CEREBRAL EMBOLISM FOLLOWING CONTUSION OF THE HEART

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Damage to the heart resulting from non-penetrating blows to the chest wall is not rare, and several series of these cases have been recorded (Barber, 1944; Bright and Beck, 1935; Kahn and Kahn, 1929; Warburg, 1940; Hawkes, 1935; Arenberg, 1943; Sigler, 1945). The heart and pericardium, lying close behind the chest wall, are very vulnerable to severe blows in the region of the praecordium, but generally any damage which may be caused is mild and not recognized. In children and youths, however, there is perhaps a greater chance of cardiac trauma than in adults, because the heart is behind a resilient sternum and can be the more easily compressed against the spinal column. Although many cases of cardiac damage have been described we have not been able to find a case recorded in the literature in which a cardiac contusion was followed shortly afterwards by a cerebral embolism. As cardiac trauma may not be recognized as a cause of unexplained hemiplegia in childhood, since the condition has not been described before, and as several points arise from it, the following case is described in detail.

Case Report

The patient was a very robust, well-developed schoolboy aged 15½, who weighed 11 st. 5 lb. (72.12 kg.).

He had lived in Tanganyika until he was 5, and whilst there had had several attacks of fever. He had otherwise been healthy, and in September, 1946, March, 1947, and March, 1948, had been found fit in routine school medical examinations. There was no history suggesting rheumatic fever.

The first incident occurred about one year before the present illness. In March, 1947, when he was boxing at school in a competition, the fight had to be stopped in the first round as he was in severe pain following concentrated punishment to the region of his heart. This pain, which was dull and aching in character, continued for the next week or so. It was aggravated by exercise, and the patient remembered that he had to stop in a cross-country run and walk home because of the severe dyspnoea and pain in his left chest which the exercise had produced. After this there was no recurrence of the pain, and for the next year he played the various school games, including football, with some distinction and without restriction.

He next took part in competitive boxing on March 29, 1948, when he had to fight the boy who had beaten him so thoroughly

the previous year. Again he received severe punishment to the left chest, and at the end of the first round he complained to his seconds of dull and sickening pain over his heart. The fight was stopped in the second round, as the boy was in agony. He thinks that he received only one glancing blow to the left side of his head, and he was in no way dazed or confused. The dull pain persisted, and some hours later he noticed a sharp pain, which from his description was pleuritic in type, over his praecordium. He slept badly that night, but the next morning the pain was less, though he felt generally ill and very stiff. He took things quietly, as he found that exercise brought on the pain. On March 31 he had improved considerably, but was still afraid to exert himself. During that afternoon he was sitting watching the boxing finals, when he became aware that his left leg had suddenly become dead below the hip and that he felt very faint. Two hours later his arm became affected, and shortly afterwards he had a general convulsion followed by confusion and headache. No further convulsion occurred, and during the next few days his condition was stationary.

He was admitted to St. George's Hospital five days after the injury. The boy was alert, right-handed, intelligent, and co-operative. The skull was normal in shape and was not tender. Neck movements were full and there was no limitation of straight leg raising. The cranial nerves were perfectly normal, and in particular there was no papilloedema and no field defect. Speech was normal. On the affected left side the leg

was very spastic, there was ankle clonus, tendon jerks were increased, and there was an extensor plantar response. Voluntary movements and co-ordination were considerably impaired in the leg. The arm was similarly affected, but to a much smaller degree. Sensation was normal to full testing. The pulse rate was 80 and was irregular. The blood pressure was 130/70. There was no clinical enlargement of the heart. The heart sounds were distant and difficult to hear. A soft systolic murmur was heard over the mitral area, and a basal pericardial rub. Blood count was normal: red cells, 5,250,000; white cells, 6,800 per c.mm., with normal differential count. B.S.R., 2 mm. in 1 hour (Westergren).

The blood Wassermann and Kahn reactions were negative. X-ray photographs of skull, heart, ribs, and lung fields all showed no abnormality.

The electro-encephalogram on April 5 (Fig. 1, A) was abnormal. There was a low-voltage alpha rhythm of 10 c/s that underlay a generalized symmetrical abnormality which consisted of runs of high voltage 1 c/s and medium voltage 3-4 c/s. There was no evidence of focal abnormality, nor of one side being worse than the other. An electrocardiogram taken at the same time was also abnormal. The curves showed evidence of partial heart-block due to two distinct lesions. First, there was an occasional omission of auricular systole, though the auricular beats were for the most part regular. This would indicate some partial sino-auricular block. Secondly, the P-R interval was prolonged and variable (Fig. 1, B). It varied from 0.2 to 0.4 second in length; which would indicate partial disturbance of conduction of the auriculo-ventricular system. The ventricular rate was 84. The terminal deflections in the three limb leads were normal.

FIG. 1.—(A) April 5, 1948. E.E.G. abnormal. A low-voltage alpha rhythm of 10 c/s underlies a generalized symmetrical abnormality consisting of runs of high-voltage 1 c/s and medium-voltage 3-4 c/s. (B) Electrocardiogram recorded from lead I on the electro-encephalogram. The E.C.G. shows evidence of sino-auricular block and varying conduction time. The paper speed and amplification in Fig. 1 were used for all the E.E.G. records.

He was kept in bed and given occupational therapy, with resultant improvement in the power of his left upper limb after a week, though the leg was still very spastic. The cardiac arrhythmia persisted, but the heart sounds improved in quality. The systolic murmur increased in intensity; it became harsh and occupied almost the entire systolic period. It was audible all over the praecordium, and was maximal in the left third and fourth interspaces $2\frac{1}{2}$ in. (6.25 cm.) from the mid-sternal line. The pericardial rub was heard only during the first two days in hospital.

An E.E.G. taken on April 13 showed a considerable change. Whereas there were generalized slow waves in the record taken on April 5, these persisted in the left hemisphere and there was suppression of all waves in the right parietal region (Fig. 2).

FIG. 2.—April 13, 1948. E.E.G. abnormal. Persistence of abnormal slow waves in the left hemisphere and suppression of all waves in the right parietal region. Recording as in Fig. 1.

On May 4 an E.E.G. showed a slight improvement. A low-voltage dominant frequency of 12 c/s had returned. There were more 6 c/s waves on both sides, and runs of 3 c/s were seen which were more evident on the right than on the left. The records were still far from normal, but showed slight and gradual improvement (Fig. 3). Further E.C.G. recordings were

FIG. 3.—May 4, 1948. A low-voltage dominant frequency of 12 c/s is returning: 6 c/s are seen on both sides and runs of 3 c/s were more evident on the right than on the left. The records are still far from normal. Recording as in Fig. 1.

made, and these showed very little change. On May 4 an intramuscular injection of 1/50 gr. (1.3 mg.) of atropine sulphate caused an increase of pulse rate to 120, the rhythm became normal, and the P-R interval shortened to 0.16 second.

The hemiplegia continued to improve so that he was fit to be discharged from hospital on May 14. Since then his convalescence has been controlled and uneventful.

An E.C.G. taken on June 10 by Dr. Alastair Hunter (Fig. 4) was reported by him to show partial heart-block of varying character without the usual Wenckebach's periods. The auricular and ventricular complexes appear normal, but the auricular rate was not regular. It appeared, therefore, that

there was a disturbance of sino-auricular activity as well as A-V conduction. On Aug. 19, nearly five months after the injury, examination showed that improvement still continued. There were only residual signs of the hemiplegia, which caused him very little disability. The E.E.G. showed a further slight improvement, the records from the hemispheres being symmetrical. The heart was not enlarged, the sounds were of

FIG. 4.—June 10, 1948. E.C.G. showing disturbance of sino-auricular activity as well as A-V conduction.

good quality, and no murmurs were heard. The pulse was still irregular, rate 78. The E.C.G. still showed sinus and A-V block, but the P-R interval was shorter than had been seen previously and varied between 0.18 and 0.22 second. He was fully ambulant and was due to start on a sedentary engineering course of instruction.

Discussion

If a non-penetrating blow to the chest is severe enough there may be various cardiac sequelae. The commoner of these include pericarditis, myocardial damage including coronary thrombosis and rupture of the heart, alterations of rhythm, and disturbances of conduction. The clinical picture which results may in severe cases be similar to that seen in myocardial infarction and may be accompanied by the corresponding physical signs and electrocardiographic changes. Injuries of this type have been produced whilst playing various sports. These include cricket (Priest, 1939), football (Lee, Ussher, and Houck, 1943), steeplechasing (Anderson, 1940), golf ("Queries and Minor Notes," 1933), boxing (Jokl, 1941), and surf-bathing (Leinoff, 1940-1). Jokl (1941) reports that professional boxers have found that the effects of well-placed hooks to the heart last for a considerable period, usually for the rest of the fight. He also considers that chest injuries in boxers are very much more common than is generally assumed. In many cases there is no evidence of fracture of the thoracic cage, and there may be no bruising of the chest wall. Kellert (1917) cites the case of a man engaged in digging in a sandbank when it suddenly caved in and he was engulfed up to his waist. When dug out he was dead. A laceration 5 cm. in length extended through the interventricular septum. Rupture of the heart was described by Hamilton (1934) in a child of 7, and by Bilderbeck (1919) in a young Gurkha rifleman without any other evidence of trauma. Barber and Osborn (1941) recorded a large contusion of the left ventricle which caused death in five hours and was the only lesion.

Experimental work has been performed on dogs by Bright and Beck (1935), Moritz and Atkins (1938), and Kissane, Fidler, and Koons (1937). The last-named authors demonstrated that the most frequent cardiac lesions seen after injuries to the chest were subendocardial and subpericardial haemorrhages. Necropsies on human cases have shown tears and contusions of the auricles. Barber and Osborn (1941) obtained a heart which had subendocardial contusions following cardiac massage by a surgeon. Osborn (1943) described bruising on the posterior aspect of the right auricle near the entrance of the inferior vena cava,

tending to spread upwards. This type of lesion is of considerable significance in relation to disorders of rhythm, as it can readily spread towards the sino-auricular or auriculo-ventricular nodes. In the case in which death followed a blow from a cricket ball (Priest, 1939) the anterior surface of the heart was oedematous and bruised and there was a tear near the interventricular septum. In many cases the damage to the myocardium results in electrocardiographic changes similar to those of coronary thrombosis, though this is not invariable. Barber (1942) found an abnormal terminal complex in 12 out of 20 accident cases. Other changes were inverted P waves, supraventricular tachycardia, slurred R waves, and partial heart-block. In one case the P-R interval was 0.35 second five months after an accident. Rosenson (1924) recorded the case of a child of 10 who developed heart-block following a hard punch with the fist over the praecordium inflicted by one of his playmates, the E.C.G. returning to normal three months later. Wood (1948) describes the case of a woman aged 36 who had previously been healthy. In 1941 she was buried for one hour by a bomb explosion, and was almost asphyxiated. When examined shortly afterwards she was found to have a tremor in her left arm and also complete heart-block, both of which disabilities have proved to be permanent. Coffen, Rush, and Miller (1941) described the case of a patient aged 21 with auriculo-ventricular dissociation which developed after a chest injury received at the age of 3. Tuohy and Boman's case (1931) had bundle-branch block.

In the present case, a healthy boy received repeated heavy blows to his chest wall, following which signs and symptoms of cardiac damage were noted. These included severe pain in his left chest, pericarditis, a loud systolic murmur which was barely audible one month later, and evidence of partial heart-block. It is very probable that his heart had been bruised one year previously and was more susceptible to damage as a result of this. It is unlikely that both the sino-auricular and the auriculo-ventricular block were present before the injury. He had been medically examined previously on three occasions, and no comment was made about his cardiovascular system. The prognosis for the cardiac condition is uncertain. The diminution in the degree of heart-block following injection of atropine is a favourable finding, as is also the shortening of the P-R interval during the five months the boy has been under observation. Bruenn (1937) studied 22 patients with acute rheumatic fever who had impairment of auriculo-ventricular conduction time and found that intravenous injection of atropine sulphate abolished the conduction defect in 19. Acceleration in ventricular rate did not necessarily parallel decrease in conduction time. He concluded that in acute rheumatic fever the impairment of auriculo-ventricular conduction is due, in part at least, to an increase in "vagal tone" and that the site of this disturbance lies in the medulla.

There can be no proof of the site of origin of the embolus which caused the hemiplegia in this case, but in view of the other cardiac lesions it is possible that a small piece of a mural thrombus became detached. As the main lesion was to the right auricle, for this to happen there must either have been a spread of the bruising through to the left auricle or else a septal defect. A localized pulmonary lesion is another possibility. In favour of this is the pleuritic type of pain which he had during the first few days after the blows, but against it are the negative x-ray appearances and absence of other symptoms of a pulmonary lesion. The third possibility is one of fat embolism from bony damage, but very careful radiology of the ribs and sternum showed nothing suggestive of fracture. The hemiplegia, from its character, was clearly the result of an embolus, and the nature of the E.E.G. supports this view.

It is impossible to escape the conclusion that the embolus was the direct result of the myocardial damage which was so clearly demonstrated in the E.C.G., and that the actual cause of the embolus was a mural clot. The story of cardiac damage was obtained only by careful questioning after other causes for the hemiplegia had been excluded. The patient was an intelligent, observant boy, and the circumstances of his injury are dramatic. It is likely that the cardiac cause of other cases of hemiplegia in childhood may be obscured by the severity of the resulting hemiplegic picture. Although the E.C.G. produced striking changes in the present case, these are unusual, and if records are not made shortly after an injury evidence of it may be slight.

Sudden hemiplegia is not a common catastrophe in childhood. When not the result of direct brain injury, it usually occurs in the course of an acute infectious illness, particularly pertussis. Here it is the result of a haemorrhage, whereas in other infectious illnesses, such as pneumonia or diphtheria, it is thought to be embolic. Much attention has been paid in the past few years to cerebral venous thrombosis complicating infections or debilitating illnesses in children and giving rise to hemiplegia. Pyogenic infections, bacterial endocarditis, and blood diseases, also the cause of embolic hemiplegia, were excluded in the present case.

The most frequent cause of a sudden hemiplegia in an apparently healthy child is congenital heart disease, although, of course, the catastrophe is more likely to occur if bacterial endocarditis supervenes. There was nothing in the present case to suggest any congenital heart lesion; but the nature of the pathological process—an otherwise normal child with an abnormal endocardium, mural thrombus, embolus, hemiplegia—differed only in that the heart lesion was acquired. Our case suggests that when all causes of sudden hemiplegia in childhood have been considered the possibility of unsuspected cardiac trauma should be raised.

Summary

A case is reported of a boy who sustained myocardial damage while boxing, with a resulting cerebral embolus. The electroencephalographic and electrocardiographic findings are related to the clinical findings.

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A subcommittee of the Food Standards Committee of the Ministry of Food has been set up to consider the available evidence on the effect of ingesting foods contaminated with minute traces of metals or other injurious elements, and the possibility of prescribing limits for such contamination. A number of scientific bodies and research associations, including the Medical Research Council, are co-operating in the inquiry.