxone, it may be that the overdose is of a mixed nature. Provide definitive airway care by intubating the patient.

12.2.3 AMPHETAMINE OVERDOSE

Amphetamine, dexedrine, methedrine and similar stimulant drugs are frequently abused.

Signs and symptoms

The patient displays excitement, loss of appetite, tachycardia, hypertension, sweating, dilated pupils and tremors. The patient may also demonstrate frank amphetamine psychosis, with paranoia and hallucinations. These patients are apt to be violent.

Treatment

1. Determine whether the patient is violent.
2. The technique of 'talking down' the patient may be useful.
3. Provide the patient with a place to recover; the hospital is often not a very good place for this. A quiet room in the house of a reliable friend, where concerned people will be available to reassure the patient, may be preferable.

4. If the blood pressure is significantly elevated, or cardiac dysrhythmias are present, or the patient is entirely out of control, hospitalization will be necessary.

12.2.4 SALICYLATE INTOXICATION

Clinical picture

The most common toxic effect of acetylsalicylic acid (ASA) is occult gastrointestinal bleeding. Toxicity with therapeutic doses will cause headache, tinnitus, hearing loss, nausea, vomiting, sweating, hyperventilation, flushing and restlessness. The clinical presentation of the patient with severe salicylate toxicity is similar to that of a patient with diabetic ketoacidosis, including hyperglycaemia, ketonuria and a positive urine reaction with Benedict's reagent.

Management

1. Blood samples should be taken for serum salicylate, haemoglobin and PCV, electrolytes and urea, arterial pH and $Paco_2$.
2. A forced alkaline diuresis should be started if:
   - the clinical condition is poor;
   - the salicylate level is more than 1.9 mmol (300 mg)/l in children and more than 4.6 mmol (750 mg)/l in adults;
   - more than 50 tablets have been ingested.
3. Alkalinization is unnecessary if the urine pH is already more than 8.
4. If indications for forced diuresis are absent, simply observe the patient closely and encourage oral fluids.
5. Pulmonary oedema may occur (see Chapter 4).

12.2.5 INGESTED POISONS

Ingested poisons usually remain in the stomach for only a short time. There is minimal absorption from the stomach; this takes place instead after the poison has passed into the small intestine. A patient who has ingested poison presents with an acute onset of unexplained illness, especially one characterized by abdominal pain, nausea, vomiting or CNS alterations.

History

- What was ingested?
- When was it taken?

- How much was taken?
- Has the patient or bystanders made any attempt to induce vomiting?
- Does the patient have a psychiatric history?

Physical examination

Observe the skin colour for unusual hues: flushed, red or 'cherry-red' skin, for example, may be indicative of carbon monoxide poisoning. Smell the patient's breath for the characteristic odour of petroleum products, alcohols or other suggestive aromas.

Management

The general principles of management are:

- Maintain the airway.
- Decide whether to make the patient vomit or not, on the basis of the substance ingested.

Specific procedures are as follows:

STRONG ACIDS

Strong acids include many toilet bowl cleaners, rust removers, phenol and hydrochloric acid.

Treatment

1. **Do not induce vomiting**
2. Give milk of magnesia, milk, or flour in water in an attempt to neutralize and dilute the acid.
3. Start an i.v. with normal saline.

CYANIDE

Cyanide poisoning may occur through ingestion (bitter almonds, seeds of cherries, apples, pears, or inhalation of gases generated in blast furnaces or of fumigants); it can also be absorbed through the skin.

Clinical picture

The patient may be confused or stuporous. The classic odour of bitter almonds on the patient's breath is suggestive but not diagnostic. Respiration is usually rapid and laboured in the early stages, but later may become slow and gasping. The pulse is usually thready

and rapid. Vomiting, coma and seizures are frequent. Cyanosis is rare, occurring chiefly as a terminal event.

Treatment

1. Establish an airway.
2. Administer 100% oxygen. Assist ventilation if necessary.
3. Give 300 mg cobalt edetate i.v. over 1 minute. Repeat after 5 minutes if necessary. Alternately, give sodium nitrite (Table 12.2).
4. Start an i.v. with dextrose 5% in water at a keep-open rate.
5. Monitor cardiac rhythm.

PETROLEUM PRODUCTS

Petroleum products include kerosene, lighter fluid, gasoline and furniture polish.

Clinical picture

Respiratory distress, including cough, choking, pulmonary oedema, and sometimes cyanosis, is present. There may be severe abdominal pain. The patient is prone to central nervous system symptoms, ranging from irritability to convulsions and coma. Hypoglycaemia is common. Cardiac arrhythmias may occur.

Treatment

1. **Do not induce vomiting**. If a very large volume (more than about 50 ml) of kerosene or gasoline has been ingested, potential toxicity to the brain and heart requires that the poison be eliminated.
2. Give 100% oxygen with good humidification.
3. Start an i.v. with dextrose 5% in water.
4. Monitor cardiac rhythm.
5. Anticipate massive secretions; have suction ready.

ORGANOPHOSPHATE POISONING

The organophosphates are commonly used as insecticides. Overdose, either intentional or accidental, is common. These compounds bind irreversibly to the cholinesterases in the body. The cholinesterases normally inactivate acetylcholine, so that when they are not working acetylcholine levels rise; the effect of organophosphate intoxication is due to an increase in the levels of acetylcholine.

Exposure to organophosphate compounds is through the skin,

by inhalation (usually accidental in farm workers) or ingestion (usually intentional in adults, accidental in children). They vary in toxicity, malathion being of low toxicity, parathion being of high toxicity.

Diagnosis

The overactivity of acetylcholine, most pronounced at autonomic postganglionic fibres, gives rise to the following symptoms:

- Bradycardia and hypotension
- Miosis
- Vomiting, abdominal colic and diarrhoea
- Dyspnoea due to airway obstruction and respiratory muscle dysfunction
- Bronchial hypersecretion, excessive salivation and sweating
- Fasciculation and convulsions
- Disorientation, progressing to coma and death.

The setting and clinical findings are usually sufficient to make the diagnosis. However, this can be confirmed by measuring the blood pseudocholinesterase level, which is reduced, commonly to very low levels. The breath also has a characteristic odour.

It is important to remember that serum pseudocholinesterase levels are neither a good marker of the severity of poisoning nor a good predictor of recovery. Red-cell cholinesterase levels, reflecting tissue cholinesterases, are considerably more helpful in this regard. Even so, these levels can be depressed to 60% of normal without clinical signs, especially if the patient has been handling organophosphates previously.

Management

Non-specific

1. The most common immediate cause of death is respiratory failure. Check the vital capacity and blood gases hourly. This will give an indication of the progression of the illness. Respiratory failure, as defined by abnormal Pa_{O_2} and Pa_{CO_2}, requires ventilatory support. Remember that sudden deterioration in the respiratory status is common, hence the need to measure vital capacity, peak flow and blood gases regularly.
2. Remove contaminated clothing.
3. Bathe the patient. Organophosphates are excreted in sweat, and may be reabsorbed through the skin. Some authorities recommend hourly bathing.

4. The patient may be hypovolaemic due to excessive fluid loss through diarrhoea, sweating and salivation. Appropriate circulatory support, monitored using a CVP line, should be given. Remember that the lungs may appear 'wet'; this is not due to fluid overload but to excessive bronchial secretions, which may be controlled with atropine.

Specific

Atropine is the mainstay of treatment. Start with 1.2–2.0 mg i.v. stat. and then at 10-minute intervals. This order of dose will almost certainly have to be used for the first 24 hours; after this it may be possible to reduce it. Monitoring is on the basis of the development of atropine side effects. None is absolutely reliable, but a dry mouth, a pulse rate above 100 beats/min, and decreasing secretions in the lungs indicate satisfactory atropinization. When these features appear, the dose may be reduced to 0.6–1.0 mg and the interval between doses increased to 20–30 min.

It may be necessary to continue atropine for up to 2 weeks. After each 6–12 hour period, provided there is adequate atropinization, further increase the dose intervals to 1 and 2 hours. Atropine may then be given orally 4-hourly, and then gradually reduced over a 2-week period.

Atropine, while blocking the effects of acetylcholine at postganglionic nerve endings, does not affect brain cholinesterase. If the patient is seen within 12 hours of the overdose, give an oxime which **does** have an effect on brain cholinesterase. Oximes act by deconjugating the organophosphate from the cholinesterase enzymes, a process which becomes more difficult once more than 12 hours have elapsed after the overdose. Use either obidoxime or pralidoxime. Obidoxime is the most potent preparation available and it crosses the blood–brain barrier. Give 3–6 mg/kg i.v., repeated 4-hourly for 12–24 hours, depending on the response. Pralidoxime is an acceptable alternative. Give 30–60 mg/kg i.m. or i.v. 4-hourly (do not exceed 500 mg/min) for 12–24 hours, depending on response.

Atropine does not have a predictable effect on the disorientation and confusion induced by organophosphates. As an excess of atropine may also cause confusion, you may be faced with a confused, disorientated and sometimes violent patient, and not know whether this is due to an excess of atropine or not.

12.2.6 CARBON MONOXIDE POISONING

Causes

- Accidental: poor housing conditions, improperly adjusted and vented gas cylinders, appliances and fires
- Intentional: exposure to car exhaust fumes

Pathogenesis

In carbon monoxide poisoning, the system for improving oxygen transport is profoundly impaired. Carbon monoxide has an affinity for haemoglobin 240 times greater than that of oxygen. This is due to a slower rate of dissociation of carbon monoxide from the haemoglobin molecule. The formation of carboxyhaemoglobin results in a reduction in the amount of oxyhaemoglobin. In addition, carboxyhaemoglobin has a reduced affinity for 2,3-DPG, and at high levels of carbon monoxide 2,3-DPG production may be suppressed. Thus carbon monoxide is responsible for both hypoxia and suppression of adaptive mechanisms. Finally, as tissue oxygen tension falls, more carbon monoxide is bound to myoglobin and cytochrome oxidase, further impairing cellular respiration.

Clinical picture

Chronic sublethal poisoning produces alterations in mental and physical performance, particularly in patients with cardiorespiratory compromise. Acutely, the poison may rapidly produce coma and death.

Signs and symptoms

These depend on the type of exposure and the clinical status of the victim.

- At blood levels of 20–30%: headache, nausea, and vomiting.
- At higher levels: vertigo, tachypnoea and tachycardia, followed by seizures and coma.

There is no correlation between the symptoms and the blood carboxyhaemoglobin level. The two tissues most sensitive to lack of oxygen, the brain and myocardium, are the most affected. ECG changes may be immediate or delayed, with ischaemia, infarction, arrhythmias and conduction defects.

Erythematous and vesicular skin lesions may be seen at pressure

sites and should suggest the possibility of underlying muscle damage. The skin lesions show sweat gland necrosis on biopsy.

Adverse signs include congestive heart failure, leucocytosis, fever, metabolic acidosis and coma.

Treatment

1. Remove the patient from the toxic environment.
2. Ensure rapid reduction of gas levels by ventilation with 100% oxygen. Hyperbaric oxygen is the quickest method of lowering carbon monoxide levels.
3. Monitor for cardiac or skeletal muscle necrosis and cerebral oedema.
4. Fever, with the concomitant elevation of metabolic rate, should be treated.

12.3 BITES AND STINGS

12.3.1 HUMAN BITE WOUNDS

Human bite wounds are frequently seen days after the injury, when home remedies have failed and the wound has become infected. The treatment of human bite injuries depends on the type of wound, the location, and the length of time after the occurrence. Human bites can be penetrating or avulsive in type, or a combination.

Successful therapy includes copious cleansing of the wound, aggressive debridement of damaged tissue, and removal of foreign material. Closure should be considered for wounds of the nose, eyelids, ears, or lips. However, wound sepsis is a frequent occurrence following human bites, particularly in the extremities, where the blood supply is not as abundant as in the facial region. Therefore, in general, human bite wounds should be cleansed, debrided and covered with a dressing, not sutured.

The most common human bite wound infections occur with streptococci and penicillin-resistant *Staphylococcus aureus*. Therefore, prophylactic antibiotics should be selected with these specific bacteria in mind.

Human bite wounds of the extremities are particularly worrisome, typically located over the metacarpophalangeal joints and fingers. These are usually the puncture-type, with penetration of the underlying joint space or bone; deep sepsis, tenosynovitis and osteomyelitis may result. Time is an important factor in the

management of human bite wounds. When the patient presents late, incision and drainage of loculated pus, dressing, strict immobilization and elevation of the extremity, along with systemic antibiotics, are all indicated. Give tetanus toxoid.

12.3.2 ANIMAL BITE AND STING WOUNDS

Animal bites should be considered in the same category as human bites, with the additional and potentially serious problem of rabies.

DOG BITES

Dog bite wounds must be well irrigated and thoroughly debrided if primary healing is to be expected. The cheek region is a typical location for a dog bite. Although there may appear to be a number of small puncture wounds, on close inspection large subcutaneous dead space may be found below the skin. If such a cavity is present, a drain should be placed and brought out through one of the wounds. The traumatized edges of dog bite wounds may be trimmed; this will remove damaged tissue and contaminating bacteria. Puncture-type wounds should not be sutured, nor should most dog bite wounds of the extremities. However, with proper wound toilet, most bites of the facial area can be closed using a minimum number of sutures. Antibiotics are routinely given; the bacteria in dog saliva are sensitive to penicillin. Tetanus toxoid should be given.

Rabies

Rabies is a potentially dangerous complication of any dog bite. Knowledge of the health of the animal involved is important. If it is not known, the physician is faced with the decision of whether or not to begin rabies vaccination. The animal should be confined and observed for signs. If it is rabid, or has escaped capture, then treatment should be started immediately. There are two methods of treatment: vaccination with duck embryo vaccine, and antirabies immunoglobulin.

For immediate therapy where there is a short incubation period, passive immunity is confirmed using human rabies immunoglobulin, which is given in a single dose, 20 iu/kg. When feasible, half the injection should be infiltrated around the site of injury and the remainder given i.m. The duck embryo vaccine is given in 23 daily 1 ml doses, followed by a 1 ml booster dose 10 and 21 days after completion of the series. The

vaccine should be injected subcutaneously in rotating sites. Therapy may be discontinued if the laboratory examination of the animal is negative.

CAT BITES

Cat wounds should be cleansed and topical antibacterial ointment applied. *Pasteurella multacida*, a small Gram-negative rod, is a common pathogen in cat as well as dog bites. It is sensitive to penicillin, tetracycline and preventive antibiotics, and should be given to a patient with a deep cat scratch or bite wound.

SNAKE BITES

Bites from non-venomous snakes characteristically leave multiple small puncture marks, similar to scratches. There is only minimal local swelling and slight discomfort about the injured skin, with no systemic effects. Non-venomous snake bites should be cleansed and the patient given tetanus toxoid.

The bite of a poisonous snake results in envenomation in about two-thirds of cases. First there is severe pain in the bitten area, which then becomes swollen and ecchymotic, with rapid appearance of skin necrosis and sloughing of tissue. Severe envenomation is signalled by a more rapid onset of local signs and systemic symptoms, e.g. nausea, vomiting, thirst, sweating and fever. Hypotension, cyanosis, convulsions, cardiopulmonary collapse and coma can occur.

Suction is effective for the removal of substantial amounts of venom (up to 50%) from the wounds. It should be started after the skin incision and continued for 1 hour. Over 90% of snake bites occur in the limbs; in such cases the limb should be immobilized and elevated. A constricting band should be applied proximal to the bite area to impede superficial venous and lymphatic drainage; the distal pulse should be monitored.

If the dead snake has been brought in and identified, or the fang marks are identifiable, antivenin should be administered. Antivenins are available in horse serum, so reactions – anaphylaxis and serum sickness – may occur.

Early debridement of ecchymotic, necrotic and haemorrhagic tissue surrounding the puncture site can be effective. The open wound is dressed and covered later with a split-thickness skin graft. Fasciotomy may be beneficial. Since snakes harbour tetanus bacilli in their mouths, tetanus toxoid is indicated. Broad-spectrum antibiotics should also be given.

INSECT STINGS

Hypersensitivity and anaphylaxis due to systemic reactions to insect stings (bees, wasps, scorpions, spiders) cause higher mortality than do snake bites.

Treatment begins with removal of the sting, taking care not to squeeze the area. The wound should be cleansed with soapy water, and ice should be applied. Allergic reactions may be immediate (local or generalized, even anaphylaxis) or delayed, with serum sickness symptoms. If there is the slightest manifestation of an allergic response, subcutaneous epinephrine (1:1000) 0.4–0.5 ml should be given, and the area massaged vigorously to hasten absorption. The patient should be watched closely and a repeated dose may be needed after 20–30 minutes. If there are signs of glottal oedema or severe hypotension, i.v. therapy, antihistamines and steroids may be needed. Scorpion antivenom is available against scorpion stings.

MARINE ANIMAL STINGS

These may be caused by stingrays, jellyfish, sea urchins, tropical fish, corals and other spiny venomous marine species. They possess thermolabile venoms, and therefore heat application rather than the usual ice application, is the primary therapy.

In general, marine venoms are simpler and shorter-acting than those of terrestrial animals. A tourniquet should be applied for extremity stings and the wound inspected for stingers or tentacles, irrigated and debrided. Pain medication is given. Antihistamines and corticosteroids may be of help if the venom effect increases.

13

Obstetric emergencies

Summary of contents

EMERGENCIES DURING PREGNANCY 280

Hyperemesis gravidarum 280
Vaginal bleeding 281
Bleeding in the last trimester 286
Hypertension 287
Convulsions 288
Premature rupture of membranes 289

EMERGENCIES DURING LABOUR 290

Normal labour 290
Abnormal deliveries 292
Breech presentation 292
Prolapsed umbilical cord 294
Twin births 295
Premature births 295
Uterine rupture 295

EMERGENCIES FOLLOWING LABOUR 296

Postpartum haemorrhage 296
Retained placenta 296
Inadequate uterine contractions 296
Clotting disorders 296
Pyrexia 297
Pulmonary embolism 297

Emergency cases in obstetrics are divided into three categories:

- Emergencies during pregnancy
- Emergencies during labour
- Emergencies after labour.

13.1 EMERGENCIES DURING PREGNANCY

- Hyperemesis gravidarum
- Vaginal bleeding
- Hypertension
- Convulsions
- Premature rupture of membranes

13.1.1 HYPEREMESIS GRAVIDARUM

Hyperemesis gravidarum is excessive vomiting in early pregnancy that affects the general health of the patient. It occurs especially in primigravidae. Its aetiology is as yet unknown, and is mostly attributed to psychological factors.

Diagnosis

Hyperemesis gravidarum must be differentiated from morning sickness, which occurs in the great majority of pregnant women. Organic causes such as peptic ulcer, acute appendicitis or cholecystitis should also be excluded. Hyperemesis is characterized by the following symptoms and signs:

- It persists throughout the daytime, and even wakes the patient during the night.
- It is severe enough that the gastric and even the intestinal contents are vomited.
- It is usually accompanied by general fatigue, inability to do housework and loss of weight; sometimes there are cramps and, in severe cases, a tinge of jaundice appears in the eyes. There may be disturbed consciousness, irritability, dehydration, tachycardia and hypotension, tachypnoea, and oliguria with acetone in the urine.

Management

Mild cases can be treated at home as follows:

1. Fluids should be given by mouth in sips.
2. Solid food should be given in small amounts and repeated at short intervals.
3. The diet should contain a lot of carbohydrate and vitamins; fats should be cut down.

4. Antiemetics, e.g. metochlopramide or cortigen B_6, may be given as necessary.

Severe cases should be hospitalized. These cases have the following signs and symptoms:

- Rapid pulse rate above 100/min
- Blood pressure less than 90/60
- Temperature above 38°C
- Rapid respiratory rate
- Impaired consciousness
- Acetone in urine
- Signs of dehydration or oliguria below 500 ml/24 h
- Possible slight jaundice.

In such cases fluids should be given i.v. as follows:

- 5% glucose in normal saline 0.5 l
- Lactated Ringer's solution 0.5 l
- 100 ml glucose 25%
- 100 mg hydrocortisone.

13.1.2 VAGINAL BLEEDING

Causes

BLEEDING DURING THE EARLY MONTHS

Causes related to pregnancy:
- Abortion (Fig. 13.1)
- Ectopic pregnancy (Fig. 13.2)
- Vesicular mole (Fig. 13.3)

Causes unrelated to pregnancy:

- Cervical erosion and ulcer
- Varicosity in the cervix or vagina
- Cancer of the cervix.

BLEEDING DURING THE LATER MONTHS

Causes related to pregnancy:

- Placenta praevia (Fig. 13.4)
- Accidental haemorrhage (Fig. 13.5).

Causes unrelated to pregnancy:

- Cervical erosion and ulcer
- Varicosity in the cervix or vagina
- Cancer of the cervix.

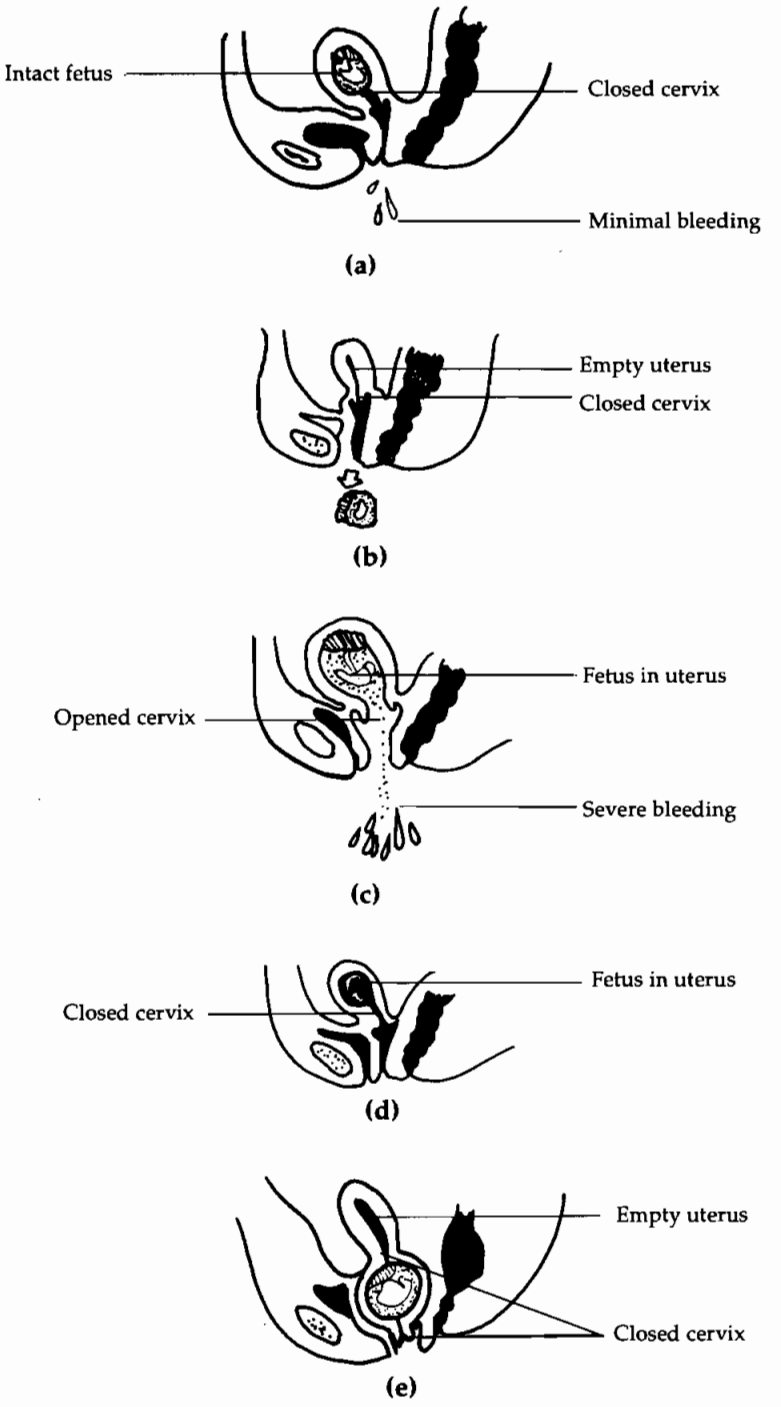


Fig. 13.1 Abortion (a) threatened; (b) complete; (c) inevitable; (d) missed; (e) cervical.

Fig. 13.3 Ectopic pregnancy.

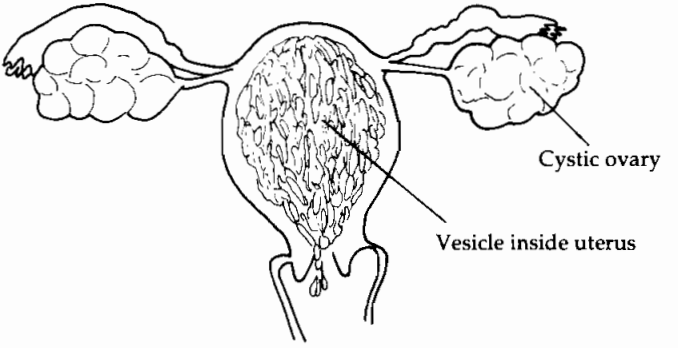


Fig. 13.3 Vesicular mole.

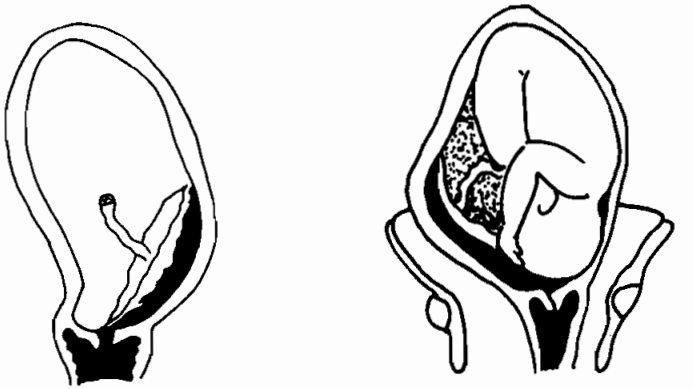


Fig. 13.4 (a) Placenta praevia; (b) placenta praevia marginalis.

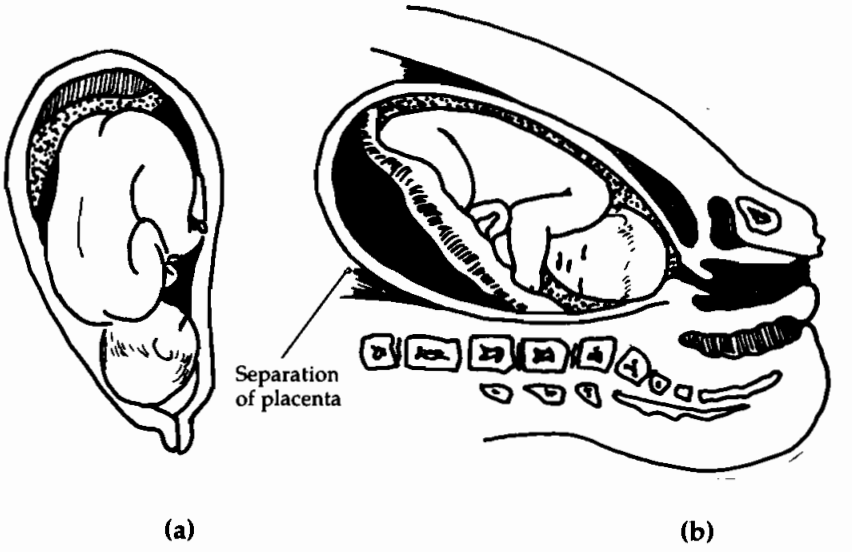


Fig. 13.5 Accidental haemorrhage.

Diagnosis

In cases of bleeding during the early months, an accurate history should be taken together with examination and investigative tests.

HISTORY

- Date of the last menstruation, to evaluate duration of pregnancy
- Nature of the bleeding
- Mild bleeding indicates threatened abortion (Fig. 13.1a), ectopic pregnancy or missed abortion (Fig. 13.1d).
- Severe bleeding indicates inevitable abortion (Fig. 13.1c) or vesicular mole.
- Colour: bright red indicates threatened abortion; dark red indicates inevitable abortion, missed abortion or vesicular mole.
  Tissue presence:
  fetal tissue in abortion
  vesicles in vesicular mole
  endometrium in ectopic pregnancy.
- Presence of pain
  Mild in threatened and missed abortion
  Severe, colicky in inevitable abortion
  Stitching pain on one side, may radiate to the shoulder, in

ectopic pregnancy
Pain precedes bleeding in abortion; follows bleeding in ectopic pregnancy.

- Pyrexia
  Mild rise of temperature in vesicular mole
  High fever in septic abortion.
- Always ask about the use of contraceptive devices, as there is a high incidence of ectopic pregnancy with such devices.
- Pain on micturition or defaecation occurs with ectopic pregnancy.
- Collapse is common if external bleeding is severe in abortion or with internal bleeding in ectopic pregnancy.

EXAMINATION

1. Take pulse rate, blood pressure and temperature to detect if shock prevails.
2. Look for signs of pregnancy in the face, breasts and abdomen.
3. Examine the abdomen for signs of internal haemorrhage.
4. Examine the abdomen for size of the uterus to determine the duration of gestation, and compare it with the date of last menstruation. If there is vesicular mole, the size of the uterus is larger than would normally be expected.
5. Vaginal examination:
   - Using strict aseptic technique determine the nature of the bleeding.
   - Look for ectopic pregnancy by feeling the adnexa. Swelling on one side and eliciting of pain on examination and on moving the cervix are sure signs of ectopic pregnancy.
6. Examine the cervix. It is closed in threatened abortion, ectopic pregnancy, missed abortion and cervical abortion; open in incomplete and inevitable abortion.

Never do a vaginal examination on any woman with third-trimester bleeding.

INVESTIGATIONS

- Urine analysis to test for pregnancy
- Sonogram for accurate diagnosis of vesicular moles, all types of abortion and ectopic pregnancy
- Listen for fetal heart sounds

Management

Threatened abortion can be treated at home with general sedatives, uterine sedatives and complete bed rest. Cases of vesicular mole, ectopic pregnancy and all types of abortion, except threatened and complete abortion, should be transferred to hospital.

13.1.3 BLEEDING IN THE LAST TRIMESTER

Causes

- Placenta praevia
- Accidental haemorrhage
- Causes unrelated to pregnancy, e.g. erosion of cervix, varicosities of cervix or vagina, cancer of the cervix

Diagnosis

HISTORY

- Duration of pregnancy
- Fetal movements
- Number of bleeding episodes. Bleeding is repetitive in cases of placenta praevia
- Amount of bleeding. This depends on the position of the placenta in relation to the internal os in cases of placenta praevia.
- Accidental haemorrhage is due to a sudden rise in blood pressure as a result of trauma to or pressure on the abdomen. It usually occurs in the primigravida, whereas bleeding from placenta praevia is more frequent in multigravidae. The amount of bleeding in cases of accidental haemorrhage does not indicate the severity of the condition, as the amount of external bleeding is minimal whereas there is severe bleeding in the uterus and behind the placenta.
- Pain and tenderness of the abdomen is a sign of concealed haemorrhage in cases of accidental haemorrhage.

EXAMINATION

1. Take blood pressure and pulse.
2. Perform an external abdominal examination of the uterus. The uterus is contracted in cases of accidental concealed haemorrhage.
3. Establish that there are fetal heart sounds. This is important to ensure the viability of the fetus. They may be easy to hear or may even be absent in cases of concealed haemorrhage.

Never do vaginal examination in these cases unless the patient is in the operating theatre, as severe haemorrhage can be initiated.

INVESTIGATIONS

- Haemoglobin estimation
- Bleeding time
- Coagulation time
- Blood grouping
- Urine analysis
- Sonography

Management

If the patient is in the last trimester of pregnancy she should be transferred to hospital immediately, regardless of the amount of bleeding. An i.v. line should be set up in all cases, and plasma substitutes or even 5% glucose or saline administered to partially replace the blood loss. Blood transfusion is preferable. Sedatives such as pethidine can be given.

13.1.4 HYPERTENSION

Hypertension is considered to be present if:

- at any time blood pressure is more than 130/90;
- the systolic pressure rises by 30 mmHg or the diastolic by 15 mmHg above the standard.

Types

The following conditions cause hypertension during pregnancy:

- Eclampsia and pre-eclampsia (Fig. 13.6)
- Essential hypertension
- Chronic nephritis
- Transient hypertension.

The differentiation between the above causes of hypertension during pregnancy is determined by history and clinical examination. There is no family history of hypertension in cases of toxaemia of pregnancy.

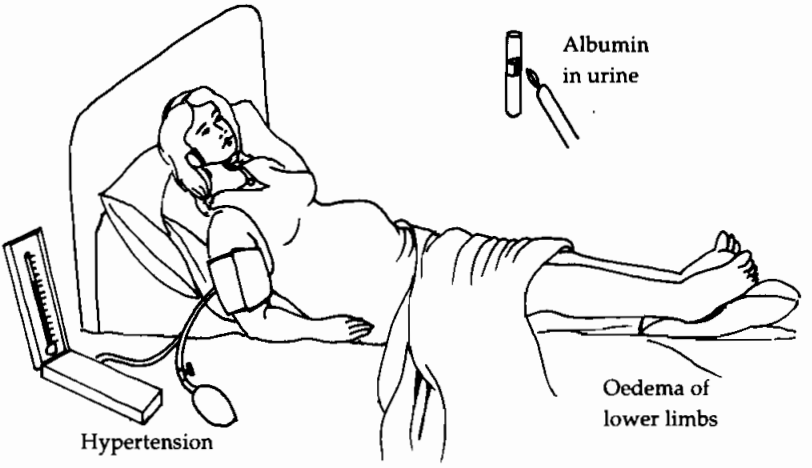


Fig. 13.6 Eclampsia and pre-eclampsia.

Diagnosis

Headache, increase in weight, oedema of the lower limbs and blurring of vision.

INVESTIGATIONS

- Urine analysis for albumin and casts
- Blood samples for blood urea, serum creatinine and uric acid

Management

It is always better to hospitalize a pregnant woman with hypertension for fear of the development of the full-blown picture of eclampsia, which is characterized by dangerous convulsions.

13.1.5 CONVULSIONS

The most common cause of convulsions during pregnancy is toxaemia of pregnancy (eclampsia). Other causes include:

- vascular disorders in the brain
- renal failure
- drop in serum calcium
- hypoglycaemia
- idiopathic causes.

Convulsions due to eclampsia

First stage The convulsion occurs for a few seconds, in the form of mild contractions in the muscles of the face and sudden movements of the eyes and sometimes the limbs.

Second stage The convulsion involves all the muscles of the body for about half a minute. This includes the respiratory muscles, resulting in apnoea and cyanosis. The patient may fall to the ground and hurt herself.

Third stage The convulsion lasts for a few minutes and takes the form of clonic convulsions that may permit some respiratory movement; blood-stained froth may appear in the mouth.

Fourth stage The convulsion lasts from a few minutes to a few days; the patient is in complete coma.

Management

1. The patient should be nursed in a dark room and kept calm and quiet.
2. See to the safety of the patient during the convulsions by keeping the airway clear with continuous suction. Place a spatula over the tongue to prevent the patient biting it.
3. Stop the convulsions by giving diazepam 5–10 g slowly i.v.
4. Give oxygen.
5. Treat hypertension.
6. Hospitalize the patient as soon as possible.

13.1.6 PREMATURE RUPTURE OF MEMBRANES

Diagnosis

- History of a gush of water from the vagina.
- Detection of embrionic cells in this water, stained orange by methylene blue stain. In 90% of cases labour starts within 24 hours.

Management

1. Transfer to hospital.
2. Bear in mind the possibility of cord prolapse.
3. Induce labour in full-term cases. If not, keep the patient under observation and give broad-spectrum antibiotics until full term is reached. Then induce labour.

13.2 EMERGENCIES DURING LABOUR

- Normal labour
- High-risk labour
  - Breech presentation
  - Prolapsed umbilical cord
  - Multiple births
  - Premature births
- Uterine rupture

13.2.1 NORMAL LABOUR

There are three stages to labour. The **first stage** begins with the onset of uterine contractions and ends when the cervix is fully dilated. It may last from 5 to 18 hours in a primigravida. The **second stage** begins with full dilatation of the cervix and ends with complete delivery of the baby. It lasts from 10 minutes to 1 hour. The **third stage** is the expulsion of the placenta and its membranes. It lasts from a few minutes to half an hour.

Management

With the commencement of the first stage it is necessary to differentiate between true and false labour pains. Keep the case under observation for 2 hours. If the pains disappear they are false; if they increase in intensity and frequency, they are true labour pains.

Generally speaking it is advisable to complete the process of delivery in a hospital once labour has started. However, it is left to the physician to judge the necessity of transferring the mother to hospital, based on:

- the presence of true labour pains and the stage, and whether the mother is a primigravida or a multipara;
- fetal position and fetal heart sounds;
- size of the cervical dilatation;
- condition of the membranes (intact or ruptured) or if there is crowning of the head;
- distance to the nearest hospital and availability of transport.

If the mother is in the second stage of labour:

- Do not allow her to go to the toilet.
- **Never** attempt to delay or to restrain delivery in any fashion.

Proceed as follows:

1. Keep her on her back, bend her knees and spread her thighs apart. Have an assistant start an i.v. line. Prepare a source of

oxygen and suction. Wash your hands thoroughly with antiseptic and put on sterile gloves. Drape the mother with four towels except for the vaginal opening. Reassure her and encourage her to relax and take slow, deep breaths through her mouth. If the baby is coming fast, forget about towels and gloves and control the delivery.

2. When the baby's head begins to emerge from the vagina support it gently to prevent explosive delivery. No attempt should be made to pull the baby from the vagina. Be sure the umbilical cord is not wrapped around the baby's neck; if it is, try to slip it gently over the shoulder and head. If this manoeuvre fails, or the cord is wrapped tightly around the neck, place umbilical clamps 5 cm apart and cut the cord between the clamps. Then deliver the shoulders and body, supporting the head at all times. When the baby is fully delivered, keep the head lower than the body to aid drainage.
3. Remember, babies are slippery. Wipe away any blood and mucus from the nose and mouth with sterile gauze and aspirate the mouth and both nostrils with any sort of mild aspiration.
4. If the baby does not breathe spontaneously at this point, stimulate by gentle slapping on the back and soles. If there is still no response, start mouth-to-mouth resuscitation with very small puffs. If spontaneous breathing begins, administer oxygen by mask for a few minutes until the baby's colour is pink.
5. If breathing is still absent and no precordial pulse is present, begin complete cardiopulmonary rescuscitation by cardiac compression and artificial respiration.
6. If the infant has been delivered normally and is breathing well, the cord should be tied (or clamped) about 20 cm from the navel, with two ties (or clamps) placed 5 cm apart. Cut the cord between the two ties and wrap it in sterile gauze.
7. Examine the baby for congenital anomalies and weigh it.
8. Wrap the baby in a sterile blanket and keep it warm at all times.
9. Delivery of the placenta occurs within 20 minutes and bleeding may be expected as the placenta separates from the wall of the uterus. Always massage the abdomen over the uterus to aid contraction. **Never** pull on the umbilical cord to hasten delivery of the placenta. Examine the placenta for completeness; retained pieces of placenta will cause persistent bleeding and sepsis.
10. Examine the perineum for laceration and repair any superficial tears. If there are deeper tears transfer the patient to hospital.

11. If the placenta is not delivered within 30 minutes transfer the mother and baby to hospital without further delay.
12. After delivery of the placenta give the mother oxytocin 10 iu added to the i.v. drip.
13. Place a sanitary pad over the vaginal opening, lower the mother's legs and transfer her to the ward. Observe for the next three days.

13.2.2 ABNORMAL DELIVERIES

Generally speaking it is essential to transfer the mother to hospital if any of the following are present:

- History of caesarean section or stillbirth;
- Abnormal presentation such as face, breech, transverse lie, prolapsed umbilical cord, prolapsed arm, obstructed labour with dead fetus;
- Multiple birth;
- Premature rupture of membranes of more than 24 hours;
- Toxaemia of pregnancy;
- History of diabetes, heart, renal or liver disease;
- History of fibroid tumours;
- Mother less than 150 cm in height or suffering from a limp;
- Signs of a difficult labour:
  General signs: rise in temperature, rapid pulse, high blood pressure, dryness of mouth and throat, signs of shock;
  Local signs: dryness of the vagina, thinning of the lower segment of the uterus, swelling and oedema of the cervix;
  Signs of fetal distress: caput succedaneum, pulse rate above 160/min or less than 100/min, passage of meconium with the amniotic fluid, weak fetal heart sounds.

Management

1. Transfer the mother to hospital.
2. Give sedatives e.g. pethidine.
3. Start an i.v. line and give crystalloids.
4. Give antibiotics.

BREECH PRESENTATION

A breech presentation (Fig. 13.7) is when the buttocks appear first rather than the head. The normal birth position is head first.

1. If a breech presentation is diagnosed before the cervix is fully dilated, transfer the mother to hospital immediately.
2. If delivery is imminent, prepare the mother as usual and allow the buttocks and trunk of the baby to deliver spontaneously. Once the legs are clear, support the baby on the palm and volar surface of your arm and allow the baby's head to deliver.
3. If the head is not delivered within 3 minutes, take action to prevent suffocation of the baby, whose umbilical cord is compressed by the head in the birth canal and whose face is pressed against the vaginal wall. Place a gloved hand in the vagina, with the palm towards the baby's face. Form a V with the fingers on either side of the baby's nose, and push the vaginal wall from the baby's face until the head is delivered.
4. Do **not** attempt to pull the baby out. Do **not** allow explosive delivery. If the head does not deliver within 3 minutes of establishing the airway, transfer rapidly to the hospital, with the mother's buttocks elevated on pillows and with the baby's airway maintained throughout in the manner described.
5. Alert the hospital of the situation in advance.

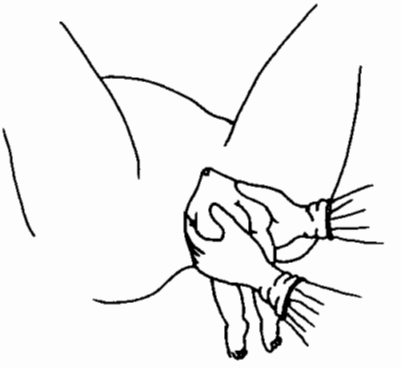


Fig. 13.7 Breech presentation.

Important: If the head is still not delivered, adopt the Mauriceau–Smellie–Veit technique for delivery of the head of a baby in a breech presentation: put the left hand below the baby with the index finger in its mouth and the right hand on its back, with out-stretched fingers on its shoulders, the middle finger lying on its head to keep the head flexed (Fig.13.8). This method should be tried in all breech presentations, especially if there is some delay in delivering the head.

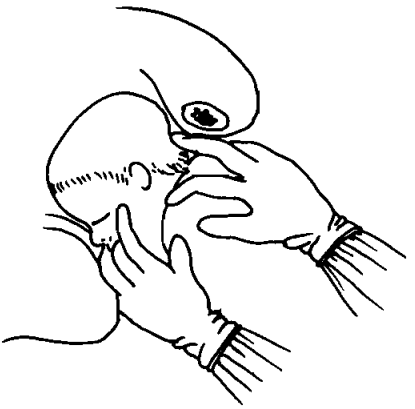


Fig. 13.8 Mauriceau–Smellie–Veit technique.

PROLAPSED UMBILICAL CORD

This refers to the situation in which the umbilical cord comes out of the vagina before the baby. The baby is thereby placed in great danger of suffocation (Fig. 13.9).

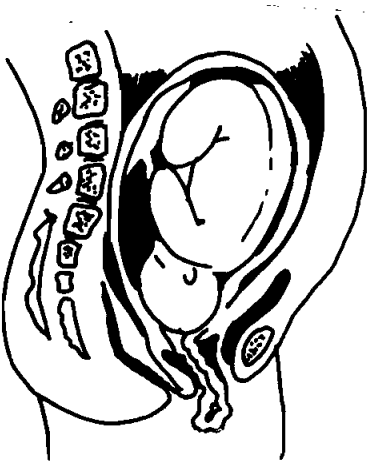


Fig. 13.9 Premature rupture of membranes and prolapsed cord.

1. Place the mother in the supine position with the hips elevated on a pillow.
2. Give the mother oxygen.

3. With a gloved hand, gently push the baby up the vagina several centimetres.
4. Do not attempt to push the cord back.
5. Transfer the mother to hospital at once, maintaining support on the baby's head and total elevation of the mother's legs and buttocks to aid the retention of the cord in the uterus.

TWIN BIRTHS

Twin and multiple births are not a problem in general, and are delivered in the same manner as single babies. Anticipate twins if the mother's abdomen appears unusually large, or if it remains large after the first baby is delivered. When the first baby is born, tie off the cord to prevent haemorrhage from its twin. If the second baby is not delivered within 10 minutes of the first, transfer to hospital. Twin babies tend to be small and, like premature infants, need special precautions against a fall in temperature.

PREMATURE BIRTHS

Any baby born before the mother has completed 7 months of pregnancy, or weighing less than 2.5 kg, is defined as premature and needs special care.

1. Wrap the baby up to keep it warm.
2. Clear the nose and mouth gently.
3. Prevent bleeding from the umbilical cord.
4. Give the baby oxygen.
5. Prevent infection.
6. If possible, put the baby in an incubator.

13.2.3 UTERINE RUPTURE

Uterine rupture is a complication of difficult labour and is manifested by sudden severe abdominal pain, with rapid development of profound shock due to internal haemorrhage, although bleeding may not be apparent externally. Always bear this possibility in mind if faced with a difficult labour. Patients with a previous history of caesarian section are at high risk of uterine rupture.

1. Transfer the mother to hospital on a stretcher immediately, preferably lying on her side.
2. Give sedation, e.g. pethidine.
3. Take a blood sample for blood grouping and matching.

4. Start two large-bore i.v. lines. Administer crystalloids or colloids as rapidly as needed to maintain blood pressure.

13.3 EMERGENCIES FOLLOWING LABOUR

13.3.1 POSTPARTUM HAEMORRHAGE

Postpartum haemorrhage may be caused by:

- a retained placenta
- inadequate uterine contractions
- clotting disorders.

RETAINED PLACENTA

1. If the patient is in shock, treat with i.v. fluids.
2. If she is not in shock and the placenta is retained for more than 30 minutes, introduce a urinary catheter and empty the bladder.
3. Start an i.v. line with 10 iu oxytocin.
4. Apply abdominal massage to the uterus.
5. If severe bleeding is still present, try to deliver the placenta manually, very gently.
6. Even if there is no severe bleeding but the placenta is retained, transfer the patient to hospital.

INADEQUATE UTERINE CONTRACTIONS

1. Start an i.v. line and take a blood sample for blood grouping.
2. Exclude the presence of cervical and vaginal tears, which should be repaired immediately or packed firmly.
3. Exclude the presence of retained placental parts, which must be evacuated.
4. If no apparent cause of bleeding is detected, then uterine inertia is diagnosed and uterine stimulants are given i.v. e.g. oxytocin, methergine and prostaglandin are tried at the same time as continuous external uterine massage.

It is always preferable to transfer the patient to hospital where there are better facilities, but in the meantime try to resuscitate the patient.

CLOTTING DISORDERS

Clotting disorders are discovered by the laboratory and the deficient factor is given.

13.3.2 PYREXIA

A temperature as high as 38°C may be physiological for the first day after delivery. After that it is considered pathological. It may be due to:

- congestion of the breast with milk
- puerperal sepsis
- thrombophlebitis of the leg veins
- urinary tract infection
- pneumonia.

Management

The patient should be examined thoroughly to differentiate between the above causes. If **milk congestion** is diagnosed as the cause:

1. Apply hot fomentation locally.
2. Encourage suckling.
3. Apply milk suction using a breast pump.
4. Give antibiotics and antipyretics.

If **puerperal sepsis** is diagnosed, transfer the patient immediately to the fever hospital.

If **deep vein thrombosis** is diagnosed from the presence of swollen lower limbs and tender calves, transfer the patient to hospital. Prevent her from moving. Advise her to keep her legs on a pillow and not to wear tight garments. Give heparine i.v. 5000 iu, and analgesics.

13.3.3 PULMONARY EMBOLISM

The sudden occurrence of dyspnoea, tachycardia or shock in the postpartum period may signal pulmonary embolism, due either to a blood clot or to amniotic fluid.

Treatment

1. Transfer the mother to hospital immediately. In the meantime give oxygen by mask.
2. Start an i.v. lifeline.
3. Try to maintain blood pressure with fluids and inotropics.
4. Give a loading dose of heparine 5000–10 000 iu as a start to the anticoagulant therapy in hospital.

14

Acquired immunodeficiency syndrome (AIDS)

Summary of contents

MODES OF TRANSMISSION 299

AETIOLOGY 301

INCUBATION PERIOD 302

CLINICAL MANIFESTATIONS 302

CLINICAL CASE DEFINITION 305

PREVENTION AND CONTROL 305

SAFETY PRECAUTIONS FOR MEDICAL PRACTITIONERS 309

ANTIBODY TESTING 311

MANAGEMENT PLAN 311

SOURCE REFERENCES 314

AIDS is a disease caused by the human immunodeficiency virus (HIV) (see Section 14.10, Source references). The words AIDS and SIDA are acronyms of the English term 'acquired immunodeficiency syndrome' and the French term 'syndrome d'immunodéficience acquise', respectively. The term syndrome refers to the group of symptoms which accompany a certain disease; immunodeficiency refers to the inability of the body's defence system to function; acquired means that the condition is not inherited, but the result of an infection.

HIV leads to the destruction of the immune system of the body. It thus becomes unable to resist many infections which the healthy body would cope with in normal circumstances. The body also becomes vulnerable to attack from certain malignant tumours.

AIDS is the subject of justifiable concern and fear throughout the world and, although the Eastern Mediter-anean Region has a relatively low incidence, there is an enormous potential threat. Recent graduates and doctors working at primary health-care level will have received varying amounts of information on the subject and may feel inadequately informed. This chapter is intended to fill the information gap. Although little may be done in the way of treatment at the primary level, it is here that suspicion and referral are most likely to occur. It is also at this level that most can be achieved in the way of prevention and community care.

14.1 MODES OF TRANSMISSION

HIV has been isolated from blood, serum and various body fluids including semen, cervicovaginal secretions, breast milk, tears and saliva. The isolation of the virus from a body fluid does not necessarily mean that the fluid is important in transmission. Detailed epidemiological studies throughout the world have implicated mainly blood, semen and cervicovaginal secretions, and indicate that there are three basic modes of transmission: sexual intercourse; contaminated blood or blood products, or contaminated skin-piercing instruments; and mother to infant.

The relative importance of the various modes of transmission differs between continents, between countries and from one group to another. Transmission is also related to the infective dose (virus titre), the intensity of exposure and the infection phase of the transmitter.

There is no evidence to suggest that the virus can be transmitted by the respiratory or enteric routes, or by casual person-to-person contact, including that which may occur in the domestic, social, work, school, camp or prison environments. Nor is there any evidence to suggest that transmission involves insects, food, water, toilets, swimming pools, seats, tears, shared eating and drinking utensils, or other items such as second-hand clothing. The issue of transmission by saliva is of concern to the public, for example through the use of articles, such as toothbrushes, belonging to infected persons, or in the course of dental work. It is reassuring to note that the rate of isolation of the virus from the saliva of infected

persons is very low (1–2%).

HIV is not spread casually but as a result of human action, which is clearly subject to control. Transmission can be prevented through screening of blood before transfusion, avoiding the reuse or sharing of contaminated needles and syringes and, above all, by educating the public as to the necessity of safe sexual behaviour.

14.1.1 SEXUAL INTERCOURSE

HIV infection should be regarded primarily as a sexually transmitted disease. Sexual intercourse, both heterosexual and homosexual, is the main mode of transmission, being responsible for some 90% of cases. At present, heterosexual transmission exceeds homosexual transmission (60% heterosexual and 40% homosexual). Certain sexual factors increase the risk of infection, e.g. number of sex partners. The presence of an ulcerative sexually transmitted disease also increases the risk of infection.

14.1.2 CONTAMINATED BLOOD AND BLOOD PRODUCTS OR CONTAMINATED SKIN-PIERCING INSTRUMENTS

Parenteral transmission may occur through transfusion of infected blood or certain blood products, and the use of contaminated needles and syringes and other skin-piercing instruments. Cases of AIDS acquired through transfusion of blood and blood products represent a small but important proportion of the total number of cases (around 5%); they are largely preventable. In studies of single parenteral exposures, the risk of acquiring HIV infection is related to the inoculum. Recipients of a single unit of blood from an infected person have a high risk of acquiring infection (nearly 90%). To date, whole blood, blood cellular components, plasma and clotting factors have transmitted HIV infection. No other products prepared from blood (immunoglobulins, albumin and plasma protein fractions) have been implicated.

Contaminated needles and syringes have been responsible for transmission in hospital settings, in particular in eastern Europe. Patients requiring treatment of their blood, e.g. those undergoing kidney dialysis, are at high risk if the process used is not under strict supervision.

There is a close relation between the problem of drug addiction and the spread of AIDS, both directly, through the use of contaminated syringes and needles in the case of intravenous drug use, and indirectly, through its relation to prostitution. Drug abuse presents a significant problem in this context, particularly as in many

countries it is not approached as a health issue but is largely handled as a crime, and dealt with by the police alone.

In some countries with high levels of infection there is an apparent association between AIDS and medical injections and, in Africa and Haiti, injections for ritual purposes. This is largely owing to the fact that disposable equipment is not generally available and sterilization procedures are not adequate.

14.1.3 MOTHER TO INFANT

Perinatal transmission may occur before, during or shortly after birth. The overall risk of HIV transmission from an HIV-infected mother to her infant ranges from 20% to 45%. Transmission is more efficient from mothers who have immune suppression, and from those recently infected. Studies of twins indicate that the first-born baby, even if delivered by caesarian section, is three times more likely to be infected than the second baby, indicating local infection through the cervix. Postnatal transmission has been described in infants of mothers who acquired HIV infection after delivery; breast milk has been suggested as the possible means of transmission in these cases. There are difficulties in interpreting a positive serological test in a neonate, as the antibodies present may have been passed on by the infected mother (passively acquired antibodies remain for up to 18 months after birth). It is now possible to diagnose infection among neonates, via the use of the PCR (polymerase chain reaction) test, which detects antibodies not inherited from the mother (e.g Igm and Iga antibodies).

14.2 AETIOLOGY

The causative agent of AIDS is a newly recognized retrovirus. It has been given different names, but is now known by the internationally agreed name of human immunodeficiency virus (HIV). The most common type is HIV-1. Cases of AIDS due to another type (HIV-2) have been reported from West Africa, where it is believed to be endemic. HIV-2 is practically the same as HIV-1 as regards the epidemiological picture, and is also believed to be so clinically. It seems, however, that HIV-2 is less pathogenic, or it may just take longer to cause immunodeficiency.

HIV penetrates different types of cells in the human body. It goes through several steps within the cell, one of which is the integration of the viral DNA into the host cell's own genetic material; each time the host cell reproduces, the new cells contain

viral genes. This results in an infection which, most virologists believe, is lifelong. In addition, infected persons are likely to be infectious for life.

The immune system of the AIDS patient is damaged largely because of the destruction by the virus of the helper T lymphocytes. These cells regulate all the immune functions of the human body, and also the activation of B lymphocytes responsible for antibody production, other T lymphocytes which are responsible for killing viruses and tumour cells, and the monocytes that are responsible for killing many parasites and bacteria. Although the virus does not infect many T4 cells at one time, it kills those infected once they are immunologically activated. The virus essentially converts the T cell from a lymphocyte to an AIDS virus factory. The damaged T cells produce the virus, which subsequently invades other T cells. The mechanism of immune response to infection suffers considerably, and thus the AIDS victim is left vulnerable to an array of life-threatening infections and malignancies.

14.3 INCUBATION PERIOD

The incubation period is not precisely known. The time scale from infection to the appearance of definite signs and symptoms of the disease seems to range from 6 months to several years. The mean incubation period is approximately 1 year in children and more than 5 years in adults. It seems to be related to the infective dose, being shorter in cases infected through blood transfusion, and to age, being shortest (average 8 months) in babies infected during pregnancy.

Within 5 years of infection approximately 25% of infected persons develop AIDS, and a similar percentage develop the pre-AIDS condition known as AIDS-related complex (ARC). The risk of the remaining percentage of infected persons contracting the disease increases as the time since infection increases. Little is known as to why some infected persons develop symptoms while others do not. It is believed that certain factors may trigger the progression of infection to AIDS. These include repeated exposure to infection, infection with diseases such as tuberculosis or malaria, which compromise the immune functions, pregnancy and, possibly, genetic factors.

14.4 CLINICAL MANIFESTATIONS

The clinical expression of infection with HIV appears increasingly complex. It includes manifestations due to opportunistic diseases, as well as illness directly caused by HIV itself. The types of

opportunistic infection and neoplasm vary not only in populations of different geographical origin, but also according to the probable route by which the infection is contracted.

HIV infection may be subdivided into stages, not all of which are necessarily present or consecutive in all patients. These stages include the acute phase; the latency period; persistent generalized lymphadenopathy (PGL); AIDS-related complex (ARC); and AIDS.

14.4.1 ACUTE PHASE

Approximately 2 weeks after infection some infected persons may have non-specific general manifestations such as fever, malaise, depression, lymphadenopathy (cervical, axillary and inguinal), muscular pains, fatigue, headache (especially retro-orbital), maculopapular rash with truncal distribution, sore throat and cough. These manifestations disappear after 1–2 weeks and the general condition then returns to normal. Laboratory examination for antibodies is usually negative at this stage, as seroconversion generally occurs 6–12 weeks after infection. Seroconversion may be associated with cutaneous manifestations and sometimes with severe illness, particularly acute encephalopathy.

The recognition of this acute phase, although difficult, is very important as there is evidence that the person is highly infectious during this stage, and capable of transmitting the virus efficiently.

14.4.2 LATENCY PERIOD

Following the acute phase there is usually a period of latency which lasts from several months to several years. During this time the virus multiplies and infects more cells, but the count of CD4 cells remains beyond the critical level of immunodeficiency.

14.4.3 PERSISTENT GENERALIZED LYMPHADENOPATHY

Following the latency period, symptoms may appear in the form of persistent generalized lymphadenopathy. Enlarged lymph nodes (greater than 1 cm in diameter) involving two or more extrainguinal sites and persisting for at least 3 months should be suspected as being due to HIV infection in the absence of any other illness or drug use known to cause lymphadenopathy. About one-third of patients with generalized lymphadenopathy are asymptomatic. Lymphadenopathy may slowly regress in size during the course of this period.

14.4.4 AIDS-RELATED COMPLEX (ARC)

The condition may progress to a complex including one or more of the following manifestations: weight loss (more than 10%), malaise, fatigue, lethargy, anorexia, abdominal discomfort, diarrhoea, fever, night sweats, headache, itching, amenorrhoea and splenomegaly. These symptoms and signs are frequently intermittent. Weight loss is found in all patients and is generally progressive. Thrombocytopoenia develops in approximately 10% of patients, but this is often transient. Although these signs and symptoms are similar to those occurring in AIDS patients, the symptoms and immunological abnormalities are less severe. In contrast to AIDS cases, neither opportunistic infections nor malignancies have been diagnosed in such patients.

Many ARC patients present with mucocutaneous lesions. These are important for the early diagnosis of AIDS or ARC. They include mainly varicella, zoster, seborrhoeic dermatitis, recurrent and persistent orolabial and genital herpes, *molluscum contagiosum*, oral candidiasis and oral hairy leukoplakia. A generalized papular pruritic eruption (prurigo) may be found in approximately 20% of HIV-infected persons, and frequently occurs at an early stage of the disease. Persistent diarrhoea is one of the major complaints in patients with ARC or AIDS. In most patients no specific cause for the diarrhoea is found.

14.4.5 AIDS

AIDS is the most severe stage of the clinical spectrum of HIV infection. The same signs and symptoms as described for ARC patients occur in patients with AIDS, but the manifestations become more pronounced. In addition, the clinical picture is characterized by the presence of opportunistic infections and tumours as a result of profound cellular immunodeficiency. The types of opportunistic infection depend largely on the past and actual exposure of the host to microbial agents. This may explain the differences in frequency of certain opportunistic infections between different countries. *Pneumocystis carinii* pneumonia is by far the most common opportunistic infection in Americans and Europeans, but is less frequently found in African patients. In contrast, the gastrointestinal system is a major site of infection in Africans with HIV disease, possibly because of a high exposure to enteric microbial agents.

14.5 CLINICAL CASE DEFINITION

There are cardinal findings, characteristic findings and associated findings among symptomatic cases of HIV infection.

CARDINAL FINDINGS

- Generalized, rapidly progressive or invasive Kaposi's sarcoma
- Oesophogeal candidiasis
- *Pneumocystis carinii* pneumonia
- *Toxoplasma* encephalitis

CHARACTERISTIC FINDINGS

- Oral thrush (in persons not taking antibiotics)
- Hairy leukoplakia
- Miliary and extrapulmonary tuberculosis
- Recurrent herpes zoster
- Chronic progressive and disseminated herpes simplex infection
- Severe prurigo
- Kaposi's sarcoma other than cardinal findings

ASSOCIATED FINDINGS

- Weight loss (more than 10%) or abnormally slow growth in children
- Chronic diarrhoea for more than 1 month
- Prolonged or intermittent fever for more than 1 month
- Cough for more than 1 month
- Generalized lymph node enlargement (extrainguinal)
- Skin infections (severe and recurrent)

14.6 PREVENTION AND CONTROL

In the absence of effective treatment or a vaccine, the use of other methods of prevention becomes a necessity. There is sufficient information on the modes of transmission to permit national authorities to initiate prevention and risk-reduction measures to limit the danger of infection. However, before detailing these measures, it is essential to emphasize that the problem is a multidisciplinary, multisectoral and multidimensional one, involving a complex range of economic, social, cultural, educational

and behavioural as well as epidemiological factors. The strategies for prevention and control are outlined below.

14.6.1 PREVENTION OF SEXUAL TRANSMISSION

The AIDS pandemic has resulted largely from the sexual spread of HIV. This mode of transmission is not easy to control because of the complex social and religious factors which affect open discussion of sexual matters. However, prevention will be essentially dependent on education aimed at changing high-risk behaviour and hence reducing the risk of exposure.

At the national level, proper epidemiological assessment of the problem will facilitate the identification of risk factors related to sexual transmission and help define behavioural issues that need to be addressed. Adaptation of communication technology to the local situation and needs is the essential phase in developing a successful public health education approach.

In the Eastern Mediterranean Region, and in developing countries in other regions, efforts should be made to take advantage of the strong religious beliefs that prevail in order to promote healthy lifestyles and abstinence from unhealthy ones.

14.6.2 PREVENTION OF BLOOD-BORNE TRANSMISSION

To date, less than 5% of cases of AIDS detected in western Europe and North America have been caused by blood transfusion. In the Eastern Mediterranean Region blood and blood products have been of relatively higher importance in the transmission of AIDS, although this risk is decreasing. Nevertheless, attention should be given to this potential mode of transmission, as follows:

- Blood transfusion should be avoided if not urgently needed. Transfusions should not be given for minor reasons.
- Blood and blood donors should be screened. The general rule should be to screen all blood units and blood donors for the presence of HIV antibodies. There is no evidence that screening for the HIV antigen increases the safety of blood transfusion beyond that obtained by antibody screening.

  Whether blood can be screened or not, ways to exclude potential donors with AIDS risk factors have to be considered, for example through the following:

  Voluntary self-exclusion systems, in which persons with risk factors for AIDS refrain from giving blood. This should be achieved through the education of donors.

Obtaining the history of possible exposure to a known risk, as well as enquiring about symptoms such as severe chronic diarrhoea, night sweats, fever and weight loss. This is easy when blood donation is voluntary, but where donors are paid it may not be an easy task, as such donors are inclined to hide symptoms.

Physical examination of the donor. Although not feasible in all blood collection settings this should be encouraged, because it can identify unusual mucosal or skin lesions, lymphadenopathy and wasting, depending on the stage of the disease.

- Blood products must be produced in a manner which eliminates the risk of HIV transmission. This is only practical for products free from cellular components.

14.6.3 PREVENTION OF TRANSMISSION THROUGH INJECTIONS AND SKIN-PIERCING PROCEDURES

HIV transmission can occur through injections and the use of contaminated skin-piercing instruments. Efforts must be made to ensure that needles and other such instruments, e.g. lancets used for taking blood samples for laboratory examination and needles used in ear-piercing and tattooing, are safe and uncontaminated.

As intravenous drug abuse constitutes one of the highest-risk activities for transmission of HIV infection, efforts should be made to reduce this risk. The sociocultural context of drug abuse is of critical importance in any effort either to understand or to change the risk behaviour associated with transmission of HIV. The provision of sterile injection equipment is unlikely to be sufficient to bring about a cultural change in the behaviour of intravenous drug users; changing the sociocultural context and the societal response to drug abuse is probably the most effective way of ensuring the ultimate aim of prevention of HIV transmission. It must be emphasized that any successful HIV prevention programme for this group should consider treatment of drug abuse.

Some cases of HIV infection have been traced to organ transplants. The necessity of screening donors before accepting organs for transplant cannot be emphasized enough.

14.6.4 PREVENTION OF PERINATAL TRANSMISSION

Women of childbearing age are usually infected heterosexually. Pregnancy may accelerate the development of clinical illness.

Prevention and control of this mode of transmission must take into consideration sensitive issues such as contraception. Infected women should be advised against pregnancy, both for their own health and to avoid transmitting infection to the baby. Prevention may also require repeated campaigns to convince people who are considering becoming parents to undertake voluntary testing and counselling before marriage.

14.6.5 PUBLIC HEALTH EDUCATION

In addition to the specific measures mentioned above, general public health education is needed. The public should be informed of the nature of the disease, its main modes of transmission and particularly the dangers of promiscuous sexual relations. They should be informed that there is no evidence that the disease is spread through casual social contact, by food or by the airborne route. Health authorities must alleviate any unjustified or exaggerated fears that may have been aroused.

Those in contact with HIV patients in the home should be advised to observe good standards of hygiene and cleanliness and to avoid contact that may involve exchange of any body fluids, particularly blood.

Educational messages should be directed towards high-risk groups who are more prone to infection because of their lifestyles and behaviour, particularly those who expose themselves to sexually transmitted diseases. Some of these groups, such as intravenous drug users, are generally difficult to reach and to educate. Nevertheless, every effort should be made to educate them about the danger of sharing needles and of promiscuous sexual relations.

It is satisfying to note that studies in some countries with high infection rates have shown that people in high-risk categories are prepared to change their lifestyles and behaviour voluntarily in order to avoid the more obvious risks of infection by HIV.

14.6.6 GENERAL REMARKS

HIV-infected people and AIDS patients should be counselled in order to minimize the risk of infecting others. They should be informed about the nature of the disease and its modes of transmission. They should be briefed on how to prevent the spread of infection to others. They should refrain from donating blood. Although infection by saliva is unlikely, infected persons should be advised against intimate kissing and orogenital contact. Tooth-

brushes, razors and other articles that could become contaminated with blood should not be shared. In the event of an accident causing bleeding, the contaminated surfaces and articles should be cleaned and then disinfected using a 200 ppm sodium hypochlorite solution (1:10 dilution with water of 5% sodium hypochlorite – common household bleach).

14.7 SAFETY PRECAUTIONS FOR MEDICAL PRACTITIONERS

Medical practitioners, as leaders in health care work, set an example to their colleagues in the health services. They are responsible for protecting themselves and others from HIV infection and they must ensure that medical service settings do not expose patients to infection. This can be achieved through strict adherence to sound preventive principles. Since personal history and clinical examination cannot detect all HIV-infected persons, it is imperative to apply the proper precautions constantly in handling blood and other body fluids. These measures are well known and have long been applied. However, their application has sometimes been lax. The emergence of AIDS has given these measures increased importance. Therefore, medical practitioners should always observe the procedures outlined below:

1. Precautionary measures should be taken to prevent the exposure of the skin and mucous membranes to blood and other body fluids. This can be done by wearing protective gloves which should be changed for every patient.
2. Although HIV has been isolated from saliva, the spread of infection by saliva is unlikely. Nevertheless, in order to minimize occupational exposure to infection during mouth-to-mouth resuscitation, mouth pieces, resuscitation bags or any other available means must be used. Resuscitation equipment should be used once and then either disposed of or well sterilized before re-use. Mucus suction by mouth must be replaced by electric suction or by machines worked manually or by foot.
3. Disposable syringes and needles should be used for injections or taking samples of blood and other body fluids from patients. If disposables are not available, syringes and needles should be thoroughly sterilized.
4. If, during any medical procedure, there is a possibility of spurts, spray or spillage of blood or other body fluids, protective clothes, surgical masks and protective goggles should be worn.

5. The hands and other exposed parts of the skin that become soiled with blood or body fluids should be well washed with soap and water immediately. They should also be washed after taking off protective gloves.
6. Care must be taken against accidental injury with needles, scalpels or other sharp instruments. Do not recap, break or bend used needles, or remove them from the disposable syringes. After use, such instruments must be put in special impermeable containers, situated as near as possible to the working area; the instruments must then be treated as contaminated.
7. Injections and skin-piercing procedures should be limited to cases where there are clear indications for such procedures, which must then be performed correctly. For example, in many cases, drugs are given parenterally when they would be equally effective if given orally. Avoiding or reducing the number of unnecessary injections is important for the protection of both the medical practitioner and the patient.
8. Accidental wounds or pricks should be left to bleed before being thoroughly washed with soap and water.
9. Used towels and linen should be put in a bag immediately after use. They should never be sorted or washed where patients are cared for. Any linen or towels contaminated with blood or other body fluids must be collected and transported in special impermeable bags. If such bags are not available, the items should be folded with the soiled parts turned inwards. Protective gloves and clothing should be worn when handling soiled items.
10. Used towels and linen should be washed with a disinfectant and water at 71°C for at least 25 minutes. If the washing has to be done at a lower temperature, suitable chemicals should be used in concentrations indicated by the manufacturer.
11. In case of accidental spillage of, or contamination with, blood or other body fluids, the soiled area should be flooded with a suitable disinfectant (preferably sodium hypochlorite with 0.1–0.5% available chlorine). The mixture of disinfectant and blood or body fluid should then be washed away and the area again wiped with disinfectant.
12. Any physician suffering from a wound or oozing skin disease should refrain from giving direct medical care to patients, or from handling the patient's articles of treatment. If necessary, broken skin or open wounds must be covered with watertight dressings. Wash hands with soap and water immediately after any contact with blood or other body fluids.

13. If doctors suspect that they have been exposed to infection, they must report without delay to the responsible health authority for counselling and follow-up.

14.8 ANTIBODY TESTING

HIV infection is often diagnosed by means of antibody tests. Detectable antibody usually develops within 3 months after infection. A confirmed positive antibody test means that a person is infected with HIV and is capable of transmitting the virus to others. Although a negative antibody test usually means that a person is not infected, infection from a recent exposure cannot be ruled out. If antibody testing is related to a specific exposure, the test should be repeated after 3 and 6 months.

Antibody testing for HIV begins with a screening test, usually an enzyme-linked immunosorbent assay (ELISA). If the screening test is positive, it is followed by a more specific confirmatory test, most commonly the Western blot. New antibody tests that are easier to perform or more accurate are being developed and licensed. Positive results from screening tests must in principle be confirmed before being considered definitive.

14.9 MANAGEMENT PLAN

There are sophisticated management plans to be applied in health care centres where advanced levels of diagnostic skills are available. However, Figs. 14.1 and 14.2 represent plans that are more suitable for application at the primary health care level, both when HIV tests are available and when they are not, respectively.

Notes for Figures 14.1 and 14.2

[A] Either an Elisa or a rapid test.
[B] The presence of three or more indicator symptoms or signs, as listed below, each of a **different** category (e.g., one skin, one gastrointestinal, one general), makes the presence of symptomatic HIV infection highly likely.

SKIN

- Severe prurigo
- Genital or anal ulcer(s) of more than 1 month's duration
- Seborrhoeic dermatitis
- Herpes zoster (shingles)
- Severe or recurrent skin infections
- Bluish skin tumours

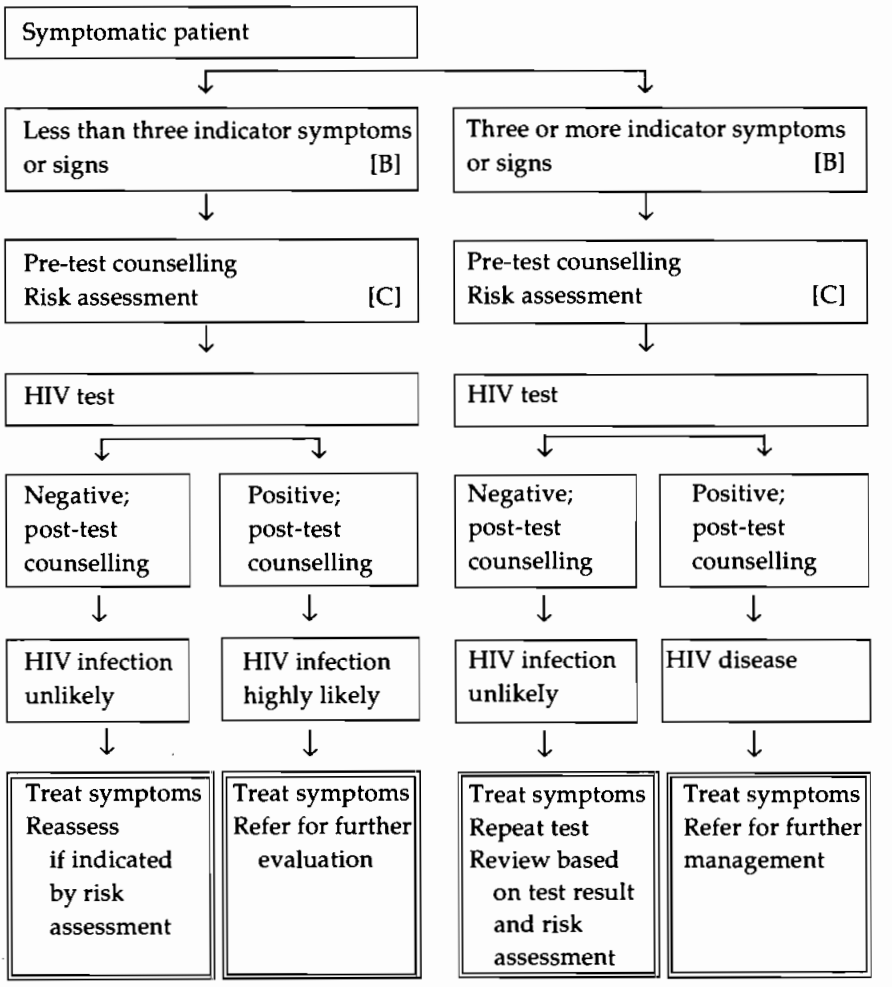


Fig. 14.1 Simplified flowchart for detection and management of HIV infection (HIV test available [A]). [A] = Either an Elisa or a rapid test; [B] = indicator symptoms or signs; [C] = risk assessment (for further details see Notes to Figures 14.1 and 14.2 in the text).

GASTROINTESTINAL

- Oral thrush (white plaques in mouth)
- Difficult or painful swallowing
- Diarrhoea of more than 1 month's duration

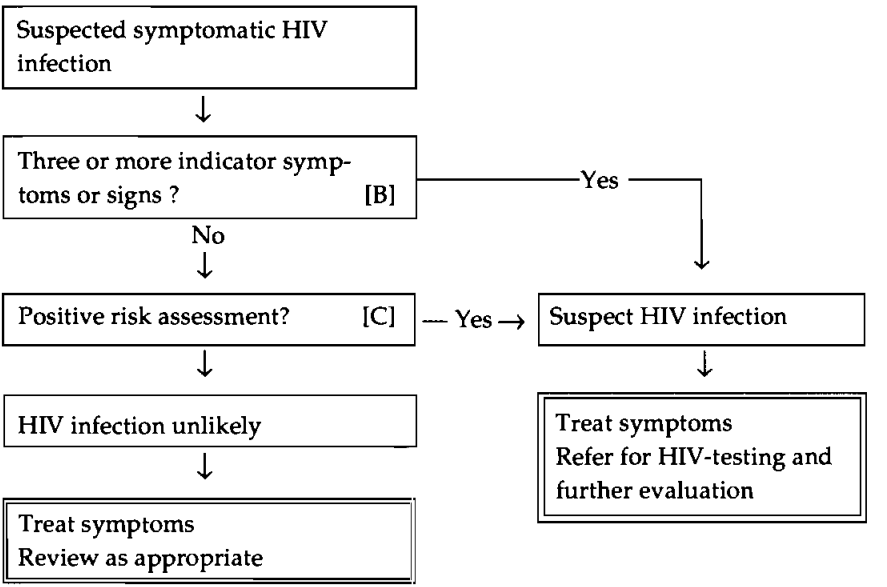


Fig. 14.2 Simplified flowchart for detection and management of HIV infection (HIV test not available). [A] = Either an Elisa or a rapid test; [B] = indicator symptoms or signs; [C] = risk assessment (for further details see Notes to Figures 14.1 and 14.2 in the text).

NEUROLOGICAL

- Headache (progressively worsening)
- Seizures ('fits')
- Stiff neck
- Loss of sensation or movement
- Mental deterioration

RESPIRATORY

- Cough of more than 1 month's duration
- Worsening shortness of breath

GENERAL

- Fever of more than 1 month's duration
- Weight loss (unexplained) of more than 10% of baseline body weight
- Enlarged glands in three or more sites, including the groins
- Chronic malaise or fatigue

[C] The presence of one or more of the following risk factors should alert the clinician to the possibility of HIV infection, the risk increasing with the number of risk factors:

- Sexual intercourse with a person with AIDS, HIV infection or known epidemiological risk factor or from an HIV-endemic region;
- Anal sex between men;
- Multiple sex partners (e.g. more than three sex partners within the last 6 months);
- Recent history of genital ulcer;
- History of transfusion of blood or blood products in the last 10 years;
- Intravenous drug use;
- History of infections, tattooing, or undergoing medical or surgical procedures with non-sterile instruments (including needle pricks in health personnel).

14.10 SOURCE REFERENCES

The following references have been used in the preparation of this chapter; readers are referred to these for further information.

Management of patients with sexually transmitted diseases, Report of a World Health Organization Study Group, World Health Organization Technical Report Series No. 810, World Health Organization, Geneva, 1991.

Epidemiology of acquired immunodeficiency syndrome (AIDS), Wahdan, M.H., WHO Regional Office for the Eastern Mediterranean, Alexandria, Third edition, 1991 (unpublished document).

A message on AIDS for medical practitioners, AIDS Information Exchange Centre, WHO Regional Office for the Eastern Mediterranean, Alexandria, 1992 (unpublished document .

Index

Abbreviations, list of xii
Abdominal injuries 152–3
  emergency management 156–7
  preoperative procedures for exploration 156–7
  signs and symptoms 153
  specific injuries 157–9
  tests 155
    laboratory 155
    radiological 154
Abdominal pain
  acute 142
    epigastric, diagnostic features 146
    history and diagnosis 143–7
    location and origin 144
    physical examination 147–8
    tests 148–9
  associated with fever 221
Abdominal thrusts, *see* Heimlich technique
Abdominal wall, injuries to 157
Abortion 281–2, 284–6
Abrasions 43
  corneal 57
Abscesses
  liver 221
  palmar 215
  pulp 215
  subcutaneous, on hand 215
Acid
  burns 69
  poisoning 270
Acquired immunodeficiency syndrome (AIDS) 298
  aetiology 301–2
  antibody testing 311
  associated with fever 221
  clinical case definition 305
  clinical manifestations 302–3
    acute phase 303
    AIDS-related complex (ARC) 304
    latency period 303
    lymphadenopathy 303
  detection algorithm 312, 313
  and drug abuse 310, 307
  incubation period 302
  management plan 311–14
  prevention and control 305–6
    blood-borne 306
    counselling 308–9
    injections and skin-piercing instruments 307
    perinatal 307–8
    public health education 308
    sexual transmission 306
  safety precautions for medical practitioners 309–10
  source references 314
  transmission, modes of 299
    contaminated blood or instruments 300–1
    perinatal 301
    sexual intercourse 300
Activated charcoal, for drug overdose 263
Adrenal insufficiciency 195
Adrenocorticosteroids, for status asthmaticus 87–8

AIDS, *see* Acquired immunodeficiency syndrome
AIDS-related complex (ARC) 304
Airway
  bag and mask ventilation 2
  cricothyroidotomy 10–12
  endothracheal intubation 5–10, 129–30
    in children 245–6
  nasal airway insertion 4–5
  nasopharyngeal airway 129
  nasotracheal intubation 8–10
  needle insertion into the cricothyroid membrane 9–10
  obstruction, *see* Airway obstruction
  oral airway insertion 3
  oropharyngeal airway 129
  orotracheal intubation 5–8
  rupture of 98
Airway obstruction 81
  complete, *see* Suffocation
  by foreign bodies, in children 236–7
  infectious 74–6
  lower 130–2
  upper 128–30
Alcohol withdrawal 181–3
Alkalis, burns 70
Aminophylline, for status asthmaticus 87
Amphetamine overdose 268–9
Amputation injuries, of fingers or hand 213
Anaphylaxis 124–6
Angiography, after abdominal injuries 155
Ankle, splinting 204–5
Anoxia, after head injuries 187
Anthrax 221
Anxiety 194
Aorta, rupture of 101
Arm, splinting 202–3
Arterial blood gases
  in respiratory failure 76–7, 82
  in shock 118
Arteries
  brachial, control of bleeding 62
  carotid, control of bleeding 62
  femoral, control of bleeding 62–3
  haemorrhage 59, 61–3
Arteriography, renal, and kidney injuries 161
Arthritis, bacterial, clinical features 219
Artificial respiration 137
Aspiration and drainage techniques
  chest tube insertion 28–9
  Ewald tube insertion 33
  gastric lavage 32–3
  nasogastric tube insertion 31–2
  pericardiocentesis 30–1
  Sengstaken-Blakemore tube insertion 33–5
  thoracentesis 26–8
  urethral catheterization 35–6
Asthma
  acute attack, in children 237–8
  see also Status asthmaticus
Atropine, for ingested poisons 273

Bag and mask ventilation 2
Barium studies, in abdominal injuries 155
β-Adrenergic agents, in shock 120–1
Biliary tract, injuries to 158
Birth, *see* Labour
Bites
  animals 276
  cats 277
  dogs 276–7
  to hand 213
  humans 275
  snakes 277
Blackwater fever 226
  and renal failure 228
Bladder
  injuries to 162
  rupture of 161
Blast injury, to lungs 102
Bleeding, *see* Haemorrhage
Blood, volume expansion in shock 119–20
Blood gases, arterial, *see* Arterial blood gases
Blood pressure, in shock 115–16, 117
Blood vessels, in neck injuries 52
Brain injury 186–7
  patient evaluation 38
Breathing, *see* Respiration

Breech presentation 292–4
Bronchial emphysema, congenital, in newborn 254
Bronchiolitis, in children 239
Bronchitis, acute-on-chronic 79–84
Bronchoscopy 81, 131
Brucellosis 222
Bruises, *see* Contusions
Bullet wounds, *see* War wounds
Burns
  acid 659
  alkali 70
  chemical 69–71
  classification 64–5
  definition and causes 64
  electrical 71–2
  eyes 57–8
  first aid 65
  hand 213
  'rule of nine' 65
  sunburn 72–3
  thermal
    history 66
    management 66–8
    minor 66–7
    moderate and severe 65–6, 67
    physical examination 66

Capillaries, haemorrhage 59
Carbon dioxide narcosis 82–3
Carbon monoxide poisoning 274–5
Cardiac arrest, CPR procedures 134–41
  in children 241–3
Cardiac massage
  alternative methods in failure of 141
  closed chest compression 138, 140
    in children 242
  open chest 140–1
Cardiac tamponade 100, 108
Cardiocirculatory arrest, causes 127
Cardiogenic shock
  causes 114
  emergency treatment 107–8
  signs and symptoms 107
  therapy 123–4
Cardiopulmonary resuscitation (CPR) 127
  basic principles 135, 136
  cardiac arrest 134–41
  in children 241–3
  complete airway obstruction (suffocation) 132–4
  lower airway obstruction 130–2
  upper airway obstruction 128–30
Cardiothoracic injuries 90–102
Cardiovascular injuries
  cardiac tamponade 100
  contusion of the heart 100
  intracardiac rupture 100
  lung blast injury 102
  physiology 99
  rupture of the aorta 101
Cathartics, for drug overdose 263
Catheterization
  urethral 35–6
    in urological injuries 160–1
  venous
    internal jugular 20–3
    peripheral central line placement 14–17
    peripheral percutaneous 11
    subclavian 19–20
    venesection (cutdown) 16–18
Central nervous system (CNS), emergencies
  coma, *see* Coma
  status epilepticus 164
  suncope 174
  transient ischaemic attacks (TIAs) 174–7
Central venous pressure (CVP), in shock 116, 117
Cerebral compression 186–7
Cerebral irritation 186
Cerebral malaria 226, 228
Cervicofacial injuries
  eyes, *see* Eyes
  face 52–5
  neck 50–2
  throat 55–6
Chemicals
  burns 69–71
    to eyes 58
  poisoning, antidotes 266
Chest
  flail 91–2
  missile and bullet injuries 48–9
  tube insertion 28–9
Chest compressions 138, 140–1
  in children 242

Chest pain 103
  acute myocardial infarction 106–7
  cardiac tamponade 108
  cardiogenic shock, *see* Cardiogenic shock
  causes 103
  characteristics 104
  diagnosis 104–5
  hypertensive crisis 111–12
  pulmonary embolism 109–11
  right heart failure 107
Children
  examination of 235–6
  surgical emergencies 252–8
  see also under specific conditions and therapies
Chlordiazepoxide, in alcohol withdrawal 182–3
Chlormethiazole, in alcohol withdrawal 182
Cholecystitis, acute, diagnostic features 146
Circulation, emergency procedures 39
Circumcision, female 161
CNS, *see* Central nervous system
Colloids, in shock 119
Colon, injuries to 158
Coma 165
  aetiology 172–4
  assessment 166–72
  diagnostic investigations 171
  with focal neurological deficits and trauma 172–3
  with focal neurological deficits without trauma 173
  without focal neurological deficits 174
  history 166–7
  hyperglycaemic hyperosmolar non-ketotic (HHNK) 192–3
  initial therapy 167–8
  intracranial tension, treatment of 171–2
  with meningeal irritation 174
  pathophysiology 165–6
  physical examination 167–71
Compression, cerebral 186–7
Computed tomography (CT), in abdominal injuries 154
Concussion 186
Confusion
  after alcohol withdrawal 181–3
  after head injuries 186
Congestive heart failure 107
Conjunctiva, foreign bodies 56
Consciousness, and patient evaluation 40
Contusions 41
  eyeball 58
  of heart 100
  multiple bruises 41
Convulsions, during pregnancy 288–9
Cornea
  abrasions 57
  foreign bodies 56–7
  reflexes, in coma 169–70
Coronary perfusion pressure (CPP) 120, 122
CPR, *see* Cardiopulmonary resuscitation
Cricothyroid membrane, needle insertion into 9
Cricothyroidotomy 10–12
Cricothyroid stab, for upper airway obstruction 129
Croup, in children 239–40
Crystalloids, in shock 120
CT scanning, in abdominal injuries 154
Cutdown 16–17
Cyanide poisoning 270–1
Cystography, in ruptured bladder 161

Defibrillation 23–4
Dehydration 195
  in children 250–1
  and diarrhoeal diseases 250
Delirium tremens 181–2
Dextran, in shock 119
Diabetic ketoacidosis 189–90
Dialysis, in drug overdose 264, 265
Diaphragmatic hernia, congenital, in newborn 255
Diarrhoea
  acute infective 221
  after antibiotics 223
  bloody, associated with fever 220
  and dehydration, in children 250

Dislocations 206–7
  patient evaluation 38
Distension, and abdominal pain 147
Diuresis, forced, for drug overdose 263–4
Drainage systems, underwater seal 29, 98
Drainage techniques, *see* Aspiration and drainage techniques
Drug abuse, and AIDS 300, 307
Drug overdose 260
  amphetamine 268–9
  antidotes 264, 266
  counselling 265
  elimination of absorbed drugs 263–4, 265
  identification of drug 260
  intensive supportive care 260
  management principles 260
  narcotics 267–8
  pharmacology and toxicity 260–1
  prevention of drug absorption 262
  salicylate intoxication 269
  sedative intoxication 267
  see also Poisoning
Dullness, and abdominal pain 148
Duodenum, injuries to 158
Dystonic reactions to antipsychotic drugs 180

Earache, associated with fever 231
Ecchymoses, *see* Contusions
Eclampsia 288–9
Ectopic pregnancy 282, 284–6
Elbow, splinting 202–3
Electrical burns 71–2
Embolism, pulmonary 109–11
  post partum 297
Emergencies
  life-saving procedures 39–40
  management 38, 40–1
  patient evaluation 38–9
Emesis
  forced, for drug overdose 262
  hyperemesis gravidarum 280–1
Emphysema, bronchial, congenital 254
Encephalopathy, hypertensive 111–12
Endotracheal intubation 5–10, 129–30
  in children 245–6
Epigastric pain, diagnostic features 146
Epiglottitis
  acute, clinical features 220
  acute infectious 75–6
  in children 240
Epistaxis 63
  anterior 63
  posterior 63
  uncontrolled 63
Ewald tube 33
Excitement 178–80
Eyes
  burns 57–8
  changes, in hypertensive crisis 111
  conjunctiva, foreign bodies 56
  cornea, *see* Cornea
  eyeball 58
  foreign bodies 56–7
  funduscopic examination, in coma 169
  injuries to 56–9
  movement, spontaneous and evoked, in coma 170
  pupillary reflexes, in coma 169

Face
  fractures 54–5
  injuries 52–3
    emergency management 55
    facial skeleton 54–5
    soft tissue 53–4
    types of 53
  lacerations 45, 53
Fever
  in children 246–8
  isolation procedures 231–4
  management 218–19
  pathophysiology 217–18
  see also specific illnesses
Fingertip injuries 45, 212
Fishhook removal 216
Flail chest 91–2
Focal epilepsy, vs transient ischaemic attacks 176
Food poisoning 149–50
  infective 150
  toxic 150–1

Foot, splinting 204–5
Forearm, splinting 203
Foreign bodies
  in airway, children 236–7
  in eyes 56–7
Fractures
  closed 197
  complicated 198, 199
  definition and causes 196–7
  diagnosis 199–200
  emergency management 200
  first aid 200–1
  hand 213
  mandible 55
  maxillae 55
  midface 55
  open 198, 205
  patient evaluation 38
  patterns of 197
  pelvis, in female 161
  ribs 90–1
  skull 185
  types 197–9
  vertebral bodies 207–8
  see also Splints

Gag reflex, in coma 169–70
Gallbladder, injuries to 158
Gastric lavage 32–3
  for drug overdose 262
Gastroenteritis, in children 250–1
Gastrointestinal obstruction, congenital 256
Genital injuries, external, treatment 161
Genitourinary tract injuries, *see* Urological injuries
Guillain-Barré syndrome 77

Haemarthrosis 206
Haematocrit measurement
  in abdominal injuries 155
  in shock 118
Haematoma 42–3
  subungual 43
Haemodialysis, in drug overdose 264, 265
Haemodynamic management, in shock 119–20
Haemoglobin measurement
  in abdominal injuries 155
  in shock 118
Haemoperfusion, in drug overdose 264, 265
Haemoptysis 88–90
Haemorrhage
  arterial 59, 61–3
  capillary 59
  classification 59–61
  external 59–60
  internal 60
  management 61–3
  mechanism of arrest 61
  pathological 60
  post partum 296
  primary 60
  reactionary 60
  secondary 60–1
  sites of 59–60
  spontaneous 60
  traumatic 60
  venous 59
  see also Vaginal bleeding
Haemothorax 96
  massive 197
  minimal 96–7
  moderate 97
Hand
  infections 213–15
  injuries 210–11
    diagnosis 210
    initial management 211–12
    specific injuries 212–13
  splinting 203
Head injuries 185
  associated injuries 187–8
  in children 251–2
  clinical picture 185–7
  management 187–8
  and medication 180
Heart
  contusion of 100
  intracardiac rupture 100
Heart attack, *see* Myocardial infarction
Heart failure
  associated with respiratory failure 83
  left ventricular failure 194
  right heart failure 107
Heat stroke 225–6
Heimlich technique
  adults
    conscious patient 133

unconscious patient 133
children 134
Hernia, diaphragmatic, congenital 255
Herpes simplex eruption, associated infections 221
Hip, splinting 204
Hirschsprung's disease 257
HIV (Human immunodeficiency virus), *see* Acquired immunodeficiency syndrome
Hostile patient 183–4
Human immunodeficiency virus (HIV) , *see* Acquired immunodeficiency syndrome
Hypercarbia, difficulty in diagnosis 76–7
Hyperdynamic shock, *see* Septic shock
Hyperemesis gravidarum 280–1
Hyperglycaemic hyperosmolar non-ketotic coma (HHNK) 192–3
Hyperpyrexia 195
Hypertension, during pregnancy 287–8
Hypertensive crisis 111–12
Hypertensive encephalopathy 111–12
Hyperthyroidism 194
Hypoglycaemia 190–1
Hypovolaemia
absolute 113
relative 113–14
Hypovolaemic shock
causes and signs 113–14
management 122
Hypoxaemic failure 79–84
Hypoxia, in thyrotoxic crisis 195
difficulty in diagnosis 76–7
Hysteria 181

Incised wounds 47
Infants, *see* Children
Infarction, myocardial 106–7
Infectious airway obstruction 74–6
Injuries
emergency procedures 39–40
evaluation 38–9
management
general principles 38
specific 40
summary 40–1
transportation of patient 41
Insecticide poisoning 271–2
Internal injury, patient evaluation 39
Intestine, small, injuries to 158
Intracardiac rupture 100
Intracranial tension 171–2
Intravenous cannulation, in children 243–4
external jugular 244–5
hand 244
scalp 243–4
Intravenous pyelography (IVP)
in genitourinary injuries 160
in kidney injuries 155
Intubation
chest 28–9
endotracheal 5–10, 129–30
in children 245–6
nasogastric 31–2
nasotracheal 8–10
orotracheal 5–8
Intussusception 258
Isolation procedures 231–2
stool precautions 233–4
strict isolation 232–3
wound isolation 233
minor wounds 232
IVP, *see* Intravenous pyelography

Jaundice, associated infections 221
Jaw, fractures
mandible 55
maxillae 55
Joints, injuries 205–6, *see also* Dislocations

Ketoacidosis, diabetic 189–90
Kidneys
arteriography 161
failure, in hypertensive crisis 112
injuries 162
intravenous pyelography 155
Knee, splinting 204

Labour
abnormal deliveries 292–5
breech presentation 292–4
normal 290–2
post partum haemorrhage 296
post partum pyrexia 297

Labour *cont'd*
premature births 295
prolapsed umbilical cord 294–5
pulmonary embolism, post partum 297
twin births 295
uterine rupture 295–6
see also Pregnancy
Lacerations 44–5
chest and lung, in war wounds 48
eyeball 58
face 45, 53
fingertips 45
mouth 45
tongue 46
Laryngoscope
in nasotracheal intubation 9
in orotracheal intubation 6–8
Laryngotracheobronchitis, *see* Croup
Larynx, injuries to 50–2
Leg, splinting 204
Leptospirosis 222
Life-saving procedures and priorities 39–40
Lithium toxicity 180
Liver
abscesses 221
injuries to 157
Ludwig's angina 75
Lungs
blast injury 102
missile and bullet injuries 48–9
Lymphadenopathy, persistent generalized 303

Malaria 226–8
Mandible, fractures 55
Masks
partial rebreathing 78
see also Bag and mask ventilation
Masses, and abdominal pain 148
MAST (military antishock trousers) 26–8
Mauriceau–Smellie–Veit technique 293–4
Maxillae, fractures 55
Maxillofacial injuries, emergency management 55
Mediastinal flutter 93
Meningeal irritation, in coma 171, 174
Meningitis, clinical features 219
Meningoencephalitis, clinical features 219
Military antishock trousers (MAST) 24–8
Moles, vesicular 283, 284–6
Mouth
electrical burns to 72
lacerations 45
Myocardial infarction, acute 106–7

Nailbed injuries 212
Nails, subungual haematoma 43
Narcotic overdose 267–8
Nasal airway insertion 4–5
Nasogastric tube insertion 31–2
Nasopharyngeal airway 129
Nasotracheal intubation 8–10
Nasotracheal suction 80–1, 131
Neck injuries 50–2
Nerve injuries
of hand 212
in neck 52
Noise, reaction to, in coma 168
Nosebleed, *see* Epistaxis

Oedema, pulmonary 84–5
Oesophageal varices 151–2
Oesophagus
congenital anomalies in newborn 253–4
injuries 51–2
Oral airway insertion 3
Organophosphate poisoning 271–2
Oropharyngeal airway 129
Orotracheal intubation 5–8
Osteomyelitis, clinical featurs 220
Otitis media
acute, clinical features 221
in children 248–9
Oxygen therapy, in respiratory failure 81–2

Paediatrics, *see* Children
Pain
initial medications 40
response to, in coma 168–9
see also Abdominal pain
Pancreas, injuries to 158
Pancreatitis, acute, diagnostic features 146

Paracentesis, in abdominal injuries 155
Paradoxical breathing 93–4, 95
Paronychia 215
Partial rebreathing mask 78
Pelvic inflammatory disease 220
Pelvis, fractured, in female 161
Penis, injuries to 161
Peptic ulcers, perforated, diagnostic features 146
Pericardiocentesis 30–1
Peristaltic sounds, and abdominal pain 148
Persistent generalized lymphadenopathy (PGL) 303
Petroleum product poisoning 271
Pickwickian syndrome 77
Placenta praevia 283
Plague 222
Pneumonia
  in children 249
  clinical features 219
Pneumothorax
  closed 93
  open 93
  tension 94–5, 98
Poisoning
  antidotes 266
  carbon monoxide 274–5
  cyanide 270–1
  ingested poisons 269–73
  organophosphates 271–2
  petroleum products 271
  strong acids 270
  see also Bites; Drug overdose; Stings
Positive pressure breathing 132
Pregnancy, emergencies during
  convulsions 288–9
  ectopic 282, 284–6
  hyperemesis gravidarum 280–1
  hypertension 287–8
  premature rupture of membranes 289
  see also Labour; Vaginal bleeding
Psittacosis 222
Psychiatric emergencies 177–8
  attempted suicide 178
  dystonic reactions 180
  excitement 178–80
  hysteria 181
  lithium toxicity 180
  stupor 180
Psychoses, *see* Excitement
Pulmonary capillary wedge pressure, in shock 116
Pulmonary embolism 109–11
  post partum 297
Pulmonary oedema 84–5
Punctures 47
Purgatives, for drug overdose 263
Pyelography, *see* Intravenous pyelography
Pyrexia, post partum 297
Pyrexial illness, acute
  diagnosis 219–23
  investigations 223–4
  treatment 224–5

Q fever 222

Rabies 276–7
Rectal examination, in abdominal pain 148
Renal injuries and failure, *see under* Kidneys
Reproductive organs, female, injuries to 159
Respiration
  artificial 137
  emergency procedures 39
  paradoxical 93–4, 95
Respiratory distress, patient evaluation 38
Respiratory failure
  airway obstruction 81
  associated heart failure 83
  carbon dioxide narcosis 82–3
  causes 127
  diagnosis 76–7
  hypoxaemic failure 79–84
  infection 80
  mixed ventilatory and hypoxaemic 79–84
  oxygen therapy 81–2
  pure ventilatory failure 77–8
  sputum retention 80
  stimulants 83
Respiratory symptoms in newborn 253–5
Resuscitation, *see* Cardiopulmonary resuscitation
Retrograde urethrography, in urological injuries 160

Ribs, fractured 90–1
Rigidity, and abdominal pain 147
Rupture
  airways 98
  aorta 101
  bladder 161
  intracardiac 100
  premature, of membranes during pregnancy 289
  uterus, during labour 295–6
Salicylate intoxication 269
Scalp wounds, control of bleeding 188
Scarlet fever, clinical features 220
Sedation, and respiratory failure 83
Sedative intoxication 267
Seizures, in children 240–1
Sengstaken–Blakemore tube insertion 33–5
Septic (hyperdynamic) shock 114
  management 123
Septicaemia 230–1
Shock 113
  cardiogenic 107–8, 114, 123–4
  hypovolaemic 113–14, 122
  management of 118–24
  monitoring 115–18
  patient evaluation 38
  septic (hyperdynamic) 114, 123
  types and causes of 113–14
Shoulder, splinting 202–3
SIDA (Syndrome d'immunodéficience acquise), *see* Acquired immunodeficiency syndrome
Skull fractures 185
Spinal injuries 207–8
  aetiology 207–8
  clinical picture 208
  emergency management 208–9
  initial therapy 210
  life support measures 209
  patient evaluation 38, 208
  transportation of patient 209–10
Spleen, injuries to 157
Splints
  ankle 204–5
  arm 202
  elbow 202
  foot 204–5
  forearm 203
  hand 203
  hip 204
  improvised 201
  knee 204
  leg 204
  shoulder 202
  standard 201
  thigh 204
  wrist 203
Sprains 205
Sputum retention in respiratory failure 80
Stab wounds 44
Status asthmaticus 85–8
  in children 238–9
Status epilepticus 164–5
Stings
  insects 278
  marine animals 278
Stomach, injuries to 158
Stupor 180
Sucking wound (open pneumothorax) 93–4
  war injury 48–9
Suffocation 132
  adults
    conscious patient 133
    unconscious patient 133
  children 134
Suicide, attempted 178
Sunburn 72–3
Sympathomimetic agents, for status asthmaticus 87
Syncope 174
Syndrome d'immunodéficience acquise (SIDA), *see* Acquired immunodeficiency syndrome
Synovitis, traumatic 205–6

Tamponade, cardiac 100, 108
Tenderness, and abdominal pain 147–8
Tendons
  acute tenosynovitis 215
  injuries, of hand 213
  reflexes, in coma 170
Tenosynovitis, acute 215
Tension pneumothorax 94–5
  in airway rupture 98
Tetanus 49–50
Thigh, splinting 204
Thoracentesis 26–8

Thoracic injuries 90
  airway rupture 98
  flail chest 91–2
  fractured ribs 90–1
  haemothorax, *see* Haemothorax
  physiology 90
  pneumothorax, *see* Pneumothorax
Throat, cut 55–6
Thromboembolic complications, in thyrotoxic crisis 195
Thyrotoxic crisis 193–5
TIAs (transient ischaemic attacks) 174–7
Todd's paralysis, vs transient ischaemic attacks 176
Tongue, lacerations 46
Tonsillitis, bacterial, clinical features 220
Tourniquets
  dangers of 61–2
  for finger lacerations 212
Toxaemia of pregnancy 288–9
Toxic shock syndrome 223
Trachea
  injuries to 50–2
  intubation, *see* Intubation
Tracheostomy 130
Tracheotomy 131
Transient ischaemic attacks (TIAs) 174–7
Transportation
  of injured patients 41
  of patient with spinal injuries 209–10
Transtentorial herniation (TTH) 166
Typhoid 228–30
Typhus
  louse-borne 222
  tick-borne 222–3

Ulcers, *see* Peptic ulcers
Ultrasonography
  in abdominal injuries 154
  in urological injuries 160
Umbilical cord, prolapsed, during labour 294–5
Uncontrolled patient 183–4
Underwater seal drainage system 29, 98
Units, list of xii
Urethral catheterization 35–6
  in urological injuries 160–1
Urethral injuries, in male 161–2
Urinary tract infection, clinical features 221
Urinary tract injuries, *see* Urological injuries
Urine output, in shock 116, 118
Urological injuries
  diagnosis 159–61
  management goals 159
  treatment 161–2
Uterus, rupture during labour 295–6

Vaginal bleeding, during pregnancy
  accidental haemorrhage 284, 286–7
  causes 281–3, 286
  diagnosis 284–5, 288
  in the last trimester 286–7
  management 286, 287
Vaginal examination, in abdominal pain 148
Valsalva manoeuvre 28
Varices, oesophageal 151–2
Vasodilators, in shock 121–2
Veins
  catherization, *see* Catheterization, venous
  haemorrhage 59
Venesection 16–17
Ventilation
  bag and mask 2
  mechanical, in status asthmaticus 88
  see also Airway; Respiration
Vesicular moles 283, 284–6
Vomiting, *see* Emesis

War wounds
  chest and lungs 48–9
  danger of infection 46
  management of 47–8
  types of 46–7
Wounds
  abrasions, *see* Abrasions
  bullet, *see* War wounds
  closed 41–3
  contusions, *see* Contusions
  definition 41
  general management 40

Wounds *cont'd*
haematoma 42–3
incised 43
isolation procedures 233
minor wounds 232
lacerated, *see* Lacerations
open 43–6
penetrating, into joints 206
punctures 44
scalp, control of bleeding 188
stab 44
tetanus prophylaxis 49–50
see also Bites; Stings; War wounds
Wrist, splinting 203

X-rays
abdominal injuries 154
abdominal pain 149
urological injuries 160

The Management of Emergencies by the Primary Health Care Physician
World Health Organization
Eastern Mediterranean Regional Office

The role of the primary health care physician is central to The World Health Organization's global strategy for 'Health for All by the Year 2000'. The WHO's Regional Office for the Eastern Mediterranean has taken a number of initiatives in establishing strong foundations for primary health care, particularly in integrating the role of the first referral hospital and introducing basic surgery to primary health care delivery by newly qualified physicians.

This practical and didactic handbook covers accidents, medical, surgical and obstetric emergencies. It provides a comprehensive but concise reference for general practitioners and primary health care physicians. Each chapter discusses the aetiology, differential diagnosis and clinical considerations of emergencies and outlines procedural steps for their management and follow-up.

Also available

Advanced Cardiac Life Support
The practical approach
Advanced Life Support Group
Paperback (0 412 48390 4), 212 pages

The Management of Minor Head Injuries
I.J. Swann and D.W. Yates
Paperback (0 412 29080 4), 102 pages

Clinical Tropical Medicine
G.O. Cowan and B.J. Heap
Paperback (0 412 43330 3), 314 pages

ISBN 0-412-54800-3

CHAPMAN & HALL MEDICAL
London · Glasgow · New York · Tokyo · Melbourne · Madras