Blunt injury to the heart

Blunt damage to the heart is widely believed to be difficult to detect and impossible to treat in a district general hospital. If this were correct there would be little else to write. Fortunately, diagnostic methods for assessing myocardial injury are improving, and lives can be saved by the timely application of widely available medical and surgical skills.

Cardiac trauma is potentially fatal,¹ and—in common with much other violence—the victims are often healthy young people. Most are injured in an accident on the road, at work, or at play, but some have been assaulted. Blunt cardiac trauma has been caused by bullets hitting soft body armour.²

Just how often the heart is damaged in blunt injuries to the chest is unknown. Most minor contusions probably resolve without detection, and those injuries which cause fatal arrhythmias may be elusive at necropsy. Some cardiac trauma probably occurs in many patients. The clinical effects may be apparent immediately or be delayed for days, months, or even years.³

One of the most common lesions is myocardial contusion,^{1 4} which produces pathological changes similar to those of acute infarction, with the attendant risk of arrhythmias and heart failure.^{5 6} Rarely there is direct damage to one of the major coronary arteries.^{7 8} More serious injuries may cause structural changes including rupture of the septum and damage to the papillary muscles or the chordae tendineae, producing valvular insufficiency.^{3 9} Sometimes cardiac tamponade occurs as a result of tears of the atria or right ventricle.¹⁰ Tears of the more sturdy left ventricle are rare.¹¹

A neat distinction is sometimes made between cardiac contusion and "concussion."¹² Cardiac concussion is defined as the occurrence of an arrhythmia without electrocardiographic changes or a rise in the serum enzyme activities indicating muscle necrosis. Most of these arrhythmias are transient, but sudden death has been reported after relatively minor blows to the sternum.¹³ This finding should not come as a surprise in view of the known effects of blows on the chest during cardiac resuscitation.

Any severe blow to the anterior chest wall should be treated with suspicion. Clinically, cardiac injury may declare itself as pericardial tamponade, an arrhythmia, or heart failure.⁶ Awareness, careful examination, and prompt investigation are the keys to diagnosis.

General surgeons have reported successful repairs of atrial tears presenting as tamponade.¹⁰ The immediate presence of a

cardiac surgeon and cardiopulmonary bypass facilities may save the occasional life,¹¹ but these services are not available in most cases.

In less urgent circumstances electrocardiographic changes typical of infarction may give the diagnosis. Changes may be non-specific, however, and the electrocardiogram is not sensitive enough to detect all cardiac injuries. Despite this, it appears that complications are most likely to develop in patients with electrocardiographic changes.¹² Tests assessing changes in specific cardiac enzymes such as the myocardial band of creatine phosphokinase detect more injuries,^{12 14} and refined techniques such as electrocardiogram-gated scintigraphy⁴ and high-frequency electrocardiogram analysis¹⁵ may be more sensitive still. Whether this increased sensitivity is a practical advantage remains to be seen.

The management of a patient with a cardiac injury is little different from that of myocardial infarction, but in the presence of other major injuries care must be of a high standard. Electrocardiographic monitoring is mandatory, and measurement of intracardiac pressures with a balloon-tipped catheter is a useful guide to fluid replacement during resuscitation. Careful auscultation must not be neglected: murmurs may indicate septal defects or valvular incompetence. These may appear early or late and demand full cardiological assessment.

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Parathyroid hypertension

Since the advent of multichannel autoanalysers the number of patients diagnosed as having primary hyperparathyroidism has risen sharply. Hunter Heath *et al*¹ reported a fourfold increase in the diagnosis after measurements of serum calcium concentrations became routine. The clinical range has dramatically shifted from the classical description of "bones, stones, and abdominal groans" to that of the asymptomatic patient discovered by routine biochemical analysis of the blood.

Hypertension is common in hyperparathyroidism but for some reason this association is not well recognised. It was first studied systematically by Hellström *et al*² in 1958. Since then various authors³ have reported hypertension to be present in between 10% and 60% of patients with primary hyperparathyroidism. The wide variation in these figures may be due to different criteria for selection of patients and defining hypertension.

At present the mechanism of parathyroid hypertension is not clear. The two conditions appear together too frequently to be due to a chance association. Avioli⁴ has suggested that hyperparathyroidism is 10 times more frequent in patients with otherwise "essential" hypertension than in the general population. Conversely, another recent case-control series¹ reported a higher chance of hypertension in patients with primary hyperparathyroidism. The known links between hyperparathyroidism and other endocrinopathies, including pheochromocytoma, Cushing's syndrome, Conn's syndrome, and acromegaly, are too rare to be blamed for the hypertension. So can the association be explained?

The hypercalcaemia of hyperparathyroidism is known to cause renal damage, and this might be blamed for the raised blood pressure. Hellström and colleagues² reported a good correlation between the severity of renal impairment and the presence of hypertension. Some authors^{2,5} have suggested that preoperative renal function might be an important determinant of the response of the blood pressure to parathyroidectomy. Other studies,^{6,7} however, have shown that hypertension does not correlate closely with renal damage and that renal function and blood pressure do not improve after surgery and may even become worse—or

impairment may develop for the first time. The part played by the renin-angiotensin system in the pathogenesis of parathyroid hypertension is equally controversial. Brinton and colleagues⁸ found raised plasma renin activity in four patients with hyperparathyroidism and hypertension. Serum calcium concentrations and the plasma renin activity returned to normal in all four patients after surgery; the blood pressure also returned to normal in three and decreased considerably in the fourth. On the other hand, Zawada and colleagues⁹ claim that hypertension associated with hyperparathyroidism is not dependent on renin. They showed that infusion of the competitive angiotensin antagonist saralasin failed to lower the blood pressure in all of their five patients including three with high plasma renin activity.

Calcium plays an essential part in the excitation and contraction of both striated and smooth-muscle cells.¹⁰ Studies in both animals and man^{11,12} have shown that intravenous infusion of calcium has a peripheral vasoconstrictor effect, and increased peripheral resistance may play a part in the hypertension associated with primary hyperparathyroidism. Experimental evidence¹³ also shows that the release of catecholamines is calcium dependent. Higher concentrations of catecholamines and their metabolites have been reported in patients with primary hyperparathyroidism together with increased vascular reactivity to noradrenaline infusions. This effect was most pronounced in patients with hypercalcaemia who also had hypertension. Enhanced sympathetic activity might therefore account for the raised blood pressure in hypercalcaemic patients. The inconsistencies in reports of the effects of parathyroid surgery on the blood pressure, despite the drop in serum calcium concentrations, suggest that the calcium itself is unlikely to be a pressor agent.

Raised concentrations of parathyroid hormone cannot be blamed for the hypertension, as administration of the hormone and its end terminal 1-34 fragment is associated with a fall in blood pressure.¹⁴ Again, a fall in hormone concentrations after surgery does not correspond to a fall in blood pressure.

An interesting hypothesis implicating skeletal sodium suggests that in hyperparathyroidism sodium is liberated from skeletal stores, so raising the blood pressure in susceptible subjects.¹⁵ Possibly this sodium mechanism may complement the sympathetic activity.

Though hypertension is undoubtedly common in hyperparathyroidism, this does not explain the association of hypercalcaemia with high blood pressure in epidemiological studies. Bulpitt *et al*¹⁶ found a positive correlation between the serum calcium concentration and systolic blood pressure in the Whitehall study. A link between blood pressure and calcium is further supported by Kesteloot and Geboers,¹⁷ whose epidemiological study of Belgian soldiers showed an independent and highly positive correlation between serum calcium concentrations and both systolic and diastolic pressure. In this case, however, they could be sure that the relation was not influenced by the use of thiazide diuretics in hypertensive patients, but a study in Renfrew, Scotland,¹⁸ has supported the relation between calcium and blood pressure in a population not using thiazide diuretics. This association between serum calcium concentrations and blood pressure in the population is particularly surprising as blood pressures are generally lower in populations resident in areas supplied with drinking water with high calcium content.¹⁹

The links between the parathyroids and hypertension and the part played by calcium in the pathogenesis of essential hypertension seem likely to receive more attention in the next few years. More information is needed on the place of surgery