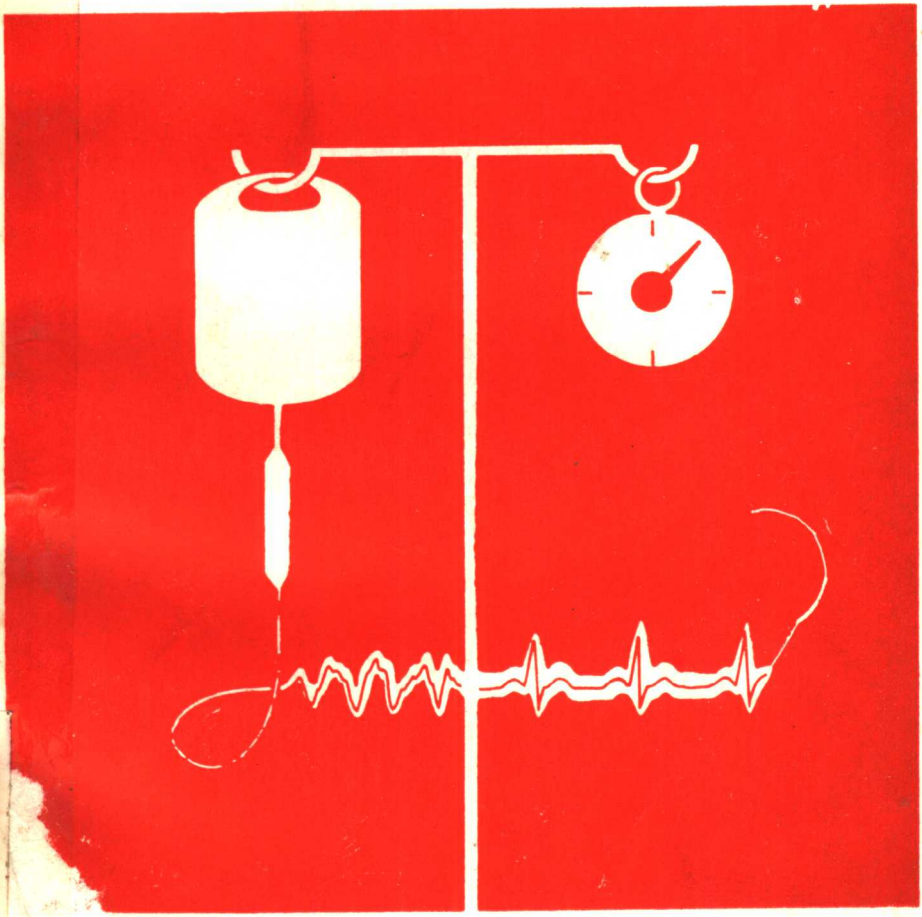
D. W. Yates and A. D. Redmond

Lecture Notes on Accident and Emergency Medicine



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LECTURE NOTES ON

Accident and Emergency Medicine

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Preface

Our aim has been to cover all aspects of the work of a doctor in a busy accident and emergency department. Much of the text should be of interest to medical students during their clinical studies, but we hope that the book will be of special value to doctors studying for postgraduate diplomas.

Clearly some restriction must be placed on our definition of accident and emergency work unless we are to be overwhelmed, both here as authors and in our departments as casualty officers. These lecture notes have not been conceived as comprehensive treatises on the management of the many topics discussed — for only some aspects of most diseases and injuries are germane to the accident and emergency department. But it is precisely these aspects which are often inadequately covered in the standard texts. This book is based on our daily experience of the plethora of conditions presenting to what is inevitably an unstructured environment.

Sections devoted to 'Urgent action' are designed to facilitate the book's use during emergencies. The remaining text continues this rather didactic format but is supported by background material which attempts to justify our dogma. It is, however, not always enough to state that the advice given here has worked in practice (but it has!). Emergency medicine, like all other branches of medicine, must be based on sound scientific principles. A significant recent development has been the acceptance that emergencies are better treated by the experienced than used to gain experience. Future developments must ensure that the scientific method is woven into the fabric of the department.

The academic base for accident and emergency medicine in Manchester has been created by the foresight and hard work of three men. We wish to record our thanks to Professor M. H. Irving, Dr G. S. Laing and Professor H. B. Stoner who have given so much to the specialty in its early days and who have supported us during the preparation of this book. The doctors, nurses, clerical staff and patients of Salford and Stockport have

also given us invaluable help. Their encouragement has been greatly appreciated. Jackie Fortin typed some of the preparatory work and Julie Rostron exercised great skill and patience in compiling the manuscipt.

Veronica Yates responded to our vague requests for pictures with precise illustrations, and our publishers displayed remarkable tolerance during a long gestation.

August 1984

D.W. Yates
A. D. Redmond

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Chapter 1
The Pathophysiology of Injury

In general terms, the metabolic and endocrine responses to accidental trauma are far greater than those which follow elective surgery. For example, a fracture of the femoral neck will produce greater changes in metabolic fuel utilization than a total hip replacement.

The tendency to overlook the importance of these events is reinforced by the ability of young, previously fit people to recover adequately from modest injury without any thought having been given to metabolic requirements.

Improved techniques of resuscitation now increase the likelihood of immediate survival after very major injury. It cannot be assumed that all the consequent metabolic responses have evolved by a process of natural selection. There is nothing natural about survival after major trauma.

To manage these patients effectively, it is essential to have a good working knowledge of the associated pathophysiological changes and to understand how their manipulation might be achieved to improve prognosis.

Apprehension often precedes injury. This will evoke discharges from the hypothalamus, thus increasing adrenal medullary activity and inhibiting some baroreceptor reflexes.

Muscle blood flow and heart rate will increase, improving oxygenation of vital tissues. Muscle tone will increase and limbs will assume a protective attitude.

These beneficial pre-injury changes may be modified by drugs, the most important of which is alcohol. All sedatives reduce muscle tone. This is particularly relevant to the pathogenesis of brain injury. Uncontrolled head movement increases, subjecting the brain to greater shearing forces.

Other drugs may be important. For example, β-adrenergic blockade will reduce the response to sympathetic discharge.

The injury, once sustained, will provoke local and then systemic responses, mediated through the cardiovascular and

neuroendocrine systems and will involve major changes in metabolism.

Local effects

Loss of function. Division of a ligament, a tendon or a nerve will result in appropriate loss of function. Some localized injuries (e.g. penetration of pleura, intestine and bladder) can have widespread effects due to loss of their function as 'limiting membranes'. Divided blood vessels may cause distal ischaemia and systemic hypovolaemia.

Inflammation. A contused wound or a crushing injury will provoke an inflammatory response. After transient reflex vasoconstriction of the small vessels, histamine, 5-hydroxytryptamine, bradykinin and prostaglandins are released from damaged cells and produce local vasodilatation and increased vascular permeability. This is reinforced by a local axonal reflex (the triple response). The integrity of the nerve up to the dorsal root ganglion is essential. Division of the nerve proximal to the ganglion does not abolish the reflex. This forms the basis for the histamine test, used to assess the level of a brachial plexus lesion (p.87). Prostaglandins produce local hyperalgesia by increasing the sensitivity of sensory nerve fibres. The extent of their release appears to be related to the duration of local stimulation as well as to its intensity. Inhibition of prostaglandin synthesis by aspirin-like drugs forms the basis of some of the therapeutic actions of these agents.

Associated with this local vasodilatation, the endothelium becomes more permeable. Exudates form at wound surfaces and the extracellular space increases. White blood cells migrate into the area, along with mucopolysaccharides, heralding the onset of the phagocytic and rebuilding phases.

Systemic effects

Afferent stimuli from the damaged tissue and responses from other receptors secondarily affected by the injury (e.g. volume receptors) provoke a complex response in the central nervous system.

Hypovolaemia produces reflex tachycardia, a contraction of the venous bed and selective peripheral vasoconstriction. When the capillary environment becomes acidotic, the oxyhaemoglobin

dissociation curve moves to the right so that more oxygen is unloaded at the same partial pressure.

These haemodynamic and chemical changes tend to maintain oxygen delivery to vital tissues. Precapillary and arteriolar vasoconstriction reduce fluid shift from the plasma to the extracellular space. Haemodilution is initially beneficial — the reduced viscosity facilitates capillary blood flow. Oxygen transport actually increases as dilution occurs down to a haematocrit of 30, if normovolaemia is maintained. However, this simple relationship rarely obtains after trauma because of the influence of hypovolaemia.

If hypovolaemia persists, these homeostatic changes are unable to prevent local tissue hypoperfusion and there is a gradual loss of control of the microcirculation. Precapillary sphincters dilate as they appear to be more sensitive to lack of oxygen than are the post-capillary sphincters. The capillaries fill, but flow through them is sluggish. Fluid leaks into the extracellular space and red cell and platelet aggregates and fibrin microthombi further reduce the local circulation, compounding the hypoxia.

The sympathetic discharge provoked by hypovolaemia leads to a rise in plasma catecholamines. Plasma steroid concentrations increase directly with injury severity but after very severe injury low levels have been recorded. This is thought to be due to failure of perfusion of the adrenal cortex and indicates the need for more aggressive volume replacement in such patients. There is no convincing evidence at present that severely injured patients would benefit from the administration of systemic corticosteroids.

The main metabolic effects of adrenaline and steroids are to increase glycogenolysis and lipolysis and facilitate the conversion of protein to glucose by the liver (gluconeogenesis).

Insulin concentration is very low immediately after severe injury, possibly due to the high concentration of circulating adrenaline. Later, the insulin concentration rises disproportionately to the level of glucose and there is resistance to the normal effects of the hormone.

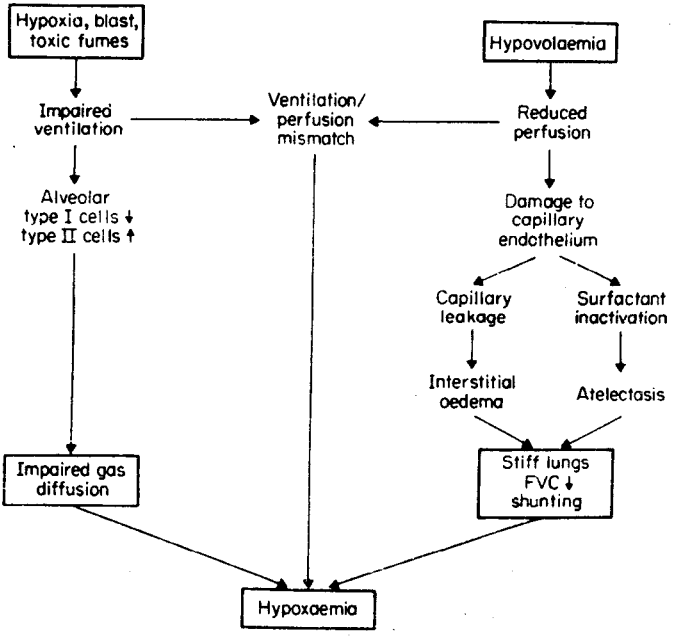
All the above changes tend to lead to high concentrations of glucose, to the mobilization of protein (with a reduction in its synthesis as well as an increase in its catabolism) and to the widespread availability of fat. Indeed, fat is used preferentially

after injury, despite high levels of glucose. The reason for this is unknown but recent work confirms that fat metabolism is obligatory after trauma and cannot be 'switched off' by the administration of parenteral glucose.

Special problems

The microcirculation of the intact *brain* responds to changes in carbon dioxide levels, but is protected from modest changes in blood pressure within the systolic range 80–200 mm Hg. After head injury, these responses may be impaired. Usually, CO_2 responsiveness is decreased and autoregulation is disturbed in some, but not all, parts of the brain. Hypoxic brain damage can follow either poor airway control or inadequate gas exchange.

Inadequate cerebral blood flow may also be due to systemic hypovolaemia or cardiogenic shock. It has been postulated that



Box 1.1 The pathophysiology of shock lung (adult respiratory distress syndrome).

Box 1.2 The injury severity score (ISS).
(1) Each injury is scored on the abbreviated injury scale (AIS) from one to five. Use the AIS-80 dictionary, available from — AAAM, 40 Second Avenue, Arlington Heights, Il. 60005, U.S.A.

e.g. One fractured rib	1
Compound tibial fracture	3
Lacerated liver	5

(2) Determine the highest scoring injury for each of the following body regions.

Head	Abdomen (and pelvic contents)
Face	Limbs (and bony pelvis)
Chest	Surface

(3) Using only the three highest scoring regions, square each AIS and add together.
(4) The resulting ISS will lie on a scale from 1 ($1^2 + 0 + 0$) to 75 ($5^2 + 5^2 + 5^2$). Similar scores, derived from patients with dissimilar injuries, indicate that it is possible to make broad comparisons of metabolic and systemic responses if a sufficiently large group of patients is examined. Comparisons cannot be made between individual patients. The ISS is primarily a research tool.

the hypothalamus is particularly sensitive to hypovolaemia. This may be an important element in the neurohormonal response to hypovolaemia but, clearly, hypothalamic activity and the competence of neurohumoral control will be adversely affected by very severe hypovolaemia and hypoxia.

Inflammatory changes within the *lungs* usually do not produce clinical manifestations for many hours, such is the reserve capacity of the healthy lung. However, deterioration of function at alveolar level may begin almost immediately after any injury. The downward trend to 'shock lung' can only be arrested if hypoxia and hypovolaemia are prevented (see Box 1.1).

Damaged lung tissue is far more sensitive to increases in pulmonary intravascular pressure than is normal lung. Pulmonary oedema will develop more readily in patients with pulmonary contusion. Injury also produces a discrepancy between the distri-

bution of respiratory gases and blood (ventilation/perfusion mismatch), causing hypoxaemia.

Established pulmonary complications in the multiply injured patient are more difficult to treat than to prevent and are discussed further on p.68.

Measuring the severity of injury

Well-controlled laboratory studies have provided a great deal of information about the complex pathophysiological responses to injury. Research in injured man is clearly limited by ethical considerations and also by the great variety of injuries sustained.

The recent development of the abbreviated injury scale and the injury severity score has removed some of these constraints (Box 1.2). Injured patients have been studied clinically and biochemically and it is clear that groups with similar scores behave, in general terms, in similar ways. The score is of no particular value in the assessment of a single patient. Heterogeneous groups of injured patients can, however, now be compared and this is already providing a valuable stimulus for the further study of the responses to trauma in man.

Chapter 2
Multiple Injuries

Urgent action (details pp.8–21)

If you suspect cardiac arrest turn to p.167. Otherwise:

(1) Secure the airway, but be gentle with the neck. If the gag reflex is depressed or absent intubate the trachea (p.207). Give oxygen.

(2) Protect the neck with a rigid collar.

(3) Stop external bleeding by direct pressure.

(4) Cover 'sucking' chest wounds with airtight dressings.

(5) Establish venous access — preferably at two sites — large-bore cannulae in large veins (p.10). Withdraw 20 ml blood for grouping, cross-matching and baseline glucose, electrolytes and haemoglobin. Start N saline infusion, then colloid.

(6) Take and record pulse and blood pressure frequently. Attach patient to cardiac monitor.

(7) Examine front and back of chest. Confirm lung expansion is equal and adequate and mediastinum is central (p.17). Insert a chest drain on clinical suspicion of pneumothorax (p.66). Consider cardiac tamponade (p.189).

(8) Cover exposed abdominal viscera with warm saline packs. Examine front, back and perineum for evidence of blunt and sharp impact (p.71).

(9) Search the face and scalp for significant injuries. Assess the level of consciousness and pupil response (p.25).

(10) Assess the stability of the pelvis (p.74).

(11) Confirm the presence of peripheral pulses in each limb and splint major limb fractures (p.78).

(12) Estimate arterial blood gases (p.210).

Establish the priorities for investigation and treatment. If these are not within your competence seek help at once.

Justification

The A & E department staff have specific, circumscribed responsibilities in the care of multiple injuries.

Emphasis must be placed on the prevention of further deterioration and the assessment and correction of reversible pathology. The airway has top priority. This most important principle of first aid is sometimes overlooked by doctors, either because it is assumed to be someone else's responsibility or because other problems claim their immediate attention. Nothing is more important than a patent airway (assuming the heart is beating!).

Correction of hypovolaemia should not be delayed until the late appearance of the classical signs of blood loss. Similarly, chest drains should be inserted earlier than is often taught. Detailed neurological and orthopaedic assessments are not essential parts of this first few minutes of immediate care.

Radiographs of the head, neck and trunk are valuable thereafter — particularly an erect chest film — but the doctor must be able to justify his request. The results must have implications for immediate *treatment* and not be only of long-term interest (see Table 2.1, p.21).

One person should be in overall charge whilst the patient is in the resuscitation room. He should be responsible for contemporary notes and charts and liaison with other hospital disciplines. Assistance from specialist colleagues should be requested at an early stage. Action taken by A & E staff in the first few minutes after the patient's arrival at hospital may be decisive in terms of prognosis. It must not be divisive in terms of relationships with other departments. It is not in the patient's best interests for one group of doctors to work in isolation from the many other groups who will be required to care for the multiply injured patient.

When more than one patient is admitted to the resuscitation area concurrently, it is essential that a medical and nursing team is attached to each patient. Small teams with specific responsibilities are safer and more effective than groups of staff moving from crisis to crisis.

Details of urgent action

Airway

Remove false teeth, vomitus and foreign bodies and assess the response to digital stimulation of the posterior pharyngeal wall ('gag reflex').

Even when there is a *normal gag reflex* sudden vomiting can

seriously threaten the airway and is almost a normal sequel to major trauma.

It is often impractical to nurse the patient on his side in the 'recovery' position during the invasive stage of resuscitation; careful and constant supervision of the airway is therefore essential.

A high-volume sucker should be immediately available and switched on.

Oxygen should be given in high concentrations via a face-mask; masks supplying only 24% or 28% oxygen should not be used.

When the *gag reflex is impaired or absent* insert an endotracheal tube (p.207). Clumsy attempts to clear the airway and intubate the trachea can cause local damage, oedema and bleeding, stimulate vagal reflexes producing profound bradycardia and cause laryngospasm with an associated increase in intracranial pressure. To reduce these complications the airway must be controlled with speed and efficiency by a skilled member of the resuscitation team. Ensure adequate oxygenation before attempting intubation. It should not take more than 15 seconds to insert an endotracheal tube.

Airway control in head- or neck-injured patients is particularly important. Obtain expert help before attempting to intubate a patient with a suspected cervical spine injury. Artificial ventilation can be maintained in the short term by bag and mask and frequent use of the sucker.

Much emphasis has been placed on the possibility of increasing the severity of a *cervical spine injury* during intubation. However, it is highly unlikely that gentle movement in a controlled environment will do what the initial injuring agent failed to do. It is unreasonable to suggest that the major violence required to dislocate articular facets, crush vertebrae or rupture longitudinal ligaments would not extend to injuring the cord. Certainly, rough handling, especially rotation or flexion, could complete a partial cervical cord injury, so care is essential. However, it is probable that many of the documented neurological deteriorations seen in the first few hours after the accident have a vascular basis. The cord may become oedematous, infarcted, or compressed by an expanding haematoma. A neurosurgical opinion should be sought as soon as primary resuscitation is underway. The very early application of skeletal traction may prevent a vascular complication. Alternatively, laminectomy may be considered appro-

priate. In any event the specialist must be involved in the early management of the injury.

Very rarely, anatomical variations, direct trauma to the larynx, or the presence of impacted foreign bodies will preclude intubation. In such cases a *cricothyroid stab* is preferable to tracheostomy (p.230). A skilled intubator can then be summoned or a tracheostomy planned with less haste (and less morbidity).

When the trachea has been intubated 100% oxygen is given by intermittent positive pressure using a self-expanding resuscitation bag and valve. Always check air entry in both lung fields. The tube is commonly inserted too far and enters the right main bronchus. Aspirate tracheal secretions through the endotracheal tube using a fine non-traumatic catheter.

Acute gastric dilatation may occur after trauma and is especially common in head-injured children. The patient deteriorates and the doctor's anxiety increases along with the abdominal girth. The risk of inhalation of vomit is increased. Insert a large-bore silastic nasogastric tube. Advance it cautiously if there is nasal bleeding. There are case reports of tubes passing into the cranium through the roof of the nasal cavity in patients with fractures of the base of the skull.

Circulation

Prevention of blood loss. Exert firm pressure on major bleeding points. Do not use tourniquets. Arterial 'pressure points' are of no value.

Access. Insert two large-bore (e.g. 16 G) intravenous cannulae. One should be a central line (p.173). The second may be in the antecubital fossa or the leg. The siting of intravenous lines is controversial. Most U.K. authorities advocate large-bore cannulae in the upper limbs. However, mediastinal or neck injuries may prevent this fluid from reaching the core circulation. Some centres now advocate the routine insertion of a cannula 'below the diaphragm'. The saphenous or femoral veins are most appropriate. Sepsis and thrombosis are not considered to be major complications.

Assessment of volume lost. This is difficult. If the injured person was previously fit and has been transported to the A & E depart-