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Management of acute asthma

Acute asthma is a medical emergency and an indication for admission to hospital. The first sign of the development of an acute attack is that the patient fails to obtain relief from his or her usual treatment. All asthmatic patients should understand that if their condition is deteriorating then a change of treatment is needed, and they should therefore have ready access to their doctor or to a unit with a special interest in this disease. Unfortunately the gravity of a worsening attack of asthma is not sufficiently recognised by patients, their relatives, and even their doctors. In the epidemic of asthma deaths in the late 'sixties the event was sudden and unexpected in almost 80% of patients, most of them young, who died at home or were dead on arrival at hospital.[1]

What, then, indicates that an attack of asthma is entering a severe and dangerous stage? The ability to continue a conversation is a good clinical guide: mild—frequent pauses between speech; moderate—monosyllabic speech; and severe—too dyspnoeic to speak.[2] Thoracic overinflation, tachycardia, pulsus paradoxus, and electrocardiographic abnormalities of right ventricular hypertrophy and strain are common, and the patients are usually unresponsive to aerosol adrenergic-stimulant drugs.[3] While spirometry is helpful in assessment, arterial puncture and measurement of the arterial oxygen tension (Pa_{O_2}) and carbon dioxide tension (Pa_{CO_2}) and pH are essential. Hypoxaemia is invariably present, often with normal or low levels of CO_2, but in the most severely ill patients hypercapnia and acidaemia will be found.[4 5]

The treatment of the patient with severe asthma is rehydration by intravenous drip transfusion and relief of hypoxaemia by oxygen therapy to maintain the Pa_{O_2} between 7·95 and 10·59 kPa. If the patient is hypercapnic controlled oxygen at a lower concentration will need to be given. If the patient is able to co-operate physiotherapy should be arranged—hypercapnia is invariably associated with copious retained bronchial secretions.[3] Persisting hypercapnia (Pa_{CO_2} > 7·95 kPa) is frequently an indication for assisted ventilation.[2]

Sedatives and tranquillisers are contraindicated in severe asthma, but because infection is a common precipitating cause a broad spectrum antibiotic should be given. The best treatment to relieve the airways obstruction has been the subject of much investigation. While severely ill asthmatic patients cannot obtain benefit from portable adrenergic bronchodilator aerosols, salbutamol has been claimed to be effective when administered for periods up to 3 minutes as a 0·5% solution by intermittent positive pressure ventilation using a Bennett or Bird ventilator connected to a tightly fitting face-mask.[6] More recently intravenous salbutamol (100-300 μg) has been shown in severe asthma to lead to a mean increase in peak expiratory flow of 44% accompanied by a rise in mean pulse rate of 24 beats/min.[7] No side effects were reported from this treatment, but the mean pulse rate of 120 before treatment rose to 140 ten minutes after the injection. Intravenous salbutamol in comparable doses has been shown to produce a definite rise in free fatty acid levels and in plasma insulin, glucose, and lactate values.[8] While the clinical implications of these findings are not certain, high free fatty acid concentrations, high catecholamine levels, and hypoxaemia are thought to be important in the development of arrhythmias after myocardial infarction,[9] and for this reason caution is advisable in the use of parenteral beta-adrenergic drugs in severe asthma.

Parenteral aminophylline is a long-established method of treating severe asthma resistant to beta-adrenoceptor stimulants, but it must be given slowly and well diluted to avoid toxicity. The optimal intravenous dose is 375 mg followed by a total daily dose of not more than 1 g.[10] The combination of aminophylline with beta-adrenergic stimulants in large doses may be undesirable because of its lipolytic and cardiac-stimulant properties.[11]

The most important drugs in the treatment of severe asthma are the corticosteroids, but there is uncertainty about how these drugs act, how they should be given, and in what dosage. One of their effects is to restore sensitivity of the bronchial muscle to exogenous or endogenous catecholamines, leading to bronchodilatation.[12 13] A bronchodilator effect has been shown one hour after the intravenous injection of prednisolone, the peak effect occurring after eight hours. Intravenous hydrocortisone produces an earlier peak effect at five hours, and a definite response is apparent at one hour.[14] When prednisolone is given orally an effect is evident at three hours, the maximum change is at 9 to 12 hours, and no change is detectable at 36 hours.[15] It has been suggested[16] that in severe asthma enough corticosteroid should be given to maintain a plasma cortisol level of 100-150 μg/dl. This may be achieved by giving hydrocortisone hemisuccinate intravenously at a dose of 4 mg/kg body weight about every three hours. If hydrocortisone is given by continuous intravenous infusion a lower dose regimen of 3 mg/kg body weight every six hours has been found satisfactory after a loading dose of 4 mg/kg hydrocortisone intravenously.[17]

With either treatment oral potassium supplements should be given daily. After intravenous therapy, when the patient is improving, prednisone will need to be given by mouth, the dose being reduced slowly.

Because of the delay in the appearance of the maximal effect of parenteral corticosteroids it is prudent for the doctor to give 200 mg of hydrocortisone intravenously to the patient who is being sent to hospital from an outlying district. Corticotrophin is not usually recommended for the treatment of acute asthma in patients on regular treatment with corticosteroids because the adrenal response in terms of cortisol output is likely to be inadequate. But patients with severe asthma who were not dependent on steroids have done well when given tetracosactrin depot (1 mg intramuscularly on admission followed by repeated injections of the same dose at 24-hour intervals for three to five days). Though the plasma cortisol levels rose significantly on this regimen they did not reach 100 μg/dl.[17] Again, oral potassium supplements must be given.

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Transplant sensationalism

Anyone who wonders why British transplant surgeons have difficulty in getting cadaver organs need look no further than the front page of the *News of the World* for 28 September.[1] A banner headline "The Body Snatchers" introduced an article suggesting that the Department of Health's recent circular[2] on the interpretation of the Human Tissue Act 1961 "opened the way for no-consent transplants." In fact, the circular did no more than confirm the advice given by our legal correspondent[3] as long ago as 1973: that when patients die in hospital the "person lawfully in possession of the body" is the hospital authority, and that in those circumstances the kidneys may be removed without the relatives' specific consent provided that reasonable inquiries have failed to show any evidence of objection by the patient or his family.

Sadly, the Medical Defence Union persists[4] in taking an opposite view and in advising its members that they may risk civil action if they follow the Government's guidance. Yet the circumstances in which the legal uncertainty is relevant are relatively few; for there would be no shortage of kidneys if full use was made of the opportunities presented by patients dying in intensive care and neurosurgical units. Almost always in such cases the relatives are available for consultation; but only too often the clinicians concerned prefer to ignore the possibility and make no approach for consent.

Part of this reluctance is, no doubt, due to pressure of work and a natural unwillingness to intrude into the relatives' grief, but a second important factor is the antagonism to transplantation still to be found in some members of the public and whipped up by newspaper sensationalism.

The disappointing response by the public to the Department of Health's donor card scheme may well be attributable—at least in part—to the antagonism shown by some sections of the press to transplantation. Anti transplant propaganda—like other vociferous protest campaigns—commonly combines emotion and ignorance and often misrepresents the facts.

[1] *News of the World*, 28 September 1975.
[2] *Health Service Circular*, HSC (1S) 156.
[3] *British Medical Journal*, 1973, **3**, 360.
[4] *Daily Mail*, 29 September 1975.

Painful redistribution

There are two major issues about the allocation of resources to the National Health Service. The first is what proportion of the nation's income should go to the NHS. The second is how best to share out the available resources within the Service. Uncertainty about the former gives added urgency to the latter: the less money there is around the more important it is to ensure that it will be distributed in an equitable way. Given our present economic plight, it is therefore not surprising that the Department of Health should have set up a working party in May this year to look into the distribution of resources within the NHS and that the working party in turn has put on an unusual turn of speed to produce its first interim report.[1] If its recommendations are accepted and implemented the report's effects will be both unprecedented and considerable: there will be a cut in the total revenue funds allocated to some of the regional health authorities in the next financial year.

The NHS inherited unequal distribution of resources among the different regions of the country, seemingly unrelated to available indicators of need.[2] The persistence of these inequalities persuaded the DHSS in 1970 to introduce a new formula for allocating revenue funds to the regions. This was designed to iron out some of the more glaring discrepancies over 10 years by allowing the budgets of the worst-off regions to increase at a faster rate than those of the best-off ones. Nevertheless, the success of this approach—which was in any case criticised for its leisurely timetable—depended crucially on the overall growth in the resources of the NHS as a whole: the scope for bringing about equity by a differential growth rate obviously diminishes if the growth rate itself falls (or if there is no growth).

This, then, is the problem to which the working party—composed predominantly of NHS administrators and DHSS officials—addressed itself. The 1970 formula was based on three rough and ready indicators of need: population structure, occupied beds, and case load. This clearly favoured the status quo: by including bed numbers as indicators of present need (as distinct from past policies) it loaded the