extensor apparatus, whereas the dashboard fracture is less an injury of the extensor apparatus than a violent contusion of the components of the knee-joint. (2) Absence of appropriate primary treatment: The demands of complicating life-endangering injuries often dictate the nature and timing of treatment. (3) Age of the patient. Apart from the Service patients, whose average age was 25 years, the youngest of this series was 32 and the oldest 66, the average being 45. The age of the patient in a gross injury of the knee-joint influences the result; the only perfect result of a comminuted fracture of the lower pole occurred in the youngest patient.

Discussion

The road accident problem of Britain is largely that of collision between car and pedestrian or cyclist; the American problem is of collision between car and car (*Autocar*, 1949). The fracture which has been described has been recognized for many years as a clinical entity in America, and, according to Ferciot (1939), is being encountered with increasing frequency. Most orthopaedic surgeons will confirm a similar tendency in this country.

The injury has been named "dashboard" fracture; but it can be produced by impact with any rigid projection, such as glove-tray, umbrella handle hand-brake, and radio set or heater controls which oppose the front seat; and the victim of a collision who escapes with nothing more than a fracture of the patella is fortunate. The knee injury, however, is often only one of multiple injuries sustained in a similar manner; but it is the well-defined mode of production which is important, for it serves to illustrate a mechanism of injury in automobile accidents which merits serious consideration.

The orthopaedic surgeon called upon to treat the results of collisions at all hours of the day and night may be excused if he shows a professional interest in certain features of automobile engineering which have received little attention from the industry and none from the public. It should be possible to prevent, or at least to modify in degree, the variety of injuries produced by violent contact with the dashboard, windscreen, and steering wheel; suggestions to this effect have come from the profession in the past (Ferciot, 1939; Woodward, 1948). There is something wrong with a design which produces in the same patient a bilateral fracture of the patella with the edge of the dashboard and a bilateral compound fracture of the upper third of the tibia with the sharp edge of the glove-tray located immediately below.

Must the dashboard be rigid? Need it be studded with projecting knobs, switches, and glass-faced dials to break the teeth and disfigure the face of a child when the driver brakes in an emergency? Can it not be sprung or hydraulically mounted, padded with rubber of suitable consistency, and generally made less dangerous? Is the rigid glass windscreen which produced head or facial injuries in half the patients of this series essential? Is a plastic windscreen which ejects on contact, said by Woodward (1948) to be fitted to one make of American car, impracticable? If the steering column can be made adjustable for angle and length, and the wheel to absorb road shocks, could they not also absorb a blow from the chest rather than inflict fractures of sternum and ribs and grave injuries on the vital underlying structures? Is the compulsory fitting of safety-belts a fantastic idea; or is it just a matter of time?

These are but a few of the many possibilities which suggest themselves in the course of investigating the causes and complications of a particular injury. Such safety measures could have no effect on the incidence of road accidents; but they could exert a favourable influence on morbidity and residual disability.

Summary

The mechanism of injury in dashboard fractures of the patella is described.

Twenty-five fractures are reviewed in regard to type, complications, treatment, and result.

The difference between "dashboard" and "ordinary" fractures is stressed.

Suggestions are advanced for the protection of the front-seat occupants of a car from dashboard fractures and associated injuries of similar mechanism.

REFERENCES

- Autocar*, 1949, April 15, p. 340.
Ferciot, C. F. (1939). *Amer. J. Surg.*, 44, 139.
Woodward, F. D. (1948). *J. Amer. med. Ass.*, 138, 627.

HEART FAILURE IN CHILDREN WITH ACTIVE RHEUMATIC CARDITIS

BY

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Heart failure is generally regarded as not uncommon in children with rheumatic carditis, and it is accepted as important evidence that that carditis is active (Bland and Jones, 1951). Differing views have been expressed on the efficacy of digitalis in its treatment. Thus Schwartz and Schwedel (1930) found no response in 12 cases, whereas Sutton and Wyckoff (1931) noted improvement in all their 22 cases. In the more recent series of Walsh and Sprague (1941) there was a beneficial effect in 15 cases but no response in 20.

In our experience at Taplow, heart failure in rheumatic carditis has been uncommon, but it has usually responded well to digitalis. The differences between our findings and those of others may be due to the distinction we make between pericardial effusion and heart failure: abnormal signs may easily be attributed to heart failure, whereas they are in fact due to effusion. The distinction may be difficult, for in both conditions there may be dyspnoea with reduced vital capacity, increased jugular venous pressure, and enlargement of the liver. Furthermore, pericardial friction may not be heard in effusion, the electrocardiogram may be normal, and the radiological appearances may be similar to those of dilatation. The question is of more than academic interest, however, for the prognosis of effusion is better than that of failure: of 23 patients with effusion and no evidence of failure seen here during the last four years, four died; of seven with failure, three died.

The diagnosis of effusion was confirmed in 11 out of 23 cases: by aspiration of fluid in one, by radiography after the passage of a catheter (Wood, 1951) in six, and at necropsy in four. These 11 cases and the remaining 12 of the pericardial effusion group presented a single complex of clinical and radiological signs. The seven cases showing failure presented another and different complex, and the absence of fresh pericardial effusion was confirmed at necropsy in three. The object of this paper is to delineate the two complexes of signs which characterized these two groups, according to the classical method for the definition of clinical syndromes.

Salient Features of Pericardial Effusion

The clinical and radiological signs of rheumatic pericardial effusion have been described previously (Thomas,

Besterman, and Hollman, 1952; Besterman and Thomas, 1952). The present series of 23 cases includes 11 from the earlier one and 12 that have been seen recently. Effusion may develop at any time during the course of the disease, and not infrequently in the early stages of a first attack. Fever—100–104° F. (37.8–40° C.)—at the onset is invariable. Tachypnoea is common; it occurred in all cases of our earlier series, though not in all those of the later one. Chest pain is usual, but often of short duration. The jugular venous pressure is raised at the onset unless the effusion is small; enlargement of the liver frequently occurs later. Abnormal signs at the lung bases are almost invariable; they are most pronounced on the left side, develop early, and are due usually to pleural effusion. Diminution in intensity of the apex beat and dullness beyond the apex beat are much less common. The sedimentation rates are invariably high and in most cases there is a leucocytosis. The most striking radiological signs are rapid changes in heart size and shape; in 13 cases in which skiagrams had been taken at frequent intervals the cardio-thoracic ratio (C.T.R.) increased from a mean of 54% to 68% over a period of 11 days.

The Findings in Seven Episodes of Heart Failure

By contrast, the findings in seven episodes of what I believe to have been failure, in all of which there was raised venous pressure and enlargement of the liver, were as follows: subnormal temperature, 95–97° F. (35–36.1° C.)

in six and 97–98° F. (36.1–36.7° C.) in one; a sedimentation rate consistently below 10 mm. per hour in four and falling from high to lower levels in three (from 60 to 11, from 60 to 30, and from 80 to 35 mm. per hour); rales at the lung bases in five (three had hilar congestion on x-ray examination, but none had pleural effusions); peripheral oedema in two; ascites in two; leucocytosis in none. In three cases the skiagrams showed no change in heart size, in three there was a slight increase (the C.T. ratio increased from 57 to 61%, from 66 to 71%, and from 66 to 72%), and in one a decrease during digitalis therapy; in none was there any rapid change in size or shape as had been observed in effusion.

A gain in weight of more than 1 kg. occurred in only one episode (2 kg. over two weeks); there was obvious peripheral oedema and ascites. In another with oedema and ascites, however, there was no weight gain. In two there was a gain during recovery from failure (2 kg. over seven weeks in each); this gain was maintained and could be accounted for by resumption of growth and reaccumulation of fat and muscle with subsiding rheumatic activity. It is thought, therefore, than an absence of gain in weight does not exclude the diagnosis of failure in active rheumatic carditis, for a gain due to the water and salt retention of failure may be balanced by a loss of tissue due to inanition and increased metabolism; substantial gains occur only when fluid retention is gross enough to cause peripheral oedema. All seven episodes were treated with digitalis; in four there was a good response (Fig. 1), though in two digitalis therapy had to be supplemented by low-salt diet and mersalyl; in the other three there was some initial response, but signs of failure later recurred despite digitalis, and death ensued within two to six weeks.

Failure occurred only in patients with well-marked valve lesions and cardiac enlargement, and usually after long-continued rheumatic activity or in second attacks. Thus five had had continued activity for between twelve and eighteen months in a first known attack, and one for nine months in a second. The remaining patient went into failure ten weeks from the onset of her first known attack, but this was a particularly severe one and death occurred after four and a half months.

FIG. 1.—Response to digitalis in case diagnosed as having heart failure. Prompt rise in temperature and fall in pulse rate and venous pressure; later, diminution in size of liver and disappearance of ascites.

Comparison of the Findings in 7 Cases with Heart Failure and 23 Cases with Pericardial Effusion

	Months of Preceding Rheumatic Activity			Temperature	E.S.R.	Leucocytosis (Above 13,000)	Oedema	Ascites	Pleural Effusion	Rapid Changes in Heart Size and Shape	Hilar Congestion	Response to Digitalis Therapy
	0-1	2-6	6+									
Heart failure (7 cases)	0	1	6	95-97° in 6, 97-98° in 1	Normal or fell in 7	Nil	+ in 2	+ in 2	Nil	Nil	+ in 3	+ in 4
Pericardial effusion (23 cases)	17	5	1	101-104° in 23	Raised in 23 (above 100 in 12)	+ in 21/23	Nil	Nil	+ in 22	+ in 21/21	Nil	Nil

Heart Failure Precipitated by Hormone Therapy

Two patients developed clinical and radiological signs similar to those described above as indicating failure during hormone therapy for prolonged rheumatic activity; in both, these signs were attributed to heart failure due to water and salt retention. Within two days of starting treatment the venous pressure rose, the liver became enlarged and pulsatile, the temperature fell to 95° F. (35° C.), and the sedimentation rate dropped from 52 to 3 mm. an hour in one case and from 55 to 12 mm. an hour in the other. Peripheral oedema developed later in both, and in one a chest x-ray film showed hilar congestion. One patient responded well to injections of mersalyl; hormone therapy could then be continued without further signs of failure. The other showed little response to mersalyl alone but had a good diuresis on digitalis and mersalyl; an even better diuresis was obtained when an ion-exchange resin ("zeo-karb") was added. Frequent skiagrams throughout these two episodes showed no significant change in heart size. The findings on which these two patients were regarded as having failure, as distinct from those that might have occurred in effusion, were the subnormal temperature and fall in sedimentation rate, the liver pulsation, the ascites and hilar congestion in one case each, the absence of any sudden change in heart size or shape, and the good response to treatment. Both had well-marked murmurs and considerable cardiac enlargement at the start of therapy.

The points on which I believe pericardial effusion may be distinguished from heart failure are shown in the Table. They may be summarized as follows. Effusion may develop at any time and not infrequently within four weeks of the onset of the first known attack, whereas failure usually occurs only in patients with well-marked heart lesions and after long-continued activity; the temperature and sedimentation rate are invariably high in effusion, whereas in failure the temperature is subnormal and the sedimentation rate falls; signs of fluid at the lung bases are common in effusion but rare in failure; peripheral oedema and ascites may occur in failure but not in effusion; skiagrams show rapid changes in heart size and shape in effusion but no such rapid change in failure; hilar congestion is not uncommon in failure but does not occur in effusion; there is often a good response to digitalis in failure but no response in effusion; a leucocytosis is common in effusion and rare in failure.

These differences may be further illustrated by reference to two cases; one patient was admitted on two separate occasions, initially with failure and later with pericardial effusion; the other was first diagnosed and treated as heart failure, but was later proved to have had effusion as well.

Case 1

A girl of 7 was admitted four months after the onset of her first known attack. Despite the short history, she had loud murmurs and marked cardiac enlargement and showed dyspnoea, a raised venous pressure, enlargement of the liver, and ascites; the temperature was subnormal and the sedimentation rate low (3 mm. an hour). She was treated with digitalis, to which there was a good

response within three days, but skiagrams showed only a 1-cm. reduction in heart size over the following ten weeks (Fig. 2, A and B). This episode was therefore regarded as heart failure.

On her second admission she also showed a high venous pressure and enlargement of the liver, despite previous maintenance doses of digitalis. This time, however, her temperature and sedimentation rate were high, there were signs of fluid at the lung bases, and she had a polymorphonuclear leucocytosis; there was no ascites. During the next eighteen days the temperature, sedimentation rate, and venous pressure fell, and skiagrams showed a reduction in

FIG. 2.—Case 1. Contrast in change in heart size in heart failure and effusion. Heart failure: A, September 21, 1949, td./id. (transverse diameter of heart-internal diameter of chest) = 14.5/21.5; B, November 30, 1949, td./id. = 13.5/21.5; reduction in heart size of only 1 cm. over ten weeks. Effusion: C, November 21, 1950, td./id. = 16.5/21.0; D, December 9, 1950, td./id. = 14.5/21.0; decrease of heart size of 2 cm. over 18 days.

FIG. 3.—Case 2. A, August 25, 1952 (td./id. = 11.5/21.5). Moderate generalized enlargement and slight hilar congestion. B, September 11, 1952 (td./id. = 14/21.5). Marked increase in heart size due to development of pericardial effusion; also small left pleural effusion. C, September 25, 1952 (td./id. = 14/21.5). Hilar congestion but no further increase in heart size after eight days on cortisone. D, December 19, 1952 (td./id. = 11.5/21.5). Return to previous heart size and shape from resolution of effusion.

heart size of 2 cm. (Fig. 2, C and D). It is thought that this episode was due to pericardial effusion and not to heart failure.

Case 2

A boy of 10 was admitted four weeks after the onset of his first known attack of rheumatic fever. He had loud murmurs, and a skiagram showed moderate generalized enlargement and slight hilar congestion (Fig. 3, A). The temperature was 103° F. (39.4° C.), sedimentation rate 134 mm. per hour, and white count 19,000, with 80% polymorphs; the venous pressure was raised, the liver was enlarged, and there were rales at the lung bases. In view of the hilar congestion and the absence of radiological evidence of effusion, he was thought to have failure and was put on to digitalis. Four days after admission he developed a left pleural effusion. There was then some slow improvement in his general condition, but his venous pressure was still raised after eighteen days, when digitalis had to be stopped as there was electrocardiographic evidence of first-degree heart-block. His slow improvement continued despite stopping digitalis, and his venous pressure returned to normal seven days later.

A review of his chest skiagrams at this time showed that a pericardial effusion had developed during the first two weeks of admission; the heart size had increased by 2.5 cm. over 17 days (Fig. 3, A and B). There is little doubt that many of his abnormal signs had been due to this effusion and not to failure, and that this accounted for the high fever, raised sedimentation rate, and polymorph leucocytosis at the onset, and, later, the signs of fluid at the left base and failure to respond to digitalis. One week later he was put on to cortisone on account of persistent signs of rheumatic activity. On the eighth day of starting treatment he became dyspnoeic, the liver became enlarged, the venous pressure rose, and there were many coarse rales throughout both lungs; the sedimentation rate and temperature were normal and a skiagram showed marked hilar congestion but no change in heart size (Fig. 3, C).

A diagnosis of heart failure precipitated by sodium retention was made; he was given mersalyl at once and put back on to digitalis. There was a good response to mersalyl; 48 hours later the clinical signs of failure had disappeared and there was no radiological evidence of congestion, though the heart size remained unchanged. It was then possible to continue cortisone therapy, but he was kept on digitalis until 14 days after the hormone had been discontinued; at no time did any further signs of failure develop. Skiagrams later showed a gradual resolution of the effusion, and the heart returned to the same size and shape as it had been on admission (Fig. 3, D).

Summary and Conclusions

Heart failure in rheumatic carditis is thought to be less common than has been previously supposed; in many cases the signs of so-called heart failure are due to pericardial effusion. The ways of distinguishing between heart failure and pericardial effusion are discussed. Chief among them are high fever, raised sedimentation rate, and polymorph leucocytosis in effusion as compared with subnormal temperature, low sedimentation rate, and normal white blood count in failure; peripheral oedema, ascites, and sometimes liver pulsation in failure but not in effusion; rapid changes in heart size and shape in effusion but little change in failure; and a good response to digitalis and other treatment in failure and no response in effusion. Heart failure is rare except in patients with well-marked murmurs and cardiac enlargement who have often had long-continued preceding rheumatic activity, whereas effusion may occur when there is little or no other evidence of heart involvement and not infrequently within four weeks of the onset of a first attack.

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REFERENCES

- Besterman, E. M. M., and Thomas, G. T. (1952). *Brit. Heart J.*, 15, 113.
Bland, E. F., and Jones, T. D. (1951). *Circulation*, 4, 836.
Schwartz, S. P., and Schwedel, J. B. (1930). *Amer. J. Dis. Child.*, 39, 298.
Sutton, L. P., and Wyckoff, J. (1931). *Ibid.*, 41, 801.
Thomas, G. T., Besterman, E. M. M., and Hollman, A. (1952). *Brit. Heart J.*, 15, 29.
Walsh, B. J., and Sprague, H. B. (1941). *J. Amer. med. Ass.*, 116, 560.
Wood, P. (1951). *Brit. Heart J.*, 13, 574.

THE GENERAL PRACTITIONER AND THE CHILD WITH HEART DISEASE*

BY

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It has long been recognized that certain medical problems can be tackled more satisfactorily by a team than by a man working on his own. As early as 1888 the British Medical Association published the results of a collective investigation into acute rheumatism, and included an inquiry into the influence of home and social conditions (Whipham, 1888). The value of joint studies has also been recognized by the College of General Practitioners, which is actively fostering research and seeking to provide facilities for its prosecution.

With this encouragement it is tempting to face the challenge written in 1952, as a commentary on the first European Congress of Cardiology. "Judging by the papers at the Congress, cardiology is more concerned with 'ends' than with 'beginnings' of disease; for, apart from one paper on the inheritance of benign essential hypertension, aetiology was ignored. . . . If research were planned logically, then the major effort would centre round the aetiology and prevention of rheumatic heart disease, atherosclerosis, and hypertension" (*Lancet*, 1952). And, to quote from a more recent leading article on congenital heart disease, "Surgery has improved the chances of these patients; but even so the outlook is gloomy enough to make us want to know more about causation" (*Lancet*, 1953).

Our surgical colleagues have made possible remarkable advances in the treatment of cardiovascular disease, but we must not allow their brilliant achievements to upset our sense of proportion. Only a small fraction of children with congenital heart disease can benefit from surgery. The same is true of patients with rheumatism. When 1,000 rheumatic children were observed for a period of 20 years, 117 developed mitral stenosis, but only 12 of these had significant pulmonary hypertension (Bland and Jones, 1951), and none needed valvotomy. The problem of rheumatism is medical, not surgical, for after 20 years 301 of the 1,000 patients were dead (87% of the deaths being directly or indirectly attributable to rheumatism) and another 139 were more or less severely crippled by their cardiac disability.

To the general practitioner heart disease seems to begin at 40, since until then defects of function are compensated by the body's powers of resistance; afterwards cardiovascular disease becomes increasingly manifest (R. J. F. Pinsent, 1954, personal communication). This is not wholly the result of essential hypertension and atherosclerosis, for the Registrar-General's returns

*A paper given before the Birmingham Division of the B.M.A. on February 16, 1954.