PAS[23] and possibly also isoniazid[24] can interfere with the estimation of SGOT to produce spuriously high values.

Some means of predicting a patient's hypersensitivity to antituberculous drugs is urgently needed. Unfortunately, in vitro tests such as the lymphocyte transformation test, in which the patient's lymphocytes are exposed to the drug under suspicion, have yielded conflicting results for antituberculous agents.[25–28]

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Adult Respiratory-Distress Syndrome

The adult respiratory-distress syndrome can be defined as noncardiogenic pulmonary oedema occurring in patients with previously normal lungs. The conventional teaching is that pulmonary oedema occurs as a result of an imbalance of the normal hydrodynamic forces across the alveolar capillary membrane. This most often occurs as a consequence of left ventricular failure or left atrial hypertension leading to an increase in pulmonary venous pressure.[1] However, it is now clear that pulmonary oedema can occur when the pulmonary venous pressure is not raised and where the lungs were previously normal. In most such patients there is damage to the alveolar-capillary membrane and increased pulmonary capillary endothelial permeability; there is invariably tissue hypoxia, hypotension, and respiratory or metabolic acidosis; and in some instances there are additional, more complex aetiological factors such as disseminated intravascular coagulation.[5]

Clinically the patient with the adult respiratory-distress syndrome is shocked, acutely dyspnoeic, and cyanosed and has diffuse alveolar exudates on the chest radiograph. Arterial blood gas analysis shows severe hypoxaemia (Pao_2 below 6·63 kPa) when breathing room air, a markedly widened alveolar-arterial oxygen gradient on supplemental oxygen, and sometimes hypercapnia.[6] Conditions in which the syndrome occurs include blast injury to the lung, severe hypoxaemia after burns or extensive injury, septicaemia, endotoxaemia, renal failure, cerebral trauma, fat embolism, exposure to high altitudes, aspiration and viral pneumonias, drug overdose particularly with heroin and the barbiturates, and after cardiopulmonary bypass: and this list is not exhaustive.[2–5]

Whatever the precipitating cause of the condition, these patients are gravely ill and when admitted to hospital are usually soon transferred to an intensive care unit. Treatment should be directed towards improvement in oxygenation, artificial ventilation given where indicated, correction by suitable intravenous therapy of the acid-base disturbance and treatment of infection (when present) by appropriate antibiotics. However, the pathophysiological consequences of the adult respiratory-distress syndrome may lead to myocardial dysfunction and left ventricular failure, which must be recognized and treated, because when it is treated promptly the prognosis is improved. But how is the presence of left ventricular failure in the adult respiratory-distress syndrome recognized? Unger *et al.*[6] have recently claimed that physical examination is unreliable; they found that tachycardia, raised jugular venous pressure, cardiomegaly, hepatomegaly, and adventitious sounds in the lungs were equally common in patients with adult respiratory distress syndrome with or without left ventricular failure. Nor was the electrocardiograph helpful. Only the presence of peripheral oedema and a left ventricular third heart sound were slightly more common in those with left ventricular failure than in those without, but these signs of themselves were not diagnostic.

Of the tests done by Unger *et al.* the most useful in detecting left ventricular failure was measurement of the pulmonary wedge pressure. This we know is a good indicator of left ventricular end-diastolic pressure and thus of left ventricular failure.[7] Of their 14 patients, 10 had a pulmonary wedge pressure below 12 mm Hg (mean 5 mm Hg, range 2·0-9·5 mm Hg) and were therefore considered not to have left ventricular failure, whereas 4 patients had pressures above 12 mm Hg (mean 20 mm Hg, range 15-23 mm Hg) and were considered to have left ventricular failure. In three of the four patients in the failure group a decrease in pulmonary wedge pressure was noted after fluid and salt restriction, diuretics, dialysis, or digitalis. All three had at the same time clinical improvement, a decrease in infiltrates on the chest film, and an increase in arterial oxygen tension. Three of these four patients survived, whereas of the 10 patients with the adult respiratory-distress syndrome without left ventricular failure only six survived. The four who died had necropsies; and two had normal hearts, one had evidence of an old healed myocardial infarct, and one had extensive acute bronchopneumonia and an agonal myocardial infarct (his pulmonary wedge pressure rose to abnormal levels terminally). Of the four with a raised pulmonary wedge pressure, the one who died had severe pneumonia and extensive, widespread fibrosis of the myocardium.

There are important lessons to be learnt from this experience, though the number of patients studied is small. Right heart catheterization is a simple, safe bedside procedure and

can supply extremely useful information in the management of these patients.[8]

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Epilepsy in the Elderly

The onset of epilepsy in adult life is an important and familiar clinical problem, but discussion has usually centred on the relative merits of methods of detecting cerebral tumours with little attention to the practical management of the patients. This aspect assumes particular importance when epilepsy develops in the elderly, in whom the more rigorous neuroradiological investigations are inappropriate and in whom the hazards of craniotomy are increased by the infirmities of age. Hildick-Smith[1] has emphasized recently that most published reports[2–5] have been on patients seen in neurological centres, to which patients with the multiple disabilities commonly seen in geriatric hospitals are unlikely to be referred. In her own series of 50 geriatric patients with epilepsy of recent onset a potentially reversible cause was found in four: one had intermittent heartblock, one drug-induced hypoglycaemia, and two raised levels of blood urea. A cerebral tumour was found in five patients, four being metastatic and one primary; these diseases were not amenable to other than palliative treatment and survival was brief. The commonest cause was cerebrovascular disease culminating in hemiplegia, and another common clinical association was dementia of unknown cause. Clinicians are uneasily aware that both sudden hemiplegia and dementia are among the modes of presentation of meningioma, and assessment of the necessity and value of elaborate investigation remains a difficult problem.

Most tumours found to be the cause of epilepsy after the age of 60 are malignant, only one recent series containing any number of benign tumours.[4] After the age of 70 tumours become increasingly uncommon as a cause of fits.[5] In at least half the patients no cause can be found, there is no evidence of disease, and good control over the fits (often entirely nocturnal) can be established with anticonvulsants. The indications for investigation to discover a primary intracranial cause are not therefore very strong. However, an elderly patient whose health would justify craniotomy if a benign tumour should be found ought to be investigated by harmless means.

A high degree of success in finding brain tumours has been claimed for the combined use of the electroencephalograph and isotope scan.[6 7] Both techniques will, however, also show abnormalities in patients with cerebral infarction, though with less tendency to persist. In fact the E.E.G. contains a high proportion of "abnormalities" in aged but otherwise apparently healthy individuals,[8] and only a slow wave (delta) focus can be considered relevant. The combination of these techniques with radiography of the skull and echoencephalography has been claimed to detect all cerebral tumours[9]; but the many patients with false positives submitted unnecessarily to angiography would certainly be even greater in the elderly and is unacceptable.

In practical terms cardiac and metabolic causes must be excluded, and radiography of the chest and skull may show evidence of primary or secondary carcinoma, or of pineal shift, or raised intracranial pressure. The dorsum sellae is often poorly calcified in old age and this may be misleading. Lumbar puncture should not be done unless meningitis (infective or carcinomatous) is suspected. More elaborate investigation must largely depend on the individual patient. There can be no virtue in searching for brain tumours in patients over 80 or in obviously poor general health. Any patient who is otherwise well and has no evidence of brain disease or has symptoms and signs of a progressive focal lesion should certainly be investigated by both E.E.G. and scan. If there is clear evidence of a focal lesion thought not to be due to an infarct the decision on whether to proceed to angiography should be taken in consultation with the neurosurgeon. If no such focal lesion is shown further investigation is not required, and anticonvulsant treatment has a good chance of success.

In a patient who, on a reliable history, seems to have had a stroke and later develops fits it would be reasonable not to investigate further unless there is evidence of progressive disease. Both E.E.G. and scan will be abnormal, and there is nothing to suggest that serial investigation is more reliable than serial physical examination. The elderly patient with dementia and epilepsy is extremely difficult to investigate. Movement may prevent scanning, the E.E.G. will be very abnormal whatever the cause, and air encephalography is harmful. Such patients are most unlikely to harbour a remediable intracranial cause for their symptoms. No doubt all these uncertainties will be clarified when computerized transverse axial scanning (the E.M.I. scannner)[10] is available in centres throughout the country.

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Sodium Nitroprusside in Anaesthesia

Induced hypotension during anaesthesia provides the surgeon with optimum operating conditions for such procedures as primary excision in massive burns, radical surgery of the head and neck, and the surgical treatment of phaeochromocytoma, cerebral aneurysms, and coarctation of the aorta. Profound hypotension is of great value in transsphenoidal hypophysectomy, dacrocystorhinostomy, and middle ear surgery.[1] In the past few years interest has been reawakened in the use of sodium nitroprusside (SNP) as a hypotensive agent. It was first[2] used clinically in 1929 and introduced into anaesthetic practice[3] in 1962, and since then has been used increasingly in anaesthesia and for the treatment of hypertensive encephalopathy.