peritoneal lavage was carried out, employing a peritoneal dialysis catheter, inserted through a small sub-umbilical incision in skin and linea alba. Five hundred ml normal saline were run in and returned clear. The catheter was removed and the skin closed with a single nylon stitch. Erect chest and supine abdominal films were normal.

The patient was managed conservatively with intravenous fluids and regular careful observation. Two h after presentation, while her general condition was unchanged, palpation revealed very obvious surgical emphysema in the lower abdominal wall. On a second erect chest X-ray, gas was clearly demonstrated under both hemi-diaphragms and laparotomy was, therefore, undertaken. There was a considerable amount of bile-stained fluid and gas in the retroperitoneal tissues around the duodenum, but only a small amount of free fluid in the peritoneal cavity. The duodenum was mobilised to display a 2 cm tear on the posterior surface of the junction of the second and third parts which was repaired. The patient made an uneventful post-operative recovery.

The detection of surgical emphysema in the anterior abdominal wall clearly heightened suspicion of major intra-abdominal injury. The gas must have come from a perforated or leaking viscus. The possibility of iatrogenic perforation by the dialysis catheter was considered, but seemed unlikely as there was no faecal or bile-staining of the returning irrigant. Since initial X-rays were normal, slow leakage of gas from the retroperitoneal tissues into the lesser sac and eventually into the general peritoneal cavity seems a likely sequence of events. In the supine patient, gas could rise to be expelled by contraction of the abdominal musculature through the small slit in the linea and into the subcutaneous tissues. This mechanism produced an unexpected but important clinical finding following initially negative peritoneal lavage which lead to the early detection of traumatic duodenal rupture, an injury in which diagnosis and treatment are commonly delayed for over 24 hours, and complications and mortality are correspondingly high.

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REFERENCES


Catecholamines and the heart

Sir
We read with interest the paper by Little et al. (1986, Vol. 3, pp. 20-7) and feel it deserves further comment. The importance of the heart as a catecholamine-producing organ was first suggested by Braunwald. In the normal heart under resting conditions,
noradrenaline is extracted from the blood and only released under substantial stress, such as rapid cardiac pacing whereas, in patients with ischaemic heart disease, the heart releases noradrenaline even at rest (Braunwald et al., 1964). Although baseline studies in patients with coronary artery disease do not show particularly high levels of noradrenaline (Goldstein, 1981), there is an exaggerated release in response to relatively minor sympathetic stimuli, such as a cold pressor test (Mueller et al., 1982).

The appearance of noradrenaline in the blood represents a spill-over from the synaptic cleft and is dependent on the level of neural stimulation, neuronal release and re-uptake. This latter process is an active one which is impaired by myocardial ischaemia, probably due to the metabolic accompaniments of ischaemia, i.e. decreased oxygen availability, acidosis and increased extracellular potassium (Dietz et al., 1981; Wollenberger & Shahals, 1965; Waldenstrom et al., 1978). This exaggerated release and reduced uptake indeed appear to be capable of creating a vicious circle leading to more free circulating noradrenaline which may in turn predispose to arrhythmias and sudden death as described in this study. Raab described many years ago the strikingly high noradrenaline concentrations in the hearts of people who died suddenly (Raab & Gige, 1955). Furthermore, elevated levels of circulating noradrenaline have been found to persist for at least a month following myocardial infarction and may play an important role in both pain and arrhythmias during this period (Valori et al., 1967).

The traditional concept regarding the potential danger of sympathetic (catecholamine) overactivity to the functional and structural integrity of the heart is usually attributed to a discrepancy between oxygen supply/coronary blood flow and increased oxygen consumption resulting from catecholamine-induced augmentation of cardiac work. However, sympathetic stimulation exerts its most immediate effects by liberating noradrenaline at the postganglionic sympathetic nerve terminals (Bassengeu et al., 1982) and produces widespread alpha adrenergic vasoconstriction. This can compete with and overcome metabolically-induced coronary vasodilatation, particularly in regions of decreased coronary reserve (Mudge et al., 1976). Moreover, Raab has shown the persistence of myocardial abnormalities in response to injections of adrenaline and noradrenaline which last much longer than the transient augmentation of cardiac work elicited by these hormones (Raab et al., 1962).

Although there is no denying that increased sympathetic efferent activity will occur due to activation of baro and chemoreceptor reflexes, there is increasing evidence that environmentally mediated stress exerts its effect through alpha adrenergic vasoconstriction (Billman & Randal, 1981). Even a simple mental stress test in patients with coronary artery disease produces an increased urinary noradrenaline excretion (Nestel et al., 1967) and marked disturbances of myocardial perfusion (Deanfield et al., 1984). The psychological and social results of acute myocardial infarction must be many times more stressful than a mental stress test and, therefore, attention to this is of particular importance. Considering that beta-blockers may, in fact, exacerbate alpha-adrenergic vasoconstriction (Robertson et al., 1982) their role in these cases must be viewed most cautiously and we advise against their routine use in the accident and emergency department. Additionally, the use of diamorphine can then be viewed as not only relieving pain but decreasing the environmentally produced hyperarousal. Since elevated sympathoadrenal medullary activity is now well-documented during the post-infarction days and weeks, physicians in coronary care units and during follow up would...
do well to remember this very important point which often seems to be lost in the plethora of multi-centre pharmacological trials.

Finally, the elevated cortisol levels found in this study are most important but are not at all surprising. Whilst emotional stresses and physical exercise cause sympathetic stimulation and catecholamine discharges, only the former is associated with simultaneous liberation of corticoids (Steptoe, 1981). Both Seyle (1974) and Raab (1971) have described the marked sensitisation of the heart by corticoids to the necrotising cardiotoxicity of both injected and stress-induced catecholamines and Troxler et al., (1977) have found important associations between elevated early morning cortisol and coronary artery disease. It appears that cortisol potentiates the action of noradrenaline either by increasing its extra neuronal uptake or by interfering with its subsequent degradation by catechol-O-methyltransferase (Kater & Higlieri, 1982).

These results are also in keeping with the literature of neuroendocrine arousal in patients with coronary artery disease in whom elevated noradrenaline and cortisol levels are found (Henry, 1983; Glass, 1977). Such hormonal levels are considered to be a response by the patient to the struggle to maintain their sense of control over a threatening environment (most marked in the coronary prone personality), or distress and helplessness when they perceive that they have failed (Buell & Eliot, 1980). Such circumstances must be almost at a peak during myocardial infarction. In a recent study at our institution, patients were studied following coronary arteriography to assess their suitability and need for coronary artery bypass grafting and, at a later date, when they had come to terms with the decision making process. Those patients who excreted more noradrenaline on the first occasion than the second had more episodes of silent ischaemia and longer periods of myocardial ischaemia, as documented by ambulatory ST-segment monitoring. Cortisol excretion was also significantly higher on the first occasion and there were more reports in their diaries of tension, anxiety and emotional upset. These results tend to add further support to the concept of psychological stress as an important dynamic factor, exacerbating fixed underlying coronary stenoses, perhaps by alpha-adreneric vasospasm (Freeman et al., 1986).

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REFERENCES


Letters to the editor


Erratum


It should be noted that the defibrillator/monitor/recorder chosen for this piece of equipment was a Cardiac Recorders 2006 Defibrillator/Monitor/Recorder Unit. This machine does not fully meet Part 1 of the BS5724 and neither does it meet the Part 2 requirements which will come into operation in April 1987. Cardiac Recorders have advised that they have no intention of upgrading this model to comply with Part 2 and that they now consider this to be an old and out-of-date model themselves. It is to be discontinued in favour of subsequent models.