children fell into social class III. For convenience, classes I and II are added together, as their numbers are small, and classes IV and V are similarly joined.

The figures are set out to show a comparison between the social class of the duodenal ulcer group and the controls, and between the fathers of these two groups. In addition, the difference, where there was one, between the social class of father and son has been recorded as "Up" or "Down."

It will be seen that the social class of the duodenal ulcer patients is not significantly different from that of the controls, that the social class of the fathers of both groups likewise does not differ significantly, and that the movement from one social class to another that has taken place has been the same for both groups, the number of those that have raised their social status being approximately equal to those who have declined.

No attempt was made to classify them by actual occupations, as it was felt that the numbers concerned were too small for any conclusions to be drawn, and this has already been done very thoroughly by others.

Discussion

The findings suggest that there is no difference between people with duodenal ulcers and others in the size, composition, or social class of the family into which they were born, in their educational standard at school, or in illness in childhood. Neither do duodenal ulcer patients seem to be more fortunate or less fortunate than others in early separation from parental influence. In none of the points investigated could the findings of other authors be confirmed. It may well be that those points are irrelevant to the development of the "ulcer personality," and that investigation of other childhood features might have shown some important differences between the groups studied: it may be that factors operating in late adolescence or early adult life are responsible for the characteristics said to be shown by these patients. However, unless further investigations show some clear-cut differences between duodenal ulcer patients and the general population regarding factors operating before the appearance of symptoms, the possibility that the "ulcer personality" may be the effect rather than the cause of the disease must be considered.

Summary

In view of the possible aetiological importance of personality in duodenal ulcer a comparison was made between the childhood background of a group of 250 male patients with duodenal ulcer and a similar group of patients with other diseases. No difference could be found between the two groups in any of the points investigated.

I should like to express my gratitude to Dr. F. Avery Jones for his encouragement and inspiration in this work, and to Dr. Richard Doll for much useful advice and criticism.

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AN UNUSUAL CASE OF TRAUMATIC HEART DISEASE WITH CLINICAL RECOVERY

BY

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Cardiac damage, as the result of non-penetrating injuries or blows to the chest wall, has been gaining increasing recognition in the medical literature of recent years. That cardiac lesions of clinical and pathological significance may arise as the result of blows on the chest wall is a fact well established by the work of Barber (1938, 1940), Beck (1935), Warburg (1938, 1940), and others. There has been, however, an unfortunate tendency on the part of certain authors to attribute all sorts of perfectly natural and totally unrelated phenomena of the heart to preceding traumata of insignificant character. Such a tendency can deserve only our severest criticism.

In view of the unusual nature of the cardiac damage and its proved relationship to a previous non-penetrating injury to the chest wall, the following case report is considered worthy of publication.

Case Report

An artillery officer aged 36 has been under our observation since the middle of 1949. Except for childhood ailments like mumps, measles, and scarlet fever, and attacks of malaria and paratyphoid, all within the first decade of life, the patient had enjoyed extraordinarily good health until the time of the accident here described. He had always led an active or athletic life, indulging freely in boxing, swimming, hockey, and cross-country running. There was nothing in the past history to suggest any form of cardiac distress or disease. The family history was also negative, there being no cases of heart disease, sudden death, apoplexy, or high blood pressure.

About the beginning of April, 1948, whilst on a gunnery course at the School of Artillery, he was suddenly struck over the left side of the chest by the "perch handle" of a 25-pounder gun. The sudden impact caused a momentary loss of consciousness, followed by a moderate degree of pain in the chest. During the succeeding weeks he was unable to run or to exert himself on account of the pain; at night the pain used to worry him, necessitating the adoption, throughout the night, of a right lateral decubitus. After a period of three weeks, during which he continued massaging of the chest wall with "iodex," the pain seemed to decrease during the nights, but during the early mornings, when climbing a hill, he experienced a peculiar sense of constriction, which would compel him to stop; this feeling would pass off after a period of nausea and retching.

On two occasions he woke up in the middle of the night with a "strange feeling" in his chest and a profuse "cold sweat"; these feelings were relieved by vomiting. One evening, while cycling, the feeling of constriction in the chest became so intense that he could not get home in spite of all his efforts. A similar sense of constriction in the chest but of much greater intensity overtook him suddenly on the night of May 9, 1948, during a railway journey. The pain was intense enough for him to "scream for assistance." He was carried to a military hospital and given an injection of morphine, in spite of which the pain continued unabated until the night of the 11th. The pain, which was more intense on coughing and deep breathing, for the first time had been bilaterally symmetrical and agonizing.

and was accompanied by a "feeling of deadness" in the left arm and by restlessness, lassitude, and extreme weakness.

On or about June 15 he felt well again and was free of all pain. The systolic blood pressure and erythrocyte sedimentation rate, which had been 104 mm. Hg and 56 mm. respectively in June, 1948, had improved to 124 mm. Hg and 7 mm., respectively, by September of that year. From September, 1948, to August, 1949, there was steady improvement in health and he gradually resumed strenuous duties and arduous hours; there were, however, occasions when sharp and transitory pains were experienced, on and off, in the chest and back.

On August 29, 1949, he again felt a severe pain in the chest, particularly noticeable on coughing and deep breathing and accompanied by fever. He was granted a month's leave and advised to obtain expert advice on his case. An electrocardiogram taken on September 2 (by R. J. V.) showed "atypical right bundle-branch block of Wilson type" with prominent and slurred S waves in leads I, II, and CR₁. The Q wave was prominent in leads CR₁ and CR₂, with inverted T waves. All electrocardiograms taken subsequently have conformed more or less to the same general pattern.

A clinical examination carried out on October 25 was reported as follows:

"A well-built and athletic individual of sober habits and hypersthenic habitus. Teeth good, tongue coated, no engorgement of neck veins, no oedema, and no deformities of chest. The apex beat, neither visible nor palpable, was localized roughly by auscultation as being situated in the fifth left interspace along the mid-clavicular line; this was confirmed by percussion. The second heart sound was found accentuated in both the basal areas, whilst the first sound in the mitral area was both muffled and accompanied by a soft systolic bruit. No pericardial friction sounds were audible at the time. The pulse rate at the time of the clinical examination was recorded as being 54 a minute and the blood-pressure readings as 122 mm. Hg systolic and 76 mm. Hg diastolic. Examination of the other systems revealed nothing of importance except for a small degree of non-tender enlargement of the spleen."

Teleskiagrams were taken, with barium in the oesophagus, on October 6. The radiologist's report was as follows:

"Symmetrical thoracic cage of sthenic type. The straight film (postero-anterior) reveals a well-defined opaque line about 1½ in. (3.8 cm.) long, in the anterior axillary region above the upper borders of the fourth and fifth left ribs, suggestive of periosteal new bone formation. The ribs show normal shape and regular contours. Normal translucency of both lung fields; normal broncho-vascular markings. The cardiac silhouette, in straight P.A. view, shows normal size and shape and smooth outlines; the right lower arch shows normal configuration, and the right cardio-phrenic angle appears clear. Normal appearance of the cardiac silhouette in the right anterior oblique position. In the left anterior oblique view the posterior contour of the left lower arch appears somewhat flattened, with a tent-like angulation about one fingerbreadth above the diaphragmatic dome. Also seen in this view is a faint shadow, fairly well defined although less dense than the cardiac shadow, at the cardio-phrenic angle, in front of the right ventricle. The oesophagus, as visualized by barium swallow, shows normal course in all three views."

On fluoroscopy, it was noted that the irregularity at the sinistro-posterior contour of the heart, seen in the left oblique view, was a persistent feature not dependent on the cardiac pulsations. Also, the cloudiness observed in front of the right ventricle was present during both systole and diastole.

On the basis of the above findings it was concluded that a circumscribed pericardial adhesion existed in the region of the posterior aspect of the left ventricle as well as circumscribed mediastino-pericardial adhesions in front of the right ventricle.

The accompanying electrocardiographic tracings, recorded on October 7, 1949, conform closely to one of the varieties of so-called atypical or unusual form of right bundle-branch block, described by Wilson *et al.* (1932, 1934), by Bayley (1934), and by others. In accordance with their criteria, all the ventricular deflections of lead I are of low voltage with the last deflection of QRS directed downwards;

Electrocardiogram of the case taken on October 25, 1949.

also, in leads II and III there is a small R followed by a broad deep S, while T is upright. The QRS duration works out at 0.164 second. The electrocardiographic diagnosis of atypical right bundle-branch block suggested by the standard limb leads is fully substantiated by the precordial leads.

In the present tracing, although the limb leads fail to furnish any evidence of an associated myocardial infarct, the unipolar chest and extremity leads do suggest the co-existence of an anterior infarct. For instance, precordial lead V₂, which faces the epicardial surface of the right ventricle, instead of displaying the typical rsR' pattern of uncomplicated right bundle-branch block, shows a wide Qrs pattern with an elevated S-T segment and inverted T wave of coronary type. Such a change in pattern, according to Goldberger (1949), is strongly suggestive of an associated anterior infarction. This is substantiated by precordial leads V₃, V₄, V₅, and V₆, which show elevated RS-T segments and inverted T waves in association with QR patterns, facing as they do the epicardial surface of an "injured" left ventricle. Final evidence of anterior infarction is supplied by unipolar extremity lead aVL with its deep and wide QS pattern, elevated RS-T segment, and inverted T wave. The present tracing supports the contention of Rosenbaum *et al.* (1944) that, in the event of a right bundle-branch block and anterior infarction appearing conjointly, it is the precordial lead and not the standard lead of the tracing that clinches the diagnosis.

On October 13 the erythrocyte sedimentation rate at the end of one hour was 6 mm.; the red cell count was 5,250,000 and the leucocyte count 6,500 per c.mm. (59% polymorphs, 38% lymphocytes, 1% monocytes, and 2% eosinophils); and haemoglobin 15 g. per 100 ml. The blood urea worked out at 40 mg., whilst the urea clearance was 59% of average normal.

Since that time we have followed the patient up quite regularly. Although exercise tolerance has improved enormously and he is devoid of all symptoms of heart disease, the physical and electrocardiographic signs remain practically unaltered. Recently, on the suggestion of a colleague, the patient was received in hospital for a few weeks and subjected to a course of anticoagulant therapy with moderate doses of dicoumarol. Though the patient reported some degree of subjective improvement after that treatment, the objective findings remained the same.

Discussion

Experimental proof of cardiac damage or rupture as the result of non-penetrating injuries to the chest wall has been afforded by the studies, in dogs and rabbits, of Külbs (1909, 1937), Schlomka (1934), Kissane *et al.* (1937), and others. They have succeeded in inducing, by the use of controlled and measured traumata to the chest wall, various types of heart lesions, including haemorrhagic and necrotic lesions, cardiac failure, various forms of arrhythmias, and sudden death from ventricular fibrillation and cardiac arrest. Moritz and Atkins (1938) and Bright and Beck (1935) recorded examples of lesions resembling myocardial infarction in animals as the result of blows applied directly to the exposed heart.

Clinical proof of the existence of traumatic heart disease as a true entity is not far behind. In the clinical and pathological reports of Urbach (1922), Kahn and Kahn (1929), Tuohy and Boman (1931), Beck (1935), Barber (1938), and Warburg (1938, 1940), numerous observations are recorded which lend credence to the concept of heart disease arising secondarily to non-penetrating injuries of the chest wall.

The earlier references in the literature to the subject of traumatic heart disease were notoriously pessimistic in their outlook, most of such cases, if not all, being regarded as fatal. For instance, in the series of 175 cases of non-penetrating lesions of the heart reported by Bright and Beck (1935), as many as 163 cases (93%) were reported as proving fatal. Of late, however, it has been increasingly recognized that the percentage of recoveries, either partial or complete, after chest-wall injuries is much higher than has hitherto been believed. The former pessimism has been ascribed by Beck (1935) to "a failure to recognize or a fear to diagnose" traumatic heart disease.

Enough evidence has by now accumulated in the literature for it to be established that a perfectly normal heart with no evidence whatsoever of pre-existing disease (as in the case of the present case) is capable of damage from non-penetrating chest-wall injuries. However, as has been stressed by Külbs and Strauss (1932), Friedberg (1949), and others, a "blunt injury" to the chest wall is more likely to damage a diseased heart than a healthy one. Another fact of prime importance (though not applicable in the present case), recently established by the studies of Bilderbeck (1919), Hamilton (1934), Bright and Beck (1935), Warburg (1938, 1940), and of Reed and Berger (1945), is that serious heart damage, even inclusive of rupture, may ensue in spite of there being no significant or visible external bruising or rib-fracture.

The possibility of a "cause and effect" relationship between a chest-wall trauma and the subsequent development of a myocardial infarct or clinical picture of acute coronary occlusion has been the subject of considerable discussion in medical and medico-legal circles. This subject, obviously of great medico-legal importance, demands a much more extensive and unbiased investigation. For a proper appraisal of this question, however, it is important that we draw a clear line of demarcation between cases of so-called "contusio cordis" or "commotio cordis" (where myocardial haemorrhage with subsequent necrosis and fibrosis arises directly as the result of trauma) and cases of genuine coronary occlusion with myocardial infarction; such a distinction, although theoretically simple, is not easy to determine in actual practice. In spite of an extensive literature on this subject, there are actually not many instances reported of *proved* cases of coronary occlusion secondary to non-penetrating injuries of the chest wall. Because of the possibility of coincidence or an error of interpretation, it is not an easy matter to implicate a previous injury to the chest wall as the causative factor in any given case of coronary occlusion. Schmincke (1925), Warburg (1938, 1940), Kienle (1938), Macdonald (1941), and others have reported cases of this type in which blunt injuries or blows to the chest wall have been directly followed by attacks of typical coronary occlusion. The clinical syndrome of coronary occlusion in these cases has developed either immediately or even days, weeks, or months after the actual causative trauma.

In the case report presented here we find a young and healthy male of athletic habits who, for the first time in his life, develops symptoms and signs suggestive of heart disease from the moment he endures an injury to the chest sufficiently heavy to cause a momentary loss of consciousness. From that time onwards he presents, from time to time, the typical symptoms and signs of acute coronary spasm and, later, of acute coronary occlusion. The diagnosis of acute coronary occlusion is proved by the electrocardiographic picture, the high erythrocyte sedimentation rate, and the pyrexia.

Conclusions

In view of (1) the healthy past history of the patient; (2) the development of anginal symptoms immediately after a severe crush injury of the chest; (3) the radiological demonstration of fairly recent fractures of the fourth and fifth left ribs; (4) the development of definite symptoms and signs of acute coronary occlusion, including an electrocardiographic picture of anterior wall infarction with a typical right bundle-branch block; and (5) the radiological demonstration of pericardial and mediastino-pericardial adhesions in front of the right and behind the left ventricle, we feel justified in regarding this case as one of traumatic or, more correctly, post-traumatic coronary occlusion.

Cardiac arrhythmias of diverse character have been reported from time to time after injuries to the chest wall. Of these, auricular fibrillation is said to be the commonest (Hay and Jones, 1927; Friedberg, 1949). In the opinion of Kahn and Kahn (1929), Levison (1928), Reed and Berger (1945), and others, auricular fibrillation may arise *de novo* in a perfectly normal heart as the result of an injury to the chest wall. It was observed in about a third of Warburg's (1938, 1940) series of 32 cases. Sinus bradycardia has been reported after chest injury by Külbs (1937), auricular flutter by Kahn (1930),

and heart-block, complete or incomplete, by Laubry *et al.* (1921), Gallavardin (1922), Coffen (1930), Tuohy and Boman (1931), and others. But, so far as we can ascertain, this is the first case on record of atypical right bundle-branch block with anterior wall infarction arising as the result of a chest-wall injury and attended by clinical recovery.

Summary

A case report is presented of anterior wall infarction with atypical right bundle-branch block, arising directly after a crush injury to the chest wall, in a perfectly healthy young man and attended by clinical recovery.

So far as can be ascertained, this is the first case of its kind in the medical literature.

A short review is presented of present-day views on the subject of traumatic heart disease.

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Professor A. C. W. Hutchinson, giving his inaugural lecture at Edinburgh University (*Scotsman*, October 30), traced the course of dental history in Edinburgh. In 1876 the Edinburgh Dental Hospital and School was established, owing its beginning to the generosity of a few members of the dental profession who not only provided the funds but also gave up their time to teach the students. The first woman student was admitted 14 years later, and became the first woman to qualify as a dental surgeon; she was Lilian Lindsay, who celebrated her 80th birthday this summer. To-day there were 21 full-time posts on the establishment of the hospital and school, and the major problems were those of demolishing the old parts of the buildings and erecting new ones in their place, and of improving still more the standard of the teaching. Before the war negotiations were begun with the University with a view to making the dental school an integral part of the University. These had to be discontinued during the war, and were resumed after it; in 1948, at the beginning of the Health Service, the school was transferred to the University. The course for the degree of L.D.S. was instituted in 1949. He said the greatest task of all must be to substitute prevention for repair, and as a foundation students should be instructed in developmental factors, including nutrition, and more attention would have to be paid to children's teeth.

DIATHERMY TREATMENT OF ANGIOMATA

BY

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Benign tumours of blood and lymph vessels are relatively common, but there is no unanimity of opinion regarding their treatment. The present-day method of treating angiomas depends on the worker and his tools, and, on the whole, it may be said that the surgeon uses the scalpel or diathermy, the radiotherapist x rays or radium, and the dermatologist carbon dioxide snow. As the majority of these tumours are usually located in conspicuous areas, a good cosmetic result is an important consideration.

This paper is concerned with an attempt to improve the diathermy method, which has fallen into disrepute on account of disfigurement and scarring resulting from necrosis and ulceration of the healthy skin through which the current must pass. Nevertheless, in diathermy we do possess a safe and sure agent for the treatment of angiomas, provided that the high-frequency current is applied to the tumour without damage to the skin or the normal tissues through which the electrode is introduced. With this in view a special insulated needle electrode has been prepared and used with great success in more than 80 cases.

Some General Remarks

An angioma is a tumour composed of blood or lymph vessels. Normal blood vessels are derived from a vascular network, and it is said that the primary factor in the development of an angioma is the persistence of part of this network. They are therefore not true neoplasms but anomalies of development. Clinically the angiomas are divided into two distinct groups: (a) the haemangioma, which is composed of blood vessels containing circulating blood; and (b) the lymphangioma, which consists of lymphatic vessels and contains lymph. The subdivisions of these two groups which are suitable for treatment are discussed below.

It has been stated (Ribbert, 1898) that angiomas have no anastomotic communications with surrounding blood vessels, but my experience does not confirm this view. If the blood supply were a limited one, many of the attempts to destroy this tenuous supply would cause spontaneous disappearance. Cure cannot be expected unless every portion of an angioma is destroyed.

The smaller haemangiomas seen in children often disappear without treatment, but spontaneous disappearance of the larger lesions should not be awaited, because early treatment with low-intensity diathermy is safe and gives the best result.

Method

The diathermy coagulates the tissues by an intense heat generated in the vicinity of the active electrode. If the needle electrode is non-insulated the tissues surrounding it will be charred throughout the length of the needle. This causes unnecessary injury to normal tissues. In the method about to be described the needle is insulated except at its tip, and in this way the current is delivered only to the spot which requires treatment, and damage to the normal structures is avoided.