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LESSONS FROM A SYMPOSIUM ON EMERGENCY MEDICINE HELD IN THE COLLEGE ON 17 OCTOBER 1997*

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As individual fields of medicine become more specialised it is important that the management of a variety of acute medical emergencies is familiar to all doctors. Continuing education is thus necessary and symposia such as this one are an effective way of achieving this. The following report attempts to reflect the tenor of the papers presented by the individual speakers.

SHOCK

Shock is not synonymous with low blood pressure but is a state of inadequate tissue perfusion and resultant tissue hypoxia. Both high and low cardiac output states can occur in shock and parameters reflecting flow may be more useful for monitoring patients than blood pressure. Protective physiological mechanisms (such as catecholamines, aldosterone, the renin angiotensin system) aim to cause vasoconstriction with redistribution of blood to vital organs. Inflammatory pathways (mediated via cytokines, arachidonic acid and nitric oxide) also influence blood flow at a regional and microvascular level. Evidence is emerging that cells adapt to a reduced oxygen supply and essentially go into hibernation (e.g. 'stunned' myocardium). Compensatory mechanisms may in themselves be harmful and contribute to re-perfusion injury; thus aggressive resuscitation may improve ischaemic injury but worsen re-perfusion injury. Studies have shown that reduction of the tissue oxygen debt improves mortality, but such a debt may not be clinically apparent. Useful early indicators of a problem include a postural blood pressure fall and a high or rising blood base deficit; the latter, in the absence of acid ingestion, is indicative of ischaemia and can reflect the response to treatment. Strategies to diminish cellular oxygen demand (e.g. mechanical ventilation to reduce the work of breathing) may be useful. Recently developed methods of monitoring blood flow and tissue perfusion/oxygenation include oesophageal Doppler ultrasound scanning via an oesophageal probe, and measurements of tissue oxygen in the conjunctiva and of carbon dioxide in the gut mucosa. New treatment strategies include fluids with constituents able to carry oxygen (such as perfluorocarbons), cellular protectants, oxygen-scavenging treatments and cooling.

ACUTE GASTRO-INTESTINAL BLOOD LOSS

The incidence of acute gastro-intestinal haemorrhage is 47-116 per 100,000 people and is higher in less affluent areas. Peptic ulcer haemorrhage accounts for 35-52% of all cases. A mortality of 11% was reported in a 1996 audit,[1] and is not dissimilar to that reported by Francis Avery Jones in 1947 of 16%; the population is now older and the 1996 audit showed that 27% of those succumbing to GI haemorrhage were aged over 80. Factors associated with increased risk of mortality included increasing age, initial haemodynamic state, co-morbidity such as renal failure or cardiac disease, endoscopic stigmata and the occurrence of re-bleeding.

*A list of speakers and the titles of their papers presented at this symposium is recorded in *Proceedings* Vol.28, p.132.

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Endoscopic findings are important: if active haemorrhage is found there is an 80% chance of either the lesion continuing to bleed or of a re-bleed. If there is a vessel visible, 50% will re-bleed while in hospital; those with a clear ulcer base have a more favourable course.[2,3] Patients often die from an underlying condition unrelated to the acute bleed that has become decompensated secondary to the bleeding event.

Improvement in mortality can be achieved by improvement in resuscitation, the development of teams and units dedicated to managing acute GI bleeding and the use of management protocols and specific treatments.[4]

The medical treatment of bleeding includes agents that reduce acid secretion (H2 receptor blockers and proton pump inhibitors) - since stability of the clot is related to pH, agents that reduce arterial blood flow such as octreotide, and anti-fibrinolytic agents such as tranexamic acid. However, using the proton pump inhibitor omeprazole did not affect mortality in acute bleeding due to gastric or duodenal ulcers (DU).[5] Upper GI haemorrhage can also be managed surgically or by interventional endoscopic techniques.

The most effective direct treatment for acute bleeding is surgery, but this has significant mortality and morbidity. Endoscopic therapy includes thermal sclerotherapy, injection into the bleeding point using agents such as adrenaline, or mechanical methods such as the use of clips. Combination treatments such as adrenaline injection together with heat-probe treatment are also used; however, a recent study concluded that there was no advantage to combination therapy except in a subgroup that had spurting haemorrhage.[6] Operator experience rather than method of endoscopic treatment is the important factor. Complications of injection therapy include exacerbation of bleeding, perforation and necrosis, and delaying of potential surgical treatment. Factors associated with failed endoscopic therapy are a posterior DU, large ulcers, and significant coexisting disease.

Endoscopic treatment will have a significant impact on 10-15% of patients, 80% will stop bleeding spontaneously and 5-10% will require surgery.[7,8] The best management is achieved by specialists, a dedicated team and the appropriate use of endoscopy.

A LIFE-THREATENING INFECTION: MENINGITIS

Notification rates of meningitis in the UK are two to three per 100,000 population (probably an underestimate). The aetiology is usually bacterial or viral (occasionally mycobacterium in the Asian population). Viral meningitis is unlikely to kill unless it is due to infection with the Herpes virus, but it can mimic bacterial meningitis. In the immune competent person in the UK the important bacteria pathogens are *Neisseria meningitidis* and *Streptococcus pneumoniae*. Patients who are seroconverting to HIV can develop an acute aseptic meningitis; this is important to be aware of as there are therapies available now that can alter prognosis in HIV when given at an early asymptomatic stage. In the history of a patient presenting with meningitis, particular reference should be made to: any foreign travel, such as to Africa where the meningococcus is endemic, previous episodes or the use of prophylactic antibiotics (this may mean a more resistant organism), head trauma, and defects in host immunity. Examination must include the ears, joints, the presence of ventriculo-peritoneal shunts, and the exclusion of focal neurological signs.

The need for a head CT scan prior to lumbar puncture is controversial. Treatment should be instigated immediately whenever meningococcal meningitis is suspected. If there is impairment of consciousness, seizures, worsening of headaches or focal

neurological signs, a CT scan is mandatory. Raised intracranial pressure may exist with very few symptoms or signs.

Polymerase chain reaction (PCR) analysis of the CSF is helpful in viral infections (particularly due to Herpes in which PCR should be considered the gold standard,[9,10]), in the immune-compromised, in partially treated disease, in disease where response to treatment is poor, and in epidemiological studies. Sensitivity approaches 100% in invasive meningococcal disease but is only 20-40% in tuberculous meningitis. A normal or near-normal CSF may be seen in partially treated meningitis, parameningeal infection (e.g. of sinuses or bone) or in the infected but immune-compromised patient. In the Gloucestershire study[11] of patients with meningococcal meningitis, 8% initially had a normal CSF (later becoming culture-positive). Intracranial bleeds may mimic or coexist with meningitis.

For adults in the UK, conventional treatment of bacterial meningitis uses high dose benzyl penicillin or a third generation cephalosporin. This is likely to change as penicillin resistance occurs. The role of corticosteroids is controversial; in children with *H. influenzae* meningitis they reduce hearing deficits if given with the first antibiotic dose, but not mortality.[12] Steroids should be given in tuberculous meningitis with dose adjustment if there is concurrent rifampicin therapy. Chemoprophylaxis should be offered to close contacts and to hospital staff involved in a resuscitation situation. In the Gloucestershire outbreak of *N. meningitidis* in the 1980s and early 1990s, only 102 of 191 culture-positive cases were actually notified.[11] Notifying cases identifies outbreaks. The median time for spread from the index case to the next is 24 hours and thereafter spread is rapid. Most sporadic cases in the UK are due to the type B organism. There has been an increase in mini clusters with type C. With the advent of immunisation against *H. influenzae* there is likely to be a reduction in the overall incidence of *H. influenzae* meningitis infection, but children who missed the vaccination may develop the disease later.

APPROACH TO THE BREATHLESS PATIENT

Breathlessness is a sensation with a receptor, an afferent pathway, a central response, and an efferent pathway.

Every patient has a different dyspnoea threshold. Receptors for breathlessness can sense work expended on breathing, stretch, chest wall and diaphragm excursions, hypoxia and hypercapnia. The respiratory rate can be used as an objective measure of the degree of breathlessness.

Oxygen is not a major driving force to the sensation of breathlessness; the patient does not need to be hypoxic to be breathless. Pulmonary interstitial stretch receptors play a bigger role. In airflow obstruction hyperinflation leads to increased work of breathing but despite this the patient can start to feel better even without improvement in objective parameters. This is due to resetting of the breathlessness receptors. Some asthmatic patients are poor perceivers of breathlessness.

Common causes of breathlessness include asthma, chronic obstructive pulmonary disease (COPD) and infection. When a monophonic wheeze is present, obstruction from a foreign body must be excluded: an expiratory film will show hyperinflation of the affected lung and loss of volume in the other field. In neuromuscular disorders causing breathlessness such as poliomyelitis, Guillain-Barré syndrome and myopathies, vital capacity (VC) is a critical measurement; a falling VC is an indication for assisted ventilation.

CURRENT ISSUES IN ACUTE POISONING

Treatments specific to poisoning include gut decontamination, antidotes, and elimination techniques. Gut decontamination comprises induced emesis, gastric lavage, whole bowel irrigation and oral activated charcoal. The use of ipecacuhana to induce vomiting is no longer recommended as studies of gastric lavage fluids show that significant drug recovery is rare.[13] Gastric lavage is only indicated when a potentially toxic amount of a substance has been ingested within the previous two hours. Well-known antidotes are naloxone for opiates, and acetylcysteine for paracetamol.[14] Flumazinal is a benzodiazepine antagonist but is not licenced for use in overdoses. The majority of benzodiazepine overdoses will do well with purely supportive management. Benzodiazepines are often taken with tricyclic antidepressants and this leads to a deeper coma. Giving a benzodiazepine antagonist in this situation should possibly be avoided because of the risk of seizures and arrhythmias which has been demonstrated in dogs.[15]

In paracetamol overdose the commonly-quoted 'treatment line' (from the graph of paracetamol level versus hours post ingestion) is, in fact, a prognostic line developed by Prescott[16] and is based on peak disturbance of liver function in adults who had ingested paracetamol alone. The validity of this curve beyond 15 hours is uncertain, as is its applicability to children. Increased risk of liver damage following paracetamol overdose occurs in patients with chronic alcoholic abuse, eating disorders like anorexia, and concurrent treatment with enzyme-inducing drugs such as isoniazid. Paracetamol levels measured at four hours post-ingestion determine the need for N acetyl cysteine (NAC) therapy. NAC has both early and late actions: in the early stage it is a glutathione precursor, later it acts as a positive inotrope, free radical scavenger and vasodilator. Liver transplantation can be performed for hepatic failure following paracetamol overdose.

PSYCHOLOGICAL ASPECTS

Suicide rates fell during both World Wars, and have increased in men but have decreased in women since the 1970s. The mode of suicide also differs between men and women. Women tend to ingest poisons whereas men choose hanging or carbon monoxide poisoning. The discharge rate for poisonings has increased in the Royal Infirmary of Edinburgh from 1,500 in 1989 to 3,000 in 1996. Only about 50% of parasuicides are serious attempts at death.

Improved recognition of depression and the use of drugs which are less harmful when taken in overdose, e.g. selective serotonin re-uptake inhibitors (SSRIs) should help to reduce the death rate from overdose. There should be a multidisciplinary approach to the management of the self-poisoned patient with involvement of emergency staff, physicians, psychiatrists, social workers, and nurses.[17,18] The self-discharge of a patient who attempts self-harm should only be allowed after an assessment of suicide risk.

ACUTE NEUROLOGICAL HEADACHES

Focal lesions such as brain tumours and abscesses tend to present sub-acutely rather than with acute headaches. Subarachnoid haemorrhage (SAH) is classically said to occur with the 'hit over the head' history. Even with this classical description only one in four of patients with this symptom will have a confirmed SAH. Other diagnoses to be considered include migraine, 'thunderclap' headaches and exertional headaches. It is important to investigate such headaches because if they should recur there should be no doubt of the original diagnosis. Investigation should include a head CT scan

and lumbar puncture (LP). The CT scan within the first few days of a subarachnoid bleed will be positive in 95% of cases, but will not be positive after two weeks. The incidence of a warning bleed or sentinel bleeds preceding SAH is unknown. Some retrospective studies quote 40-60% of patients as having had significant headache prior to the event. A prospective study in Holland has suggested that this is an overestimate and that warning headaches are quite rare.[19] Other common causes of headaches include wry neck, meningism, coital or exertional headache, sinusitis, and temporal arteritis.

APPROACH TO THE PATIENT WITH CHEST PAIN

Chest discomfort represents approximately 20% of all general medical hospital admissions in the UK. In Ninewells Hospital, Dundee, Scotland, a recent unpublished audit has found a similar figure. This audit also found that a large number of patients self-refer and that many of these are discharged within 24 hours. Patients must be given a firm diagnosis at initial presentation or they will keep re-presenting, particularly if a cardiac cause has not been firmly excluded. Speeding up diagnosis could involve open access facilities for endoscopy, exercise tolerance testing, and rapid access chest pain clinics. A diagnosis of 'chest pain query cause' is not acceptable. A delay in reaching a diagnosis leads to patient anxiety and reduces the likelihood of a definite diagnosis being made.

Thrombolysis is the treatment of choice for acute myocardial infarction (MI). 'The door to needle time' in the administration of thrombolysis approaches 30 minutes in most units. In rural areas thrombolysis may be administered by general practitioners but there can be problems with hypotension and arrhythmias.[20] The other delay of significance is that of 'the pain to call help' time. Public education with regard to this is vital but needs to be on-going. A study on the impact of faxing ECGs to a more senior member of staff revealed agreement in management in 87% of cases; in 8% an unnecessary treatment was avoided and in 4% additional treatments were given.[21]

Significant cardiac disease can exist with atypical or minimal symptoms. Cardiac and oesophageal problems often coexist. Oesophageal acid perfusion can provoke angina (linked angina) even in patients with normal coronary arteries, and can cause ECG changes. In Cambridge, work has demonstrated the presence of a neuro-cardio-oesophageal reflex.[22] Both types of pain can get better with calcium antagonists.

MANAGING UNSTABLE ANGINA

The 'average' patient with stable angina can expect an annual catastrophic cardiac event rate of at least 1% (the OASIS Registry has reported 10% death/myocardial infarction at six months). This event rate increases in unstable angina. The Braunwald classification of unstable angina has three categories. The first is new, severe or accelerated angina not occurring at rest; the second is angina at rest with the most recent episode being at least 48 hours earlier, and thirdly angina at rest with the most recent episode being within 48 hours. Each of these categories can be further subdivided into unstable angina secondary to an extra cardiac cause, primary unstable angina or post-infarction unstable angina. Group three of the Braunwald classification occurring post infarction has the highest subsequent cardiac event rate.

Patients with unstable angina have pain and ECG changes, and a 5% risk of death or myocardial infarction in seven days and 10% in six months. The admitting ECG is helpful in determining who will develop a full thickness infarct. If there is ST segment elevation on the admitting ECG, the patient is more likely to go on to have a full thickness infarct and there is a high 30-day cardiac event rate of 11%. In the absence

of ST segment elevation, or if there is ST segment depression, should an infarct occur it is less likely to be full thickness and more likely to be non-Q wave with a lower 30-day cardiac event, and mortality, rate.

In unstable angina there is a destabilising influence which may exist for some time. In the affected coronary artery there is usually an atheromatous plaque.[23,24,25] Smaller, thinner-capped plaques are more likely to rupture allowing thrombogenic material from the plaque's core to come into direct contact with blood. Platelets are stimulated via the arachidonic/thromboxane pathway (blocked by aspirin), undergo a configuration change and interact with blood proteins particularly fibrinogen. This can lead to either an occlusive or a non-occlusive thrombus.

Early use of aspirin leads to a significant reduction in mortality from acute coronary ischaemic syndromes. Platelet activation bypasses the arachidonic acid pathway, and so aspirin is only partially effective. There are drugs that bypass the arachidonic/thromboxane pathway and inhibit the final platelet adhesion step: the glycoprotein IIb/IIIa receptor antagonists. Different IIb/IIIa receptor antagonists have undergone trials: abciximab (used in the CAPTURE,[26] EPIC[27] and EPILOG trials[28]), tirofiban (the RESTORE trial[29]) and itegrilin (the IMPACT II[30] and PURSUIT[31] trials). These drugs were used in patients undergoing invasive coronary procedures and also used in trials for unstable angina by PURSUIT. In the CAPTURE trial an initial benefit was shown but was lost by six months. In the EPIC trial a benefit was seen up to three years.[27] The future lies with oral preparations of these drugs.

The role of heparin in acute coronary syndromes remains controversial, but there is a trend towards use of lower doses. There is no strong evidence of any long-term benefit from the combined use of heparin and aspirin, but are probably useful in the early phase of unstable angina.[32] There is no benefit from high-dose over low-dose heparin with IIb/IIIa receptor antagonists. Low molecular weight heparin appears superior to unfractionated heparin (e.g. the FRISC[33]) but there is no evidence yet that there is additional benefit from its addition to a IIb/IIIa receptor antagonist. Both GTN and heparin provide symptomatic treatment but there is no advantage in using them together.

Early interventional treatments for unstable angina such as acute coronary surgery or percutaneous transluminal coronary angioplasty (PTCA)[34] have not yet been confirmed by appropriately-powered trials as having any long-term benefit. If early PTCA is to be carried out then it should be done in a good centre and a IIb/IIIa receptor antagonist should be given (e.g. the CAPTURE trial).[26]

Treatment goals in patients with unstable angina are to relieve symptoms, abolish ischaemia, reduce infarct size and improve prognosis. Management entails close monitoring of the patient with frequent ECGs, the use of aspirin, measures to control pain such as beta blockers, nitrates or heparin (beta blockers have not been known to alter mortality in unstable angina). Dihydropiridine calcium channel blockers should not be given in acute ischaemic coronary syndromes. Continuing pain requires angiography and an interventional procedure, but this is a symptomatic measure and has not yet been shown to improve prognosis.

ARRHYTHMIAS

Arrhythmias can be divided simply into regular or irregular, and the QRS complex can either be narrow or wide. A wide complex tachycardia should be assumed to be ventricular (VT) until proven otherwise. The ventricular nature is demonstrated by the presence of dissociated p waves, fusion and capture beats. Other features suggestive

of VT are an extreme left axis, a very wide QRS complex (>160ms), and concordance. Comparison with previous ECGs or with an ECG after the rhythm has been terminated is very useful.

The mode of treatment of any arrhythmia depends on whether there is any haemodynamic compromise. If there is compromise, treatment of VT is electrical cardioversion (synchronised under general anaesthetic, if time permits). If the rhythm is tolerated then cardioversion with drugs should be attempted and intravenous lignocaine is the first line drug. Other methods include ventricular over-pacing, and elective cardioversion. Verapamil should not be used in VT, nor is adenosine recommended as it can cause A-V dissociation. After correction of VT or VF, the underlying cause should be determined (usually ischaemic heart disease). Long-term therapy includes drugs, implantable cardioverter defibrillators (ICDs) and myocardial action potential (MAP-) guided endocardial resection, but the latter carries a mortality risk.[35] The recent AVID trial concluded that among survivors of ventricular fibrillation or sustained ventricular tachycardia causing severe symptoms, the implantable cardioverter defibrillator is superior to antiarrhythmic drugs for increasing survival. Referral to a specialist centre should be made.

Supraventricular tachycardia (SVT) can be wide complex in the presence of bundle branch block (A-V node re-entry, atrio-ventricular re-entry in accessory pathway), antidromic tachycardia and atrial flutter with bundle branch block. The production of A-V nodal block is useful and can be achieved with vagal manoeuvres, and by using drugs such as adenosine or verapamil. If there is haemodynamic compromise the patient needs electrical cardioversion, if not, adenosine, verapamil or agents to control rate such as beta blockers should be used.

A narrow complex tachycardia occurs in atrial flutter where there is a rate of 300/min or 150/min in the presence of a 2:1 block. Manoeuvres such as carotid sinus massage (CSM) or the administration of adenosine may not terminate the problem but will usually reveal the diagnosis. Narrow complex SVT can be treated with cardioversion (if the patient is haemodynamically compromised), carotid sinus massage, verapamil, adenosine or a pacemaker.

The treatment of a single episode of SVT without collapse, with a normal ECG in sinus rhythm and no delta wave requires that the patient be taught vagal maneouvres. If recurrent episodes occur then referral to a specialist is needed since ablation therapy may be indicated.

Atrial fibrillation (AF) is an irregular atrial tachycardia, which can be narrow complex or broad if associated with bundle branch block or an accessory pathway. It is the commonest arrhythmia requiring hospital admission. In AF of new onset, an underlying cause such as ischaemic heart disease, mitral valve disease, thyrotoxicosis, hypertension, and cardiomyopathy should be sought. In new onset AF, the aim of treatment is restoration of sinus rhythm. Electrical cardioversion should be preceded and followed by four weeks of anticoagulation since, even after a successful cardioversion, it takes time for the atrial mechanical function to return to normal. Maintainance of sinus rhythm requires drugs such as sotalol, amiodarone or propanfenone. Digoxin is only useful to control the ventricular rate but this can also be achieved with beta blockers or calcium antagonists. Patients with chronic atrial fibrillation should normally be anticoagulated with warfarin. If there are contraindications to warfarin then aspirin should be given.

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