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EMERGENCY ANAESTHESIA

EDITED BY

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PREFACE

Throughout the preparation of this book the Editors and Contributors have endeavoured to keep in mind the widely diverse conditions under which emergency anaesthesia is practised. At one extreme is the well-equipped modern urban hospital, with all necessary diagnostic and therapeutic facilities; at the other we have tried to envisage the lone practitioner in some primitive mission hospital, with little or no apparatus and only a few essential drugs and his clinical acumen to help him. We have therefore considered it necessary to include a chapter devoted to description of the standard anaesthetic agents, apparatus and techniques, with emphasis largely on practical aspects in emergency work, touching on theoretical matters as far as possible only where they bear directly on practical problems. Elsewhere in the book, where highly specialised aspects of emergency anaesthesia are discussed, as for neurosurgery, vascular and thoracic surgery, the methods of anaesthesia described are those appropriate to fully-equipped centres, for it is clear that such cases, except for first-aid measures, cannot be successfully treated outside these centres. Wherever possible, useful references have been included to help the post-graduate reader seeking guidance for further study, and it is our hope that the book will prove of value both to the practising anaesthetist faced with the often testing problems of emergency surgery, and to the candidate preparing for the F.F.A. examination.

It is inevitable in a multi-author book that there should be some overlapping of subject-matter. Although the Editors have tried to exclude unnecessary redundancy, they have deliberately retained certain repetitions where the subject was considered to be of sufficient importance to merit restatement in more than one context. The views of individual contributors have been freely stated, and if the reader should encounter some minor divergence of opinion, it would seem a natural consequence where several authors write on a specialty that is, even today, something of an Art and not yet completely a Science.

HARRY L. THORNTON
PETER KNIGHT

October 1964.

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The request to write this book was received with no small measure of diffidence by the editors, to whom the undertaking was a new one, potentially beset by many pitfalls. The fact that our efforts eventually came to fruition has been due in large part to the help of many different people, to all of whom we owe our thanks.

We mention particularly our contributors, who laboured manfully and stoically bore editorial suggestions. Help in initial reading and correction of the manuscript was given by Dr. L. Strunin and Dr. G. F. M. Pierce. Dr. Tom Oppé gave advice on the section dealing with resuscitation of the newborn, and Mr. P. A. J. Starr kindly contributed the description of the technique of retro-ocular block, found in Chapter 5. Mr. Jerry Rytina and the staff of the Photographic Department of St. Mary's Hospital were at all times most co-operative in preparing illustrations, and Dr. Robert Frew kindly acted as a subject for certain photographs of regional blocks. Other photographs and illustrations were supplied by the Photographic Departments of the Westminster, the Middlesex and the Eastman Dental Hospitals, while certain manufacturers, acknowledged in captions elsewhere, have loaned blocks. Our special thanks are due to Miss W. M. Gallagher, B.A., F.L.A., Chief Librarian to St. Mary's Hospital Medical School, whose expert and ungrudging assistance in obtaining books and papers and in tracing references was constantly at our disposal. Miss M. Waldron kindly produced certain illustrations for Chapters 5 and 7, and Miss Veronica Thornton gave invaluable help with the index. The considerable burden of typing much of the manuscript was cheerfully borne by Miss Catherine Coppen.

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H. L. T.
P. F. K.

May 1965.

INTRODUCTION

The Problems of Emergency Anaesthesia

A surgical emergency raises problems for the anaesthetist which are not usually encountered in planned cases. In the latter instance hours, and in special circumstances days, may be available for clinical and laboratory investigation and preparation for surgery, while the technique of anaesthesia can be planned without haste, for the nature and extent of the surgical procedure can usually be foreseen well in advance. In cases of doubtful diagnosis, alternative methods of anaesthesia can be prepared to meet possible surgical requirements.

Time, in planned surgery, is on the side of the anaesthetist. With increasing urgency of the surgical condition this valuable asset diminishes, and the anaesthetist may be faced with the task of compressing into the space of an hour or less, preparations for which a day might not seem excessive in planned surgery. In such circumstances a compromise has to be reached between the ideal approach and that in which optimal anaesthetic conditions are subordinated to the urgency of the surgical intervention.

In many cases of surgical emergency, operation can with advantage to the patient be postponed for an hour or so; there are, in fact, only four common conditions which call for immediate surgical intervention: they are foetal distress, uncontrollable haemorrhage, severe respiratory embarrassment, and cardiac arrest. To these may be added a few comparatively rare conditions, such as arterial embolism. In all cases, and most particularly in those where the patient's general condition gives cause for anxiety, the need for immediate surgery should be critically weighed against calculated delay, when the time so gained could be used for the benefit of the patient. It should not need saying that matters of simple convenience should never be allowed to influence judgement when assessing the urgency of a particular case; but how many of us have not, at some time, acquiesced in some minor infringement of this rule?

It is, of course, not always the major surgical emergency that presents the greatest anaesthetic problem. A "whiff of gas" or a "shot of pentothal" are frequently requested for the incision of an abscess, but should this happen to be sited in the pharynx such simple methods of anaesthesia can be hazardous, and safety will demand a meticulous and expert technique. A minor surgical emergency may carry a major anaesthetic risk that need not be immediately apparent; there are, for instance, an increasing number of new drugs (and some older ones, such as cortisone) that are given to patients without any warning of their potential danger in anaesthesia. It is unfortunately in trivial cases that the anaesthetist is likely to be taken unawares, and a careful preliminary interrogation and examination of the patient is as important in minor as in major surgical emergencies.

When considering the choice of anaesthetic technique for any surgical emergency, it is a sound rule for the anaesthetist to adhere to those methods with which he has become familiar, making such minor modifications as the particular circumstances demand. New techniques and drugs should be reserved for "cold" cases and only added to the emergency armamentarium after practice has brought familiarity and confidence. Sound clinical judgement and the capacity to recognise and anticipate dangerous situations are in the long run the most valuable assets of the emergency anaesthetist.

CONTENTS

CHAPTER 1

PREOPERATIVE ASSESSMENT AND PREPARATION OF THE PATIENT

Cases requiring emergency anaesthesia fall into three main groups:

1. Those whose general state has not been adversely affected by the surgical condition.
2. Those whose general state has been so affected.
3. Those with intercurrent disease or complication that might affect the course of anaesthesia.

(I) Cases in which the general state is not affected by the surgical condition

These may be considered as "cold" cases, and insofar as anaesthesia is concerned are subject only to the following *General Rules of Safety*:

(i) A complete physical examination must be made, and a medical history obtained where possible.
(ii) Allowance must be made for drugs given (stimulants and sedatives) prior to admission to hospital.
(iii) Measures must be taken to guard against aspiration of gastric contents during anaesthesia.

PHYSICAL EXAMINATION AND PREOPERATIVE ASSESSMENT OF THE PATIENT

It is not presumed that the anaesthetist will be required to make a surgical diagnosis; nevertheless it will be his duty to make as full an examination as possible, to exclude any factors that might affect the choice of anaesthetic or course of anaesthesia. The discipline of a full examination in every case, moreover, will help him readily to recognise deviations from the normal, and to assess the need for any special resuscitative measures.

Before proceeding to the detailed examination, note should be taken of the patient's age, and this equated with the physical appearance, the approximate weight, the general physique, state of nutrition, hydration and the level of consciousness. Any evidence of haemorrhage or the appearance of shock should be looked for. Attention should be directed particularly to any visible disease or abnormality, and where possible the history of any past illnesses, medication and anaesthesias should be obtained. If the patient admits to drug-therapy of any description, an effort should be made to determine the nature of the medication, and the date or time of the last dose. The time of the last meal or drink should also be determined, and a specimen of urine obtained to be tested for sugar, ketones, albumin and porphobilinogen (see Chapter 16).

In performing the detailed examination, priority should be given to the respiratory and cardiovascular systems.

The respiratory system

Nose and mouth. Haemorrhage, vomitus, loose or missing teeth, signs of trauma and presence or absence of dentures should be noted, and the appropriate action taken, where necessary, to clear and secure the airway. (See Anaesthesia for Maxillo-Facial Emergencies, Chapter 9.)

Respiratory function. In making an over-all clinical assessment of respiratory function, attention should be paid to the rate and depth of respiration, and its apparent mechanical efficiency, as well

as to the presence of dyspnoea or cyanosis. If possible, some simple test of respiratory reserve (such as requiring the patient to count aloud after a single breath) should be performed, and the result equated with any physical signs elicited on examination of the chest, together with any history of past or present respiratory disorder. Where respiratory function is obviously impaired, the anaesthetist should endeavour to determine whether this is due to mechanical causes, such as upper airway obstruction (usually relieved by simple measures, though perhaps requiring tracheal intubation or tracheostomy), loss of intercostal muscular activity (which may require the use of assisted ventilation) or to pulmonary disease such as asthma, severe emphysema, bronchiectasis with much secretion, or other gross disorder. (The management of anaesthesia in cases of *respiratory disease* is discussed in Chapter 16.) In traumatic cases, thoracic injury must be considered as a cause of respiratory dysfunction. Although fractures of the sternum and ribs, as in crush injuries, are usually immediately obvious, small penetrating wounds which may cause haemo- or pneumo-thorax are easily overlooked. (For details of anaesthesia for emergency thoracic surgery see Chapter 12.)

The cardiovascular system

Of particular importance are the pulse rate, blood pressure and state of filling of the peripheral veins. They should be related to the general appearance of the patient and the nature and severity of the surgical condition, to exclude the state of shock or establish its cause. Cardiac arrhythmias, murmurs, displacement of the apex-beat, or any other abnormal physical signs must be considered (where possible) in conjunction with the patient's past medical history, medication and customary physical activity. The patient should be questioned as to any past illness suggestive of cardiac ischaemia and/or failure. Thyrotoxicosis, aggravated by a surgical emergency, must not be forgotten as a possible cause of cardiac failure. Most chronic cardiac diseases, provided that they are well compensated, or in the case of atrial fibrillation well controlled by drugs, are seldom a bar to general anaesthesia, provided that certain precautions are carefully observed; they may be summarised as:

The maintenance of maximal arterial oxygen saturation.
Avoidance of hypotensive episodes.
Avoidance of atropine in premedication (most particularly in cases of ischaemic heart disease and where atrial fibrillation is controlled by digitalis).
Provision of adequate pre- and postoperative sedation.

Of all cardiac conditions only *congestive cardiac failure* need be considered an *absolute contra-indication to general anaesthesia*, but severe cardiac disability, such as might be shown by dyspnoea or angina at rest should suggest an alternative to general anaesthesia, provided that (1) the psychical ordeal to the patient does not outweigh the merits of conduction analgesia, and that (2) the avoidance of general anaesthesia will not materially hamper or prolong the surgical procedure. It is important in these circumstances that hypotension due to conduction block is avoided.

(For a fuller discussion of anaesthesia in relation to cardiac disease see Chapter 16.)

The central nervous system

The level of consciousness must be assessed. If there is drowsiness, or other signs of depression of the C.N.S., intoxication with drugs or alcohol should, if possible, be excluded (noting the size and reaction of the pupils to light, condition of the other ocular reflexes, response to painful stimuli and odour of the breath). In the absence of a medical history, the various causes of coma must be excluded, and the urine tested for albumin, sugar and ketones, by catheter-specimen if

necessary. (It is worth noting that the odour of acetone in the breath may easily be confused with that of alcohol.) Evidence of head injury must be sought, and the possibility of intracranial damage considered. (Where intracranial damage co-exists with another surgical condition, the anaesthetist should be prepared for treatment of the former to take precedence over that of the latter.) Disturbance of consciousness, from whatever cause, is a bar to the use of all drugs, which are liable to depress the respiratory centre, either in premedication or anaesthesia. For the control of restlessness, paraldehyde (4–8 ml., by deep intramuscular injection) is the drug of choice in such cases. In cases of coma, anaesthesia will of course be unnecessary, but the anaesthetist may be required to perform tracheal intubation with a cuffed tube to ensure a free airway, to guard against inhalation of vomitus and secretions, and for subsequent anaesthesia if required. In cases of increased intracranial pressure craniotomy may be followed by a dramatically sudden return of consciousness, accompanied by motor restlessness. With an endotracheal tube in place, induction of anaesthesia can be achieved with the least possible disturbance to the patient and the surgical field.

(The problems of anaesthesia for neurosurgical emergencies are fully discussed in Chapter 11.)

ALLOWANCE FOR SEDATIVE AND OTHER DRUGS PREVIOUSLY ADMINISTERED

In shocked patients with poor peripheral circulation the absorption of drugs given by subcutaneous and intramuscular injection is greatly retarded. This fact must be taken into consideration when planning premedication and anaesthesia in such cases. If morphine or a similar drug is known to have been given within a 4-hour period, no further opiate should be administered prior to induction of anaesthesia; vasodilatation under anaesthesia may result in the rapid absorption of a drug given some hours previously, thus potentiating any further drugs given for premedication. In such a case undesirable respiratory depression may be produced. All sedative drugs reduce the anaesthetic requirements and particular caution must be exercised in the use of intravenous barbiturates, such as thiopentone, in the previously sedated patient. Alcohol must not be forgotten as a drug capable of potentiating the centrally-depressant effects of the opiates and barbiturates.

"Tranquillisers"

An ever-increasing variety of drugs is widely prescribed for the alleviation of psychotic conditions, tension and anxiety states, depressions, and sometimes for no clear clinical reason at all. Although of diverse pharmacology, the psychotropic drugs include two groups that are of significance to the anaesthetist. They are the phenothiazines and the monamine–oxidase inhibitors (M.A.O.I.).

The phenothiazines are relatively benign in their influence on anaesthesia, and are, in fact frequently employed in premedication and as anaesthetic adjuvants. They all possess hypotensive properties to a varying degree, and can counteract, also to a varying degree, the vaso-constrictor action of the pressor amines. These potentialities must be allowed for when anaesthetising patients who have been under treatment with a phenothiazine drug for any length of time, and most particularly in the case of chlorpromazine (Largactil).

Monamine–oxidase inhibitors (*M.A.O.I.*). Patients who have been receiving continuous treatment with a M.A.O.I. are rendered unusually sensitive to all narcotic agents, and particularly to pethidine, so that a therapeutic dose of the latter may cause deep coma and hypotension. In addition, the M.A.O.I. all potentiate the action of the pressor amines to a dangerous degree. These side-effects of the M.A.O.I. constitute a potentially grave hazard in emergency anaesthesia unless the anaesthetist is informed that a patient has been under treatment with one of these

drugs. For example, a pressor amine may be given in good faith to avert circulatory collapse following premedication with pethidine or a similar drug, and a severe, or even fatal, hypertensive episode may well then result.* After extended periods of treatment the side-effects of the M.A.O.I. may still operate for 1–2 weeks following withdrawal of the drug. The anaesthetist should be particularly cautious in his choice of drugs and agents in any patient who admits to being under treatment for "nerves", and unless the patient can tell him the name of the drug he has been receiving, or the private doctor can be approached to supply the necessary information, it would be wise to assume that a M.A.O.I. is involved.

The *Lancet* (1964) quoting the Committee on Safety of Drugs, lists the following preparations as potentially dangerous in the present context:

Isocarboxazid (Marplan)	Mebanazine (Actomol)
Pheniprazine (Cavodil)	Phenoxypropazine (Marsalid)
Pargyline (Eutonyl)	Nialamide (Niamid)
Phenelzine (Nardil)	Pivhydrazine (Tersavid)
Tranylcypromine (Parnate)	Tranylcypromine with Trifluoperazine (Parstelin).

Anti-convulsants

With the exception of *primidone* (*Mysoline*), none of the drugs used in the treatment of epilepsy and allied disorders are likely to react unfavourably with anaesthetic agents and premedicants. Primidone therapy renders the patient abnormally sensitive to the action of the barbiturates, greatly prolonging recovery time and depressing respiration. If intravenous induction of anaesthesia with a barbiturate is considered to be essential in such cases, dosage should be reduced to the minimum necessary for abolition of consciousness. In known epileptics, where the exact nature of previous drug therapy cannot be established, the anaesthetist should proceed on the assumption that primidone has been employed.

Drugs acting on the cardiovascular system

Digitalis. It is not uncommon for elderly patients to receive regular dosage of digoxin or other preparation of digitalis for a chronic cardiac condition. Unless there is an adequate reason, such as an over-rapid heart rate, signs of impending cardiac failure or obvious decompensation, it is unwise to increase the dosage of digitalis in preparation for surgery. *Atropine should never be used in premedication in the digitalised patient*, or in cases of impending failure, since the tachycardia thus caused could well result in cardiac decompensation. The action of digitalis is sensitive to changes in the serum potassium; thus, massive transfusion of stored blood, by raising the serum potassium to a high level can partially antagonise the action of digitalis, and precipitate cardiac failure; this complication can, to some extent, be avoided by the adequate infusion of calcium gluconate (see Chapter 2, Section 1). Conversely, in hypokalaemic states (such as intestinal obstruction) the action of digitalis may be dangerously potentiated. Cyclopropane, halothane and chloroform, any of which are capable of slowing the heart rate in normal patients, should be used with extra caution in the digitalised patient lest a dangerous degree of bradycardia be produced. This stricture also applies in cases receiving *quinidine*.

Hypotensive drugs. The hypertensive patient under treatment with a hypotensive drug is partially deprived of his reflex defence-mechanism to shock and haemorrhage; he will react poorly to the action of the pressor amines, and is likely to exhibit an exaggerated response to anaesthetic drugs and agents which can depress the blood pressure. In such patients, timely and meticulous

* Chlorpromazine (Largactil) 50–100 mg. should be given intravenously to reverse the hypertension.

replacement of blood and fluid loss is vitally necessary. Thiopentone, if used for induction of anaesthesia, must be given slowly, and in minimal dosage. Halothane is best avoided, or given in low concentration, with utmost caution. If a relaxant is required, gallamine is preferable to *d*-tubocurarine, unless otherwise contra-indicated, but where the latter is considered necessary it must be given in small divided doses, allowing time for the blood pressure to recover after each dose. Postoperatively, hypotensive therapy must not be resumed until the blood pressure has recovered to a safe level.

It must also be remembered that all ganglionic blocking agents potentiate the action of the insulins in diabetic patients.

Drugs used in obstetrics

Ergometrine is liable to potentiate the action of the pressor amines to a dangerous degree (Casady, 1960). This fact must be borne in mind in the treatment of post-partum shock.

Pitocin, either by injection or infusion must never be used in conjunction with cyclopropane anaesthesia, since traces of pitressin could in such circumstances result in coronary spasm and ventricular fibrillation.

Trichlorethylene. When trichlorethylene has been used for analgesia in the early stages of labour, it is unwise to use the closed-circuit in subsequent anaesthesia, lest there remains sufficient trichlorethylene vapour in the patient's expired air to react with the soda-lime in the absorber to form toxic by-products (see Chapter 4).

The Steroids (see Chapter 16).

The Antibiotics (see Chapter 4).

The Anticoagulants.

If patients have been receiving anticoagulant therapy within 24 hours of surgery, it may be considered advisable to restore normal blood-clotting preoperatively, to minimise blood loss.

Heparin is neutralised by protamine sulphate, 2 mg. for each 1 mg. of heparin given within the last 4-hour period.

Phenindione (Dindevan) is antagonised by vitamin K_1: 10 mg. of the water-miscible solution should be given by slow intravenous injection, and may be repeated.

MEASURES TO GUARD AGAINST ASPIRATION OF GASTRIC CONTENTS DURING ANAESTHESIA

Of all the hazards of emergency anaesthesia, vomiting and regurgitation of stomach contents, and subsequent aspiration, is possibly the most common, and in its immediate and remote effects one of the most serious, should it occur at a time when the protective reflexes are in abeyance. The only certain insurance against such an accident is an empty stomach; the danger lies in the fact that a full stomach is so often unsuspected. The assurance that nothing has been taken by mouth for several hours is unfortunately generally accepted as a safe indication of an empty stomach, but although in the majority of cases this may be true, there are important exceptions.

Delay in gastric emptying can occur in association with:

Deep sedation (morphine to relieve pain).
Anxiety, pain and fright (especially in children).
Shock.
Onset of labour (delay of 36 hours has been recorded).

Presence in the stomach of hypertonic dextrose solution (this can result from faulty therapy in cases of diabetes).

In the absence of any of the above complicating factors, it is reasonably safe to assume that the stomach is empty *provided that nothing, solid or liquid, has been taken by mouth for at least* 4 *hours*, and that *normal peristalsis is taking place.*

Where there is the least doubt concerning an empty stomach, a large stomach tube should be passed, the stomach washed out until the aspirate is clear, and the tube left in place for periodic aspiration up to the time of induction of anaesthesia. (The anaesthetic management of cases suspected of having a full stomach is fully discussed in Chapters 6 and 7.)

(II) Cases in which the general condition has been adversely affected by the surgical emergency

These cases may be subdivided into two classes:

1. The *shocked.*
2. Those *with disorder of physiological function other than shock.*

The latter problem is dealt with in the appropriate special chapters and is mostly concerned with respiratory impairment of central or peripheral origin, and with fluid and electrolyte imbalance occurring in consequence of trauma.

Shock

The term "shock" is used somewhat loosely to describe the syndrome of peripheral circulatory failure, to which a number of factors are contributory. The outward signs are pallor, sweating, hypotension, diminished pulse-pressure and usually increased pulse-rate, cyanosis of the nail-beds and extremities, and shallow or sighing respiration. The skin is cold to the touch and the veins contracted. All these signs may be present without blood loss, but more commonly result from severe haemorrhage or loss of tissue-fluids. Those cases in which there is no hypovolaemia (loss of blood or tissue-fluids) are classified as *neurogenic shock* (a condition that may result from pain, fright, trauma to large nerve-plexuses or "vasovagal" episodes). It should be realised that in all cases the mechanism responsible for the syndrome is essentially the same: namely a relative decrease in the effective circulatory volume, but whereas in neurogenic shock there is no actual loss of blood or tissue-fluids, in hypovolaemic shock there is a deficit in the total blood volume. In neurogenic shock the effective circulatory deficit can be accounted for by vascular dilatation and pooling of blood, resulting from the failure of normal vascular tone. Since neurogenic and hypovolaemic factors may co-exist in the same case, it is important to distinguish which factor is predominant. Where trauma is wholly external, it will not be difficult to assess the extent to which loss of blood and tissue-fluids (as in the case of extensive limb injuries and burns) contributes to the over-all picture of shock. Where the possibility of concealed (intra-abdominal, intra-thoracic or retroperitoneal) haemorrhage is suggested by the nature of the injury, and where a state of shock persists after a reasonable period of sedation and observation, the anaesthetist should act on the assumption that he is dealing with haemorrhagic shock, for which blood transfusion or, as a temporary measure, infusion of plasma or plasma-expander is the required treatment (see Chapter 2, Section 1).

(For a detailed consideration of traumatic and burn shock see Chapter 8.)

Bacteraemic shock is a condition of profound circulatory collapse, caused by a massive invasion of the blood stream by bacterial toxins. The causative organisms are almost invariably Gram-negative. Bacteraemic shock may follow upon grave surgical conditions such as peritonitis, gas

gangrene or septic abortion, but the most common cause is instrumentation of the urethra in the presence of urinary sepsis. The mechanism responsible for the circulatory collapse is not fully understood, but it is assumed that there is both venous and capillary pooling as in neurogenic shock, and possibly increased capillary permeability. Adrenocortical failure has been suggested as a factor in the past, but this has now been largely discounted; rarely suprarenal infarction may occur as a result of prolonged hypotension.

Treatment of bacteraemic shock must be directed to:

(1) administration of the appropriate antibiotic, and
(2) support of the circulation.

An intravenous infusion of dextrose, 4·3%, with saline, 0·18%, is set up, and in cases where the identity of the infecting organism is already known (as might be the case in an acute-on-chronic urinary infection) the specific antibiotic is added; otherwise, blood having been withdrawn for subsequent culture, a broad-spectrum antibiotic, such as tetracycline, is added to the infusion (250 mg./l.: 1 l. in 8 hours).

A separate infusion of metaraminol, 10 mg./500 ml. in normal saline, may be necessary to maintain the blood pressure, and infusions of blood or plasma should also be given. The routine administration of hydrocortisone is seldom justified, and it is only advisable to resort to steroid therapy if the administration of antibiotics and pressor-amines fails to restore the blood pressure. In such a case hydrocortisone hemisuccinate, 100 mg., should be given intravenously, and repeated in one hour if response to the initial dose is favourable.

(III) Cases of intercurrent disease or complication that may affect the course of anaesthesia

This group of cases comprises a number of medical conditions requiring special preoperative, anaesthetic and postoperative management. Except for those relevant to other chapters and sections, these conditions are considered separately in Chapter 16.

REFERENCES

Casady, G. N. (1960). *J. Amer. med. Ass.*, **72**, 1011.
Lancet (1964), **1**, 506.

CHAPTER 2

SECTION I: BLOOD TRANSFUSION

The viability of tissues depends upon an adequate supply of oxygenated blood, the pH, electrolyte and colloid content of which must be maintained within certain narrow limits. The immediate effect of a profound haemorrhage is to reduce the quantity of blood perfusing certain tissues, and to lower its oxygen content.

The sudden reduction of the circulating volume that follows haemorrhage evokes compensatory changes in the unanaesthetised person which tend to support the central aortic blood pressure, and maintain an adequate flow of blood to the heart and brain. The most significant change is produced by vasoconstriction affecting the skin and the viscera, including the bowel, liver, spleen, kidneys and the lungs. If the blood loss has been even more severe, then this mechanism may fail to maintain adequate venous return to the heart, and cardiac output will fall, in spite of an increase in cardiac rate. Cournand *et al.* (1943) showed that the cardiac output may be reduced by 50 per cent if the blood loss is equal to a third of the circulating volume. An unreplaced loss of 4·5 per cent of the body weight as blood is incompatible with life (Wiggers, 1950). If the blood loss is less rapid, then fluid, salt, and later protein, pass into the vascular compartment in an effort to maintain the circulating volume. A previously normal person will compensate for a loss of one litre of blood in 36 hours (Ebert *et al.*, 1941). This will ameliorate the circulatory effects of the haemorrhage, but will produce a dilution of the blood and a fall in the packed cell volume (P.C.V. normal = 41–45 per cent) and oxygen carrying capacity. The resultant anaemia will accentuate the effects of any subsequent blood loss, as a fall in the P.C.V. below 30 will itself depress cardiac contractility (Case *et al.*, 1955).

The reduction in volume of blood perfusing the tissues, due to the combination of vasoconstriction and depressed cardiac output, necessitates a greater oxygen abstraction. Cellular hypoxia may result, and if untreated lead to irreversible damage and ultimately to cellular death. It is essential that this process be reversed as quickly and as completely as possible. Blood and fluid infusions are necessary for this purpose. They must be given in adequate amounts and at a sufficient rate to support the circulation, and obviate the physiological need for vasoconstriction. The use of vasoconstrictor drugs merely conceals the fundamental physiological defect produced by haemorrhage, and for this reason may lead to insufficient blood replacement.

Before transfusing any patient, consideration must be given to the quantity and nature of the fluid loss, and to the optimum rate of its replacement.

The fluids available for transfusion (with their respective properties), the means of assessing the extent of the loss, and the means of its replacement, are considered below.

ASSESSMENT OF BLOOD LOSS

The clinical signs of haemorrhage are so varied as to be of little value in the accurate estimation of blood lost. The average fit patient will usually withstand the loss of 10 per cent of his circulating volume without ill effects. However, a 20 per cent loss of the circulating fluid is likely to cause faintness, nausea and tachycardia. Thirst, apprehension and perspiration are other common symptoms. Beecher (1949) and Fisher (1958) emphasised the lack of correlation between pulse

rate and blood pressure and the amount of blood lost. Compensation may be so complete, especially in young people, that the blood pressure and pulse may be little affected, even by quite large losses of circulating fluid. In spite of this, a rising pulse rate of over 100/min. and a blood pressure below 80 mm. Hg systolic usually indicate a fairly severe depletion of the circulating volume.

Anaesthesia depresses the compensatory mechanisms evoked by haemorrhage. Deep anaesthesia, ganglionic blockade and spinal or epidural analgesia limit the vasoconstriction possible, so that under these conditions hypotension nearly always follows a sudden large blood loss, accompanied by a rising pulse rate if the vagal tone has not already been blocked by large doses of atropine.

Various methods of assessing the blood loss due to trauma have been suggested, to overcome the inaccuracies due to reliance on blood pressure and pulse rate. Grant and Reeve (1951) suggested estimating tissue damage in terms of volume, the standard of volume being that of the clenched fist.

1 or less hands: associated with a loss of 10–20 per cent of the blood volume.
1–3 hands: associated with a loss of 20–40 per cent of the blood volume.
3–5 hands: associated with a loss of up to 40 per cent of the blood volume.

Clarke and Fisher (1956) studied the blood loss following closed fractures. This has provided a useful guide to the magnitude of the concealed haemorrhage that may occur following trauma, i.e.

Fractures around the foot with moderate swelling: 250–500 ml. loss.
Fractures of the leg with moderate swelling: 500–1,000 ml. loss.
Fractures of the shaft of the femur: 500–2,000 ml. loss.
Fractures involving the knee joint: about 2,000 ml. loss.
Fractures of the forearm: 500–750 ml. loss.
Fractures of the upper arm extending into the shoulder: 2,000 ml. loss.

In a similar manner the "Rule of nines" method of estimating the fluid loss following burns (Berkow, 1924) has proved of great value in gauging the extent of replacement required. This envisages the body as composed of eleven units, each having a surface area of 9 per cent of the total. i.e.

Upper limb : 1 unit (9 per cent of the whole)
Lower limb : 2 units (18 per cent of the whole)
Front of trunk : 2 units (18 per cent of the whole)
Back of trunk : 2 units (18 per cent of the whole)
Head and neck : 1 unit (9 per cent of the whole)
Perineum : 1 per cent of the whole

Assessment in this way of the area of body surface burnt is used in calculating fluid requirements, 1 l. of fluid being required in each 24 hours for every 10 per cent of the surface area involved (Bull, 1954). In addition, the P.C.V., plasma protein and haemoglobin should be repeatedly estimated in any severely burnt patient. This will assess the adequacy of the replacement therapy, and reveal the occurrence of hypoproteinaemia, due to insufficient plasma or albumin replacement, and anaemia. If the burned area has extended over 25 per cent of the surface it will be necessary to give up to half the replacement fluid as blood. Burns involving deep structures will be associated with considerable destruction of red blood cells and should also be treated with blood transfusion.