Chapter 25: Intensive care and resuscitation in otolaryngology

Julian M. Leigh

The intensive care unit in a district general hospital offers a service to a wide variety of patients. The types of management available for patients may, broadly speaking, be subdivided into specific therapy, expectant monitoring and routine system maintenance.

Examples of therapy would be management of diabetic ketoacidosis with insulin, fluid replacement and alkali; the treatment of severe chest infection by antibiotics and physiotherapy; or the treatment of renal failure by dialysis.

The classic service under the heading of expectant monitoring is carried out for patients with myocardial infarction who are in danger of suffering from acute dysrhythmias or cardiogenic shock. This category would also include patients with upper airway problems which are within the province of otolaryngological surgery.

Finally, under routine maintenance would come care of nutrition, fluid and electrolyte balance in any patient unable to control these, even though the primary problem under treatment may be principally in another physiological system.

Relationships between intensive care and otolaryngology

A service to otolaryngological patients is provided by the intensive care unit in the case of children with upper airway obstruction. This is seen not only in acute epiglottitis but also in the inspissated secretion syndrome, which may occur with severe pneumonias and dehydration in small children. Adult admissions to the intensive care unit tend to be for upper airway obstruction caused usually by pharyngeal or laryngeal carcinoma. These patients are sometimes admitted for management before surgery, but are mostly admitted postoperatively to provide the intensive nursing observation and management of the airway which are necessary in the first 24 hours.

Although admission may be less important after laryngectomy, it is still regarded as a reasonable policy for such patients to receive 24 hours of management in the intensive care unit, not only for the airway but also because lengthy surgery may have caused problems with fluid balance and body temperature maintenance.

The other important relationship with the otolaryngology department arises from what might be called the 'tracheostomy service' for patients requiring that manoeuvre for long-term intermittent positive pressure ventilation (IPPV), such as patients with head injuries, and multiple trauma cases with chest complications. It is also important that the otolaryngology department should be involved in the formulation of intensive care unit policies for the 'routine' management of tracheostomies. This includes not only the policy on surgical technique but also policies on what types of tube should be used, dressings, humidification and suction, and so on. The details of the policies are not as important as their being agreed and standardization.
The intensive care services

In intensive care units, general management procedures of problems in the different body systems are not necessarily influenced by the specific cause of admission. General policies and management techniques of major body systems will now be discussed under separate headings.

Respiratory system

The corner-stone of intensive care is respiratory management, particularly intermittent positive pressure ventilation. Many surgical patients in the past have died from insidious respiratory failure in the postoperative period. Age, previous respiratory disease, prolonged surgery and, particularly, postoperative pain and endotoxaemia with humoral pneumonitis, all contribute to inadequate alveolar ventilation, sputum retention and respiratory death.

Many patients, particularly the elderly, undergoing oesophageal and arterial surgery and/or other emergency major intra-abdominal procedures, are currently ventilated postoperative in the intensive care unit. Fluid and electrolyte balance is adjusted and continuous analgesia is provided either by intravenous narcotic administration or with epidural opiates. Weaning from intermittent positive pressure ventilation takes place after about 24 hours and feeding regimens are introduced. The result of this type of management is a profound reduction in morbidity and mortality.

Physiotherapy and fibreoptic bronchoscopy are important when sputum retention is a problem, as is full hydration of the patient. The newly available technique of inserting an indwelling 'mini-trach' laryngotomy suction tube is a useful adjunct to clinical practice.

When upper airway obstruction is a problem, time can be bought by medical means by using oxyhelium therapy. This mixture of 21% oxygen with 79% helium has one-third of the density of air and, as turbulent flow at such sites is predominantly influenced by gas density, the flow of respired gases past an obstruction can take place with one-third of the pressure drop.

The table summarizes respiratory function, failure, diagnostic points, causes and treatment. In practice, any or all types of respiratory failure may coexist in a given patient. The situation must be unravelled by using clinical criteria as well as blood gas analysis.

Cardiovascular system

An understanding of the cardiovascular system is fundamental to the practice of clinical medicine.

The object of cardiovascular system is that the tissues should be perfused for the exchange of nutrients. The requirements are: first, that the heart should function as a demand pump, responsive to the magnitude of the venous return; and, second, that there should be an adequate distributive system to individual organ capillary beds. The heart pump requires the following: that its power output is kept within its own capability to supply itself with nutrients
via the coronary circulation, that it has a near normal rhythm; and that the ejection fraction of the ventricle is also near normal.

Failure in the system produces conditions known as central circulatory (cardiac) failures and peripheral circulatory failure. In the former, the central venous pressure (the preload) is high, indicating pump failure, and is attended by backward oedema. In the latter, the preload is low and ischaemia of specific capillary beds may be manifest, for example skin vasoconstriction.

The management of pump failure may require inotropic support to increase the efficiency of the myocardium and to control rhythm and, finally, measures to reduce the power output of the heart by means of vasodilators such as nitrates. Treatment of peripheral circulatory failure usually requires infusion or transfusion of the appropriate fluids.

Renal management

The function of the renal system may be summarized as the excretion of non-volatile waste products, and of surplus water and electrolytes in order to maintain the milieu interieur.

The way in which this is achieved is that the million nephrons in the kidneys filter the plasma volume over 50 times per day, whereupon the 40 miles of tubules elaborate this filtrate according to the requirements of acid-base balance, water and electrolyte surpluses or deficiencies and nitrogen excretion.

The system requires local autoregulation of blood flow at glomerular level, and active and passive processes within the tubules under the control peripherally of hormonal and humoral mechanisms initiated in the kidney by the renin-angiotensin system and, centrally, in the hypothalamus and pituitary. An additional requirement is that the elaborated urine can be voided to the exterior by an uninterrupted genitourinary tract.

From the clinical point of view, failure of urine elaboration may be prerenal, renal or postrenal. Prerenal failure occurs when the filtration pressure is low in consequence of, for example, hypotension and hypovolaemia. Postrenal failure occurs with genitourinary tract obstruction. Renal (nephron) failure may be either acute or chronic; the former commonly occurs in surgical patients as a consequence of severe hypotension and endotoxaemia, while the latter has a variety of causes including pyelonephritis and atherosclerosis.

Clinically, a distinction usually has to be made between prerenal and acute renal failure in the context of surgery. Both are characterized by oliguria, but in prerenal failure the osmolarity of urine is high, so the clearance of osmolar particles is not reduced, whereas in acute nephron failure the urine is isotonic. Retention of water, sodium, potassium, urea, creatinine and hydrogen ions will follow to a greater or lesser extent. In practice, two clinical forms of oliguric renal failure with nitrogen retention are seen postoperatively; one of these is relatively benign and potassium and hydrogen ion excretion are unaffected, while in the second type significant potassium retention and acid-base disturbance occurs.

The former condition requires fluid intake of a volume equal to the urine output plus insensible loss, by using high concentration carbohydrate solutions. In the latter condition, this
management must be accompanied by bicarbonate (alkali) therapy to combat metabolic acidosis, and glucose/insulin infusions plus potassium exchange resin in an attempt to lower potassium levels. If these measures are not effective, then either peritoneal dialysis, haemodialysis or ultrafiltration must take place.

Symptoms appear when the glomerular filtration rate (measured by creatinine clearance) falls from 120 to less than 30 mL/minute. If the glomerular filtration rate is less than 3 mL/minute, then dialysis and/or transplantation is required.

**Management of nutrition**

As malnutrition is not uncommon in certain types of otolaryngological patient, this topic will be dealt with in some detail.

**Types of patient requiring nutritional support**

Patients requiring nutritional support are those suffering from the following conditions:

1. interference with gastrointestinal function: poor dentition; dysphagia; obstruction; prolonged paralytic ileus from any cause, for example peritonitis, pancreatitis, major surgery; gut fistulae; malabsorption and short bowel syndrome
2. renal failure
3. severe burns, trauma and sepsis and other hypermetabolic states
4. cachexia caused by severe cardiac or respiratory disease
5. severe psychological disturbances, for example anorexia nervosa.

**Types of malnutrition**

Basically there are two types of protein/energy malnutrition: first, the chronic fasting-adapted starvation known as marasmus; second, the fasting-unadapted starvation of critical illness accompanied by the stress response, which may be made worse by surgical intervention. If protein and energy requirements are chronically unsatisfied because of a lack of exogenous replacement, catabolism of somatic protein and fat stores follows, leading to a kwashiorkor-like state.

A mixed condition occurs when, for example, a patient with dysphagia resulting from pharyngeal carcinoma and marasmus undergoes surgery without prior protein/energy replacement.

**Fasting-adapted starvation**

In chronic starvation, there is a progressive fall in both energy requirements and nitrogen loss. Once the carbohydrate stores have been utilized, it is the fat metabolism which supplies energy requirements, while glucose deficits are made up from the carbon skeletons
of deaminated amino acids. Early on, the ratio of fat utilization to protein is about 2.5:1, while at full adaptation this ratio increases to 7.5:1, which means that, to some extent, body protein is spared in relation to fat. This adaptive process breaks down if the stress response is initiated. Breakdown of endogenous protein is a significant source of energy under these circumstances, and the metabolic expenditure closely parallels nitrogen losses. These losses can be reversed by feeding traumatized patients with high calorie/protein diets. A full explanation of the role of protein and fat in starvation may be understood with reference to the schema of intermediary metabolism.

Carbohydrate metabolism

Carbohydrate is the usual source of energy. Glucose is metabolized via glucose-6-phosphate to pyruvate and thence to acetyl coenzyme A which is incorporated into the citric acid cycle by combining with oxaloacetate to form citrate. This pathway, in which glucose finally becomes carbon dioxide and water, liberates 38 molecules of energy-rich ATP. Carbohydrate yields 40 cal/g.

When metabolism of glucose is not required, excesses are stored as glycogen. Glycogen stores occur in the liver (up to 8%) and in muscle (up to 2%). As there is much more muscle than liver, the former constitutes the major store of glycogen, although it can be utilized only by muscle.

The steps on the main glycolytic pathway are all reversible apart from the last one, where pyruvate is metabolized to acetyl-CoA.

Fat metabolism

The metabolism of fat yields 9 cal/g. The main fats of interest in animal metabolism are glycerides. These consist of glycerol combined with up to three substituted chains of fatty acids. Metabolism yields glycerol which, by way of alpha-glycerophosphate, is incorporated in the glycolytic sequence. The fatty acids are metabolized to acetyl coenzyme A by being split off two-carbon atomas at a time, each yielding 17 energy-rich ATP molecules. When, for example, palmitic acid is totally broken down, the side chains yield 130 molecules of ATP, which explains why fat has such a high energy yield.

As acetyl coenzyme A cannot be resynthesized to pyruvate, fat metabolism does not yield glucose intermediates (other than glycerol in small quantities) for energy transport; nor does it supply energy for tissues which can use only glucose - predominantly the brain. Thus fat metabolism is incapable of maintaining the blood glucose level.

Protein metabolism

Protein metabolim, by contrast, can maintain the blood glucose level. Amino acids are deaminated, thereby yielding ammonia (which is converted in the ornithine cycle to form urea). The carbon skeletons which remain are either glucogenic, forming pyruvate which can be converted to glucose, or, in lesser quantities, ketogenic, forming the ketone bodies (acetoacetic acid, beta-hydroxybutyrate and acetone).
When there is very decreased glycolysis, as in carbohydrate starvation, the citric acid cycles slows down because oxaloacetate, which combines with acetyl coenzyme A, is not regenerated in sufficient quantities. Although there is an alternative direct path for the production of oxaloacetate by carboxylation of pyruvate, the lack of consumption of acetyl-CoA, from fatty acid degradation and from the ketogenic carbon skeletons of protein, results in the accumulation of ketone bodies; of these, beta-hydroxybutyrate can quite easily be measured in the blood.

The problem for the body in starvation/catabolism is that the carbohydrate stores - as circulating glucose or glycogen - are depleted well within 24 hours. Metabolism must, therefore, switch to the high energy yielding fat, and gluconeogenesis has to occur from amino acid metabolism.

_The role of insulin and glucagon_

Insulin regulates glucose metabolism and also controls the anabolism of both lipids and amino acids, whereas glucagon, broadly speaking, exerts the opposite effect. Insulin has a dual mechanism for lowering of the blood glucose level. First, it increases glucose uptake in the peripheral tissues, particularly striated muscle and adipose tissue; and, second, it decreases the hepatic glucose output. Therefore, when insulin is present, glucose enters cells readily from the extracellular fluid and becomes available for intracellular metabolism or fatty acid or amino acid synthesis.

The biological half-life of insulin in the blood is of the order of 2-5 minutes. However, its biological action may persist for longer and reactive hypoglycaemia can occasionally develop when glucose infusions are stopped.

In the liver, insulin decreases gluconeogenesis and glycogenolysis and encourages glycogen synthesis. Therefore, when insulin is deficient, gluconeogenesis and glycogenolysis increase with an output of glucose from the liver into the extracellular fluid. The blood glucose level further builds up, as the glucose is inhibited from entering the tissues in the absence of insulin, and glycosuria may ensue. As the glucose cannot be metabolized, ketosis follows fatty acid breakdown and thus is seen both in starvation and, more severely, in diabetics.

Insulin promotes triglyceride synthesis in the adipose tissue cells. As lipase activity is normally inhibited by the presence of insulin, the consequence of insulin deficiency is the occurrence of unopposed lipolysis. For these reasons, insulin should be added to total parenteral nutrition regimens to inhibit catabolism.

_The metabolic response to trauma_

The metabolic response to trauma consists of disordered carbohydrate metabolism with ketosis, negative nitrogen balance, increased oxygen consumption and salt and water retention. Various triggering mechanisms are responsible and include soft tissue trauma, haemorrhage, other types of fluid losses, severe illness, burns, sepsis, and both pain and psychological stress.
Mediation seems to be integrated in the hypothalamus with hormonal changes from the anterior and posterior pituitary, the adrenal cortex and medulla, the pancreas, thyroid and kidney. Humoral mechanisms, involving kallikrein and prostaglandins, are also instrumental in the initiation of the response. The metabolic phenomena can be blocked during surgery, either in part or wholly, by combinations of epidural or spinal analgesia, barbiturates, morphine, neuroleptanalgesia and adrenergic blocking agents.

**Metabolic changes in stress**

The interaction of catecholamines, growth hormone, cortisol and glucagon together with diminished insulin secretion and increased resistance to its activity, cause hyperglycaemia and inhibit both the intracellular transfer and metabolism of glucose, as indicated earlier.

Mineralocorticoid secretion and altered renal perfusion suggest that the primary disturbance at cell membrane level consists of inhibition of the sodium pump. This would explain the intracellular sodium retention with extracellular hyponatraemia, the simultaneous opposite effects on potassium, and the consequent shifts in water. The potassium losses are also exacerbated by the increase in nitrogen secretion.

Evidence for a single 'biochemical lesion' in this area of intermediary metabolism has been demonstrated by its successful inhibition by means of glucose, potassium, and insulin regimens.

**Feeding the otolaryngological patient**

The vast majority of otolaryngological patients do not require extra feeding as they are neither starved beforehand nor do they get sufficient stress response. However, those patients with upper digestive tract carcinoma and/or dysphagia often present with fasting-adapted starvation. Most of these patients do not have an ileus and can be fed before surgery either by sip feeding or by continuous or bolus administration by way of a tube which bypasses the lesion.

Tube feeding may be through a nasal or orogastric narrow bore tube, if either of these can be tolerated, or by way of a jejunostomy. A homogenized diet, which can contain useful roughage and microorganisms as well as the essential food elements cannot pass through very narrow bore tubes. If a small bore tube is passed, then only a liquid diet can be given. Under these circumstances, it is usual for a proprietary synthetic element diet to be used. These preparations contain all food principals, including trace elements and vitamins, usually in such a concentration that they supply 1 cal/mL. These solutions are thus hyperosmolar and may cause purgation; to avoid this problem, it is advisable initially to dilute the solutions to half strength. Proprietary elemental diets are obtainable in lactose- and gluten-free forms, such as Ensure, or a milk-based diet such as Clinifeed. Supplementation by other proprietary preparations, such as Complan (carbohydrate and protein) or Hycal (carbohydrate only), is possible. Milk, eggs and natural orange juice are also frequently employed as supplements to the diet.
Total parenteral nutrition

It may be easier in some of these patients to begin nutrition with an intravenous regimen, and if ileus follows surgery then it becomes mandatory. Some authorities recommend the provision of all nutrition, water and electrolyte requirements in a 3-litre bag for 24-hour administration. This may be possible once a stable situation has been achieved, but acutely ill patients require more frequent evaluation and alteration of their regimens.

The requirements for the common electrolyte substances may be determined on a daily basis by plasma estimation and, where necessary, urinary excretion measurements. The requirement for potassium is likely to be higher than normal as its excretion is accelerated if there is excessive nitrogen excretion. Other than this, the necessary supplies of these substances are covered in the total parenteral nutrition regimen.

It remains axiomatic from the biochemical information given earlier that a positive nitrogen balance is not possible if insufficient non-protein calories are given. The ratio of non-protein calories to nitrogen should be of the order of 200 cal/g.

Obtaining circulatory access

The choice lies between peripheral and central venous administration. If, for any reason, venous access is impossible then nutrition can be administered intra-abdominally after the insertion of a peritoneal dialysis catheter, and the amino acid, fat or carbohydrate solutions will subsequently be absorbed through the peritoneum.

As ill patients may require other non-nutritional fluids, the carbohydrate and amino acid solutions tend to be hypertonic in order that the maximum amount of nutrient may be administered with the minimum amount of fluid. The advantage of central venous cannulation is that these hypertonic fluids are rapidly diluted in the large blood vessels, and thus a central line is the commonest method of administering total parenteral nutrition.

A radiopaque catheter is inserted so that its tip lies in the superior vena cava or right atrium. Transcutaneous access is achieved by way of the basilic vein in the antecubital fossa or by way of either the internal jugular vein or the subclavian vein.

Central venous catheterization may be unnecessary in patients who are on enteral feeding but who are as yet unable to tolerate total enteral nutrition. These individuals can have their diet supplemented by peripheral venous feeding using combinations of fat with either amino acid or glucose solutions, so that the mixture delivered into the vein is essentially isotonic.

Assessing nutrient requirements

Caring for the metabolically compromised patient requires: first, the treatment of the stress component, such as pain, haemorrhage, tissue necrosis and sepsis, as mitigation in this way will diminish the hormonal and humoral triggering of the catabolic stress response; second, assessments to be made of the dietary components which need to be administered, including electrolytes, trace elements and vitamins.
Assessment techniques

The proteins can be separated into visceral and somatic compartments. The visceral compartment consists of all the secretory proteins which play an important role in the survival of the organism. Included here are serum albumin, transferrin and cell-mediated immunocompetence proteins.

The search for protein nutrition markers among these secretory proteins has been protracted. The ideal marker would be affected promptly by any alteration in nutritional status; it would be unaffected by catabolism and non-nutritional therapies; and it would have a low extracellular fluid concentration and a short half-life. Albumin does not fulfill these requirements and measurements of transferrin have not proved useful. More recently, retinol binding protein and thyroxine binding pre-albumin have been assessed in this respect, but have also not proved useful.

The somatic protein compartment is muscle - the so-called 'lean body mass'. Estimation of 24-hour urea nitrogen excretion reflects the status of the metabolism of the lean body mass, that is an increased nitrogen excretion indicates that protein catabolism is occurring.

The 24-hour creatinine excretion is proportional to somatic muscle mass in normal individuals. However, an increased excretion may occur when there is heightened protein catabolism in the presence of a depleted lean body mass and its measurement is inconclusive.

Even the standard anthropomorphic techniques of nutritional assessment used for the non-critically ill are less reliable. Arm circumference, triceps skin fold and body weight may all be affected by fluid shifts as a result of 'third spacing' in the extracellular compartment of oedema fluid.

In the final analysis, reliance has to be placed on clinical judgement of the status of the lean tissue mass, and on common sense. The crude, but nevertheless useful, tool of 24-hour urine urea nitrogen determination and comparison with the 24-hour intake of nitrogen, is probably the only practical method of deciding whether lean tissue anabolism or catabolism is taking place.

Complications associated with total parenteral nutrition

The insertion of central lines is beset with a number of problems. Perforation of the pleura and pneumothorax has been described both with the internal jugular and subclavian approach. Additionally, air embolism can theoretically occur during insertion of the cannula. This may be avoided by ensuring that the patient is in a head-down tilt during the procedure.

Thrombosis and phlebitis are risks more commonly associated with peripheral infusion, and they may be avoided by using solutions which are not hypertonic in combination with regular changing of the cannulation site. Long lines from the antecubital fossa produce the biggest risk of venous thrombosis.
Infection

Infection may arise for a number of reasons attributable to faulty aseptic technique:

1. during insertion
2. during subsequent dressings of the insertion site
3. when changing bottles, bags or lines
4. contamination of the infusate during manufacture or when additives are given.

More recently, subcutaneous tunnelling of the central catheter has been advocated. However, unless strict aseptic technique is adhered to during this procedure, this method will not reduce the risk of infection. The use of this technique also assumes that central line infections occur by contamination of the wound and spread down the outside of the catheter. In reality, it is almost certain that catheters are infected more often by blood-borne organisms.

Ideally, there should be a specific line for intravenous feeding only. However, in the critically ill patient this may not be possible. Therefore, regular handwashing and strict adherence to written policies/procedures is of vital importance. The practice in the author's unit is to use 70 cm lines for subclavian central venous insertion. The advantage is that the excess line outside the patient allows the points of contact with taps, etc, to be distal to the wound, which is dressed in the prescribed manner every 48 hours.

Monitoring of white cell count daily and of temperature, pulse and respiration 4-hourly may give a clue to infection of the central venous line.

Other complications

Other complications relate to circulatory overload with fluid and to glycaemic instability. Hyperosmolar (hyperglycaemic) non-ketotic syndrome and coma may ensue, as may excessive fluid losses with glycosuria. Regular blood glucose measurements at the bedside, with a chemical 'stick test' read colorimetrically, and urine analysis avoid this complication, provided that the insulin administration is adjusted accordingly. Insulin may be given mixed with the substrates in the infusion solutions, in which case allowances have to be made for binding of the insulin to the containers; or, alternatively, in concentrated form by way of a syringe pump through a separate line. The disadvantage of the latter occurs when nutrients are discontinued, and care must be taken to avoid disastrous hypoglycaemia.

Evaluation of feeding regimens

As has been indicated earlier, evaluation of feeding regimens may prove extremely difficult in practice. Measurement of beta-hydroxybutyrate in plasma is now fairly easy and levels in excess of 0.3 mmol/L indicate that non-carbohydrate metabolism (catabolism) is occurring. Under these circumstances, the blood sugar level is of no help in assessing nutritional status but is monitored to help promote glycaemic stability. Beta-hydroxybutyrate
measurements can be combined with 24-hour nitrogen balance to assess the status of the lean body mass.

**Weaning once total parenteral nutrition is discontinued**

A patient recovering from serious illness, who has been receiving long-term total parenteral nutrition, will require a gentle introduction to oral or nasogastric feeds. The onset of gut sounds and diminution of nasogastric aspiration to a minimum indicate that water may be introduced initially. If this is absorbed then nutrient substances can be introduced. As the gut which is recovering from ileus is very often lactose intolerant, it is usually wise to introduce a lactose-free synthetic elemental diet, such as Ensure, before proceeding by means of milk to a homogenized diet, whereupon the nasogastric tube can be removed.

The gastrointestinal hormones will have been in abeyance during total parenteral nutrition, and the hepatic enzymes will have adapted to the total parenteral nutritional regimen, arriving in the systemic circulation, rather than with partially processed nutrients which arrive by way of the portal vein. Consequently, as re-adaptation will be necessary, all feeding should be introduced slowly and intravenous feeding should be concomitantly reduced.

**Haematological considerations**

**Anaemia**

Anaemia has many causes but in the final analysis can be the result of either abnormal red cell production or excessive loss of blood. It may be congenital or acquired, and primary or secondary.

From a clinical point of view, the important consideration is when and by what method correction should be carried out. Clearly, if a patient is bleeding and anaemic, correction needs to be carried out by transfusion. However, for more chronic forms, medical measures will suffice. The signs of absent compensation would be breathlessness and tiredness, together with palpitations, dyspnoea on exertion, and possibly effort angina with an ejection systolic murmur.

**Oxygen transport**

Haemoglobin is not the most important factor in oxygen transport. A full understanding of oxygen transport is important for the clinician because its consideration has to be balanced against the quality of blood available for transfusion in the surgical situation.
The oxygen transported to the tissues in unit time is a function of the haemoglobin concentration, oxygen saturation of the haemoglobin in arterial blood, and the cardiac output.

\[ O_2 \text{ availability} = K \times (Hb \times SaO_2 \times Qt) \]

where

- \( Hb \) = haemoglobin level in g/100 mL whole blood
- \( SaO_2 \) = percentage oxygen saturation of the haemoglobin in arterial blood
- \( Qt \) = cardiac output in mL/minute
- \( K \) = constant, derived from the oxygen capacity of haemoglobin and appropriate decimal corrections, with a resulting value of 0.000139.

Considering some normal values:

\[ O_2 \text{ availability} = K \times (14.5 \times 100 \times 5000) = 1000 \text{ mL/minute} \]

As basal oxygen consumption is of the order of 250 mL/minute there is still a considerable reserve under normal conditions.

The relative contributions of the foregoing factors can be placed in perspective as follows:

1. each litre change in cardiac output, when the haemoglobin level is normal and there is full saturation, accounts for a change of 200 mL/minute in oxygen availability

2. each gram change in haemoglobin, when the cardiac output is 5 L/minute and there is full saturation, accounts for a change of 69 mL/minute in oxygen availability

3. each 1% change in saturation, when the cardiac output is 5 L/minute and the haemoglobin level is normal, accounts for 10 mL/minute in oxygen availability.

The impact of anaemia on a patient, especially for routine surgery, must be weighed against the disadvantages of delaying surgery until medical correction can be achieved. In the emergency situation, the disadvantage of anaemia has to be offset against the disadvantages of whole blood available for transfusion.

The quality of blood for transfusion

It is unfortunate fact that blood is usually only made available for transfusion late in its shelf life of 3 weeks, by which time it contains clots and microaggregates of dead white cells and effete red cells. Depending on the anticoagulant used - the blood may be acid in the case of acid-citrate - dextrose blood and have a high serum potassium - it may be infected, incompatible and so on. In addition, with the modern presentation as saline-adenine-glucose-mannitol blood, the plasma - which is perhaps the most important component of blood - will have been depleted. It is not too cynical to state that if blood were classified as a medicine, it would probably not pass the stringent regulations of the various safety organizations around the world. As an acute replacement for lost blood volume, plasma protein fraction (human albumin) is much safer.
If, in spite of these considerations, a blood transfusion is still deemed necessary, it is mandatory in present times that the blood be filtered with a device capable of removing particulate matter down to 20 μm because, if it is not filtered beforehand, the lungs will do so subsequently. Debris from transfused blood in the lungs causes a humoral response and pneumonitis, resulting in shunting or venous admixture, and this is an avoidable additional risk factors.

**Sickle-cell trait and disease**

Normal adult haemoglobin is given the designation HbA. In the heterozygous condition of sickle-cell trait, there is a mixture of HbA with HbS, whereas in the homozygous condition of sickle-cell disease, the haemoglobin is all HbS, and the sufferer has the designation HbSS. Equally serious are the combination of the sickle-cell gene with HbC, giving the sickle-cell C disease (HbSC), or the combination with thalassaemia, giving sickle-cell thalassaemia (HbS-thal).

Sickling of the red cells occurs with apparent crystallization of the abnormal haemoglobin at low oxygen tension and pH. There is aggregation of abnormal cells, which causes microinfarctions in the tissues, and the disease manifestations depend on the distribution of the infarcts.

Sufferers from this disease tend to be coloured patients of African or West Indian origin or from the Mediterranean region.

Homozygous patients will usually give a history of sickling crises with haemolytic anaemia, but the heterozygous patients may be found by screening with a bedside test such as the 'Sickledex'. If this test is positive and the patient will have a significant clinical problem as the positive test suggests the heterozygous condition. However, if there is time, the patient should be genotyped and a blood film examined. The care required by these patients is no more meticulous than that offered to any other. Therefore, even in countries with large populations of the type just described, management of the condition is passive rather than active. Even the use of tourniquets in orthopaedic procedures is not proscribed in these countries, although their use would not appear to be justifiable.

**Bleeding disorders**

Clinical medicine in the UK is no longer carried out without access to consultant haematologists. However, it is clearly the duty of the ordinary clinician to be able to differentiate between large diagnostic groups, for example, to determine whether a bleeding tendency is lifelong or whether there is a family history of such a condition.

Purpura and excessive bleeding from superficial cuts and mucosal haemorrhages suggest a platelet disorder, whereas the occurrence of deep bruising, bleeding into joints and haematuria, or delayed wound healing after previous surgery, would be indicative of coagulation factor deficiencies. In the laboratory, the bleeding time and platelet count can detect quantitative and qualitative platelet deficiencies, while a prothrombin time is sensitive to defects of the coagulation factors II, V, VII and X and is also prolonged in liver failure, vitamin K deficiency and during the use of anticoagulants. The activated partial
thromboplastin time indicates a deficiency of factors V, VIII, IX and X and is used to detect haemophilia (factor VIII deficiency) and Christmas disease (factor IX deficiency).

In the condition of disseminated intravascular coagulation, there is an increased level of fibrin degradation products, and both fibrinogen and platelet levels are lowered. At the present time, disseminated intravascular coagulation is treated with antibiotics for the triggering sepsis and by the transfusion of fresh frozen plasma and platelets, rather than by trying to inhibit the process of accelerated intravascular coagulation with heparin. None of these conditions should be treated without reference to, and without the close cooperation of, the haematologist.

**Prophylaxis of deep vein thrombosis**

Intra- and postoperative prophylaxis of deep vein thrombosis is carried out not only in the wards after surgery but also in the intensive care unit. It might be considered an area of controversial discussion as many disciplines are involved. Moreover, there is a shifting ground of fashion in the management of these problems.

The position used to be less problematical in the case of women taking the contraceptive pill as it had been considered beneficial for the woman to simply stop therapy. However, in the light of experience this assumption has been questioned and many have now ceased this practice. Nevertheless, a history of obesity and previous deep vein thrombosis would indicate active prophylaxis. On the other hand, a decision is more difficult when, for example, a patient has varicose veins unrelated to the current surgical/medical problem, and treatment must depend on the reasoned opinion of the individual clinician. The author has found the advice of Browse (1977), in his excellent article: 'What Should I Do about Deep Vein Thrombosis and Pulmonary Embolism' to be of great use in this respect. Crandon et al (1980) have also made an attempt to identify high-risk patients for selective prophylactic anticoagulant therapy. The present author's current practice, once a patient is in the intensive unit, is to use subcutaneous heparin therapy (5000 units, 8-hourly) and anti-embolism stockings on the basis that both these measures have been shown to be effective in the prophylaxis of deep vein thrombosis. Heparin is the anticoagulant of choice as it can be easily reversed with protamine.

**Haemolytic transfusion reactions**

Haemolytic transfusion reactions are usually the result of ABO incompatibility. More rarely, IgM antibodies acting against other blood group antigens may be the cause. Invariably, the fault lies with the transfusor rather than with the cross-matching process. In the conscious patient there is pain along the vein, flushing, palpitations and headaches, with a feeling of chest constriction and pain in the lumbar region. The full picture is rather anaphylactoid in nature, with tachycardia, hypotension, urticaria, peripheral circulatory collapse, rigors and pyrexia. If the patient is anaesthetized or sedated during this period in the intensive care unit, then much of this picture may be abolished. Tachycardia, hypotension and rigors with increased blood loss from drains or into dressings may be suggestive. The diagnosis can be confirmed by inspection of a blood film, a positive direct antiglobulin test and free haemoglobinrenaemia.
The treatment depends on the severity of the reaction. First, the blood transfusion is stopped, whereupon adrenaline, antihistamines and steroids are administered. Sodium bicarbonate is given if there is a metabolic acidosis; volume expanders are given if there is hypotension; a dopamine infusion is set up to promote renal blood flow; disseminated intravascular coagulation is looked for and is treated if it occurs. The patient may develop renal failure and this condition must be appropriately handled.

Management of the collapsed otolaryngological patient

Otolaryngological surgery is carried out on age groups at both ends of the spectrum. The commonest problem with the younger age groups is postoperative bleeding which is manifestly obvious to all concerned and does not pose a problem in differential diagnosis. However, in the case of adults, particularly the elderly, a collapse with hypotension, pallor and a change in pulse rate (tachycardia or bradycardia) can occur without postoperative haemorrhage, and a differential diagnosis needs therefore to be made.

In general surgery, a common cause of postoperative collapse is endotoxic shock and/or sepsicaemia as a consequence of complications in which parts of the gut have been breached. This situation would be rare in otolaryngologic practice except when combined laryngopharyngeal and oesophageal surgery has been carried out with mobilization of stomach or large bowel up through the chest. Consequently, endotoxic shock is an infrequent cause of collapse in an otolaryngological patient, but it should nevertheless be borne in mind, as the condition can follow instrumentation of the urethra if the patient requires catheterization following postoperative retention of urine. Additionally, any patient, irrespective of surgical procedure, can acquire an ileus as part of the humoral response to trauma and may, as a consequence, absorb bacterial endotoxin from the gut.

Other possible causes of collapse commonly originate in the cardiovascular system, and coronary thrombosis and/or arrhythmias would be the commonest of these. Pulmonary embolism can also occur, and other less likely causes would be gallbladder colic, peptic perforation, acute pancreatitis or dissection of an aortic aneurysm.

Faced with a hypotensive shut-down and an oliguric patient, how should the clinician arrive rapidly at the diagnosis? It is imperative that there be a high index of suspicion that the cause of collapse is a surgical one before a medical cause of collapse is espoused.

Previous history

The type of surgery performed and the possibility of bleeding, together with significant events in the previous history, such as ischaemic heart disease, form the background on which to build the pathway to diagnosis.

The timing of the collapse in relation to the surgical intervention is important. Collapse from endotoxic shock can follow within a very short time of, for example, instrumentation of the urethra, but it can also occur at any time during the postoperative period. A rigor may be a presenting feature in sepsicaemic shock and would indicate the diagnosis from the outset. Primary surgical haemorrhage usually manifests itself well within the first 12 hours. Fortunately, in otolaryngological practice, haemorrhage is usually apparent from the beginning
as a drainage loss. However, in the rare event of haemorrhage to the exterior not being immediately apparent, the most obvious manifestation, distinguishes it from other causes of collapse/shock, is the waxen appearance of the lips and mucous membranes of the gums. In both myocardial infarction and pulmonary embolism the features tend to be blue/grey. However, the pulmonary embolism case is typically marked by agitation, while the patient with severe myocardial infarction and shock has a resigned attitude.

Current history

One of the most helpful indices, on taking a history from the patient, is the character and distribution of any pain. The pain of myocardial infarction is characteristically central, and crushing or vice-like, and may radiate to either arm or jaw. In pulmonary embolism there may be no pain at all or, if it does occur, it will be lateralized and pleuritic in nature. In the case of sepsis or haemorrhage, chest pain is invariably absent.

The pulse rate is rapid in haemorrhagic shock, usually rapid in septicaemic shock and may be slow, rapid or unchanged in both myocardial infarction and pulmonary embolism.

The temperature may be raised in septicaemic shock but this is often not the case. A small rise in temperature does occur in myocardial infarction.

The jugular venous pressure is unequivocally lowered in haemorrhagic shock and may be raised in the other three conditions. Characteristically, however, the patient with pulmonary embolism can lie flat, whereas in severe myocardial infarction the patient cannot. In both the latter conditions, the patients may have a cough: in myocardial infarction the patient may cough up frothy pink sputum, while in pulmonary embolism the sputum will be normal in consistency but blood stained. Auscultation in cardiogenic shock may reveal crepitations at the lung bases and a third heart sound or a gallop. Initially, in pulmonary embolism, there will be no chest signs, although bronchial breathing may develop later if there is a pulmonary infection. However, a right sternal edge fourth heart sound may be heard.

Haemorrhagic shock itself is not usually associated with sputum production. A patient with septicaemic shock, however, may have infected-looking sputum if the septicaemia originates from the lungs.

A chest X-ray which shows bats-wing pulmonary oedema would be an indication of left ventricular failure associated with cardiogenic shock. A picture of widespread patchy infiltration may be consistent with the 'shock lung' syndrome associated with septicaemic shock or, if there is a consolidated patch with an air bronchogram, this may be consistent with a focus of lung sepsis associated with septicaemia. The chest X-ray in haemorrhagic shock will probably be normal, and in pulmonary embolism may be unaffected unless a pulmonary infarction occurs later on. Postoperative collapse may result from aspiration of acid gastric contents, although this is rare. In the event of its occurring, the patient will be cyanosed and may have bronchospasm and a tachycardia, and the chest X-ray will show fluffy blotched areas of consolidation.

An erect abdominal or lateral decubitus X-ray may show free gas if septicaemia is associated with gut perforation, or it may show pelvic collections of gas if gas-producing
anaerobic abscesses are present. Gut fluid levels with ileus may be present in all the named conditions.

The electrocardiogram (ECG) is an important means of investigation to assist in these cases. However, it is important to recognize that generalized ischaemic ECG changes can occur when the hypotension is not in fact caused by myocardial infarction but is merely a result of myocardial hypoxaemia.

In myocardial infarction, ST segment changes occur initially with elevation in the leads which overlie the damaged areas of myocardium. If there is full thickness myocardial infarction, then Q waves develop in the relevant leads. It is usually assumed that a pathological Q wave must be more than 0.4 second in duration and more than 25% of the succeeding R wave. It is important to realize that Q waves do not appear until muscle necrosis occurs and thus may be delayed for some hours.

If the infarction is anterior, then the most affected leads are I, AVL and the chest leads; in anteroseptal infarction, the early chest leads show the maximal changes; if the infarction is anterolateral, then V4 to V6 demonstrate the main effects; and, on some occasions, extensive infarction may be manifest in all the anterior chest leads. In inferior infarction, the main changes are in leads II, III and AVL.

In pulmonary embolism, the ECG changes are characteristic. There is right axis deviation and, with the onset of right ventricular strain, the development of an S wave in lead I, a Q with an inverted T wave in lead III, and T wave inversion in V1 to V3. There is a pronounced atrial wave (P pulmonale) and right bundle-branch block. In acid aspiration syndrome, the picture is not dissimilar from pulmonary embolism with marked right ventricular strain and P pulmonale; and more widespread ST changes may be associated with severe hypoxaemia.

**Enzyme changes**

A rise in the serum creatinine phosphokinase (CPK) begins to occur 6 hours after myocardial infarction and reaches a peak in about 24 hours, whereas in pulmonary embolism a small rise occurs. Another isoenzyme, CPK-MB, which is said to be specific for the myocardium, may prove invaluable in the future but is still not as totally specific as was hoped. Aspartate aminotransferase (SGOT) rises after 12-24 hours, while lactic dehydrogenase (LDH) rises after 72 hours. The white cell count is of little use in differential diagnosis. In septicaemic shock, it may not be significantly raised above that found in other postoperative patients.

**Direct central venous pressure measurements**

A central venous pressure line will be a further aid in diagnosis. The catheter is inserted via the subclavian vein, usually approached infraclavicularly, and connected to a saline manometer zeroed at the level of the right atrium. This will certainly enable the accurate detection of the low central venous pressure of haemorrhagic shock and thereafter enable monitoring of the response to transfusion. The central venous pressure is often lowered
in septicaemic shock as well. By contrast, it will be raised in pulmonary embolism and is usually raised in cardiogenic shock.

**First line treatment**

If the patient appears to have a myocardial infarction, he will be taken over by the appropriate team. However, initial management should consist of pain relief with diamorphine which is a vasodilator in addition to being a narcotic analgesic. In pulmonary embolism, full heparization should be commenced immediately.

In haemorrhagic shock, initial resuscitation should be by colloidal infusions, such as Haemaccel or Hespan, while awaiting cross-matched blood and, at the same time, organizing surgical intervention.

If endogenic shock is a possible diagnosis, management is aimed at initial definitive diagnosis, very early medical treatment and delayed surgical intervention, if indicated.

Initially, two samples of blood should be drawn under sterile conditions and should be inoculated into culture medium before being sent for incubation, whereupon the administering of antibiotics must be commenced immediately, with metronidazole 1 g intravenously, gentamicin 120 mg and ampicillin 1 g. This regimen will cover Gram-negative rods, staphylococci and haemolytic streptococci. Fluid therapy with Haemaccel or HPPF should be started in order to raise central venous pressure and improve renal perfusion. Steroids in the form of methylprednisolone 30 mg/kg should be given initially and every 6 hours thereafter, for 24-48 hours, to combat the effect of endotoxin which will persist in the circulation even following the destruction of the offending bacteria.

The bladder should be catheterized and, if there is oliguria, then dopamine should be administered in 'nephrogenic' doses, that is 3-5 μg/kg per h. Any necessary surgery to rectify soiling from gut spillage or to secure leaks as appropriate, must then be contemplated. All these patients are prone to develop acute renal failure unless renal perfusion is ensured. While diuretics are appropriate in severe myocardial infarction, the use of dopamine, as described, as a prophylactic measure, even in the absence of oliguria, is advisable.

**Other causes**

The pain of a perforated peptic ulcer has a characteristic distribution in the upper central hypochondrium, together with gas under the diaphragm on X-ray. However, it is unusual for these patients to be grossly shocked.

Pain and, indeed, septicaemia may follow gallbladder colic and cholecystitis. The incidence of gallbladder symptoms should be sought from the patient, and stones may be present on the abdominal X-ray. Pain radiating through to the back would assist in establishing the gallbladder as a source of problem if other characteristic features of the main diagnoses are not present. Similar pain could also be caused by acute dissection of the thoracic aorta. Theoretically, pericarditis might be a cause of the pain but, in this case, the patient should also exhibit a pericardial rub on auscultation and possibly the signs of cardiac tamponade. Acute pancreatitis can be excluded by the finding of normal serum amylase levels.
General supportive measures

In the circumstances under discussion, oxygen therapy will benefit all groups. If the patient is in severe pain then it is cruel to withhold analgesics, even if final diagnosis might not yet have been made. Short-acting drugs, such as diamorphine or fentanyl, can be used at this stage, and should be given in small doses intravenously. Patients in extremis from the causes discussed pass rapidly into a situation of respiratory failure and should be intubated and undergo intermittent positive pressure ventilation. All of these patients should be transferred to the intensive care unit.

Haemorrhage and disseminated intravascular coagulation

If it is considered that haemorrhage is in fact associated with disseminated intravascular coagulation, the occurrence of this syndrome should be confirmed in the laboratory, and management should be as indicated earlier.

Extreme emergency situations

When the situation is rapidly deteriorating and the differential diagnosis between cardiac infarction, pulmonary embolism and septicaemic shock is unclear, then the regimen outlined previously for the immediate treatment of septicaemic shock must be initiated. Such a step will not further harm the patient with an infarct or embolism, but will produce dramatic improvement in the septicaemic patient. The longer the delay the worse the outlook in the case of the latter condition, with the onset of multiorgan microcirculatory failure and its consequences - shock lung syndrome, acute respiratory distress syndrome, acute renal failure, and so on. Many clinicians and their patients have, in the past, been thankful that this approach has been adopted.