Heart-block and Coronary Thrombosis

Heart-block produces a well-defined clinical picture. Following trauma it may be considered to be proof of a deep-seated contusion. The most convincing cases are those in which it has been temporary. Coffen (1930) has recorded an example in a child and Warburg (1938) has collected others. Kissane (1937) has produced heart-block in dogs experimentally from a blow over the heart. Warburg (1939) writes: "Traumatic auriculo-ventricular block is not excessively rare." Last year I obtained a clinical history very suggestive of this condition in a hospital sister who fractured her sternum. The accident took place abroad, and the opportunity of proving the case was therefore lost. Typical coronary thrombosis proved by the electrocardiogram may come on a short time after a well-defined chest injury (Beck, 1935). Warburg (1939) has collected seven recorded cases of traumatic coronary thrombosis. A myocardial bruise or partial rupture may involve a coronary artery, particularly if it be already diseased. This exceptional accident is within the possibilities of clinical medicine.

Angina Pectoris, Auricular Fibrillation, or Valve Rupture

Clinical pictures typical of these well-defined conditions may follow a blow over the chest. Sometimes there is proof that the myocardium has been injured (Beck, 1935; Kissane, 1937; Warburg, 1938; Barber and Osborn, 1937). A discussion of these conditions would extend the scope of these notes, and the same applies to traumatic pericarditis. Numerous cases are cited in the literature comprised in the references below. Some degree of angina of effort is so common in cases of myocardial contusion that it may be regarded as one of the symptoms. It is possible, however, that a genuine angina syndrome may develop after a chest injury in the absence of definite anatomical changes.

Summary

The clinical and pathological evidence in relation to myocardial contusion is discussed.

The varying clinical pictures are described.

The judicious assessment of the clinical history and the clinical features is the most important guide in diagnosis.

BIBLIOGRAPHY

- Barber, H. (1912). *Practitioner*, **89**, 230.
 — (1938). *British Medical Journal*, **1**, 433.
 — and Osborn, G. R. (1937). *Guy's Hosp. Rep.*, **87**, 510.
 Beck, C. S. (1935). *J. Amer. med. Ass.*, **104**, 109.
 Bilderbeck, A. C. L. (1919). *British Medical Journal*, **1**, 675.
 Bright, E. F., and Beck, C. S. (1935). *Amer. Heart J.*, **10**, 293.
 Coffen, T. H. (1930). *Ibid.*, **5**, 667.
 Groom, W. (1897). *Lancet*, **1**, 1202.
 Gunewardene, H. O. (1934). *British Medical Journal*, **2**, 942.
 Hamilton, J. A. (1934). *Ibid.*, **2**, 1101.
 Kissane, R. W. (1937). *Contusion of the Heart*, Ohio State University, Columbus.
 — Fidler, R. S., and Koons, R. A. (1937). *Ann. intern. Med.*, **11**, 907.
 O'Neill, B. J. (1914). *J. Amer. med. Ass.*, **62**, 697.
 Priest, R. (1939). *J. R.A.M.C.*, **73**, 125.
 Rajasingham, A. S. (1939). *Brit. Heart J.*, **1**, 181.
 Warburg, E. (1938). *Subacute and Chronic Pericardial and Myocardial Lesions due to Non-penetrating Traumatic Injuries*, Copenhagen.
 — (1939). *Nord. Med.*, **2**, 1157.

F. Salmen (*Thèse de Paris*, 1940, No. 55), who records three illustrative cases in children aged from 10 to 13 years, states that whereas it used to be thought that Weil's disease was confined to adults, a certain number in recent years have been recorded in children. Its supposed rarity in children is attributed to difficulty in diagnosis by reason of the atypical or abortive character of the disease at that age. In the abortive form the symptoms consist merely of a meningeal reaction or actual meningitis with or without a very slight degree of jaundice. In another form the disease assumes the appearance of typhoid fever as in the pure febrile leptospirosis of the adult. The diagnosis in such cases can only be made by careful and repeated laboratory examinations such as Martin and Pettit's serum test and inoculation of the cerebrospinal fluid, blood, and urine into a guinea-pig.

ANAPHYLAXIS AFTER INJECTION OF TETANUS TOXOID

BY

A. A. CUNNINGHAM, M.D., M.R.C.P.

Municipal Medical Officer, Sector IV Headquarters, E.M.S.

The following report appears to be of interest owing to the recent attention which has been drawn to rare non-fatal occurrences of this nature. Whittingham (1940), Parish and Oakley (1940), and Cooke, Hampton, Sherman, and Stull (1940) have drawn attention to this condition in some detail and have instanced a small number of cases.

Case Report

A healthy member of the nursing staff—J. A.—volunteered for immunization with tetanus toxoid. The only item of interest in her past history was that she had had an injection of diphtheria antitoxin in 1931 that was followed some days later by an intense urticarial rash with joint pains and general malaise. She had had three injections of T.A.B. vaccine between September 16 and October 14, 1939, without untoward effects.

On October 21, 1 c.cm. of tetanus toxoid (B. W. and Co.) was injected subcutaneously into the upper arm. There were no ill effects until November 11 (that is, three weeks after the original injection), when a sudden rigor after a game of tennis was quickly followed by an intense urticaria preceded and accompanied by marked skin irritation. The lips, eyelids, and tongue began to swell, causing great mental agitation, but all the signs abated when 0.5 c.cm. of adrenaline diluted 1 in 1,000 was administered. On December 4 the second injection of tetanus toxoid was given, but in view of the previous history 5 minims of adrenaline was injected with 1 c.cm. of tetanus toxoid into the upper arm. Five minutes later tingling of the ears and some skin irritation were experienced; all went black before her eyes and she collapsed. A further 5 minims of adrenaline was given with the usual treatment for shock, and as she regained consciousness there was a rigor with vomiting and diarrhoea. The patient felt very poorly afterwards for about twenty-four hours.

Dr. H. J. Parish of the Wellcome Physiological Research Laboratories, Beckenham, very kindly came to the Park Hospital on February 27, 1940, and tested the skin reactions of the anaphylactic subject and five other volunteers, all of whom had had tetanus toxoid injections without any ill effect. The solutions used were Witte peptone, beef extract, and tetanus toxoid, and the results are briefly summarized in the accompanying table.

Initials of Subjects tested	Previous Injections					Intradermal Tests (0.1 c.cm.)				
	Diph. Antitoxin	Sc. Fever Antitoxin	Staph. Toxoid	Diph. Formo' Toxoid	T.A.B. Vaccine	History of Serum Rash	Witte Peptone 1/10,000	Witte Peptone 1/1,000	Beef Extract 1/1,000	Tetanus Toxoid 1/1,000
J. A. . .	1931	—	—	—	1939	1931	+++	Not carried out	+	+
A. A. C.	—	1933	—	—	1939	1933	++	++	—	±
M. H. . .	—	—	—	—	1939	—	++	++	—	—
E. M. . .	1919	—	—	—	1940	—	++	++	—	—
N. K. . .	1931	—	—	—	1939	—	++	++	—	—
N. W. . .	—	—	—	—	1939	—	++	++	—	—

The reactions from the intradermal test in patient J. A. were much greater using Witte peptone 1/10,000 than in the other volunteers when 1/1,000 dilution was used. There was immediate pseudopodial wheal formation, which at the end of half an hour measured 20 by 25 mm. when the surrounding flush measured 45 by 70 mm. A faint punctate erythema also spread up the arm towards the shoulder, but there was no general reaction. Owing to the severity of the local reaction we did not carry out a test with 1/1,000 Witte peptone. The evidence all

seems to favour the fact that the Witte peptone was the substance to which this patient was sensitized. An interesting point about this patient was the occurrence of symptoms three weeks after the first injection of tetanus toxoid instead of only after the second injection, as in the majority of recorded cases. The long interval of three weeks which had elapsed after the first injection makes it doubtful whether the toxoid was in fact responsible; an alternative suggestion is that the symptoms were due to some article of diet or other recent allergen. The symptoms that were observed within a few minutes of the second injection appeared to be directly caused by the toxoid, to which the patient may have been sensitized by the first dose, given six weeks before.

As recommended by other authors, adrenaline and a sterile syringe should always be readily available when tetanus toxoid is administered. It is also a wise precaution to keep all persons under observation for at least half an hour after injection.

I am informed that the medium now used for tetanus toxin production at the Wellcome Laboratories does not contain Witte peptone, but, as products of early digestion of protein are present in the new medium, there is still some risk of sensitization, and the precautions recommended above should not be relaxed.

On February 27 the tetanus antitoxin content of the patient's serum was found to be 0.2 international unit per c.cm. There is nothing unusual about this moderate titre, obtained approximately three months after the second of two injections of tetanus toxoid separated by six weeks.

Summary

A case of anaphylaxis following immunization with tetanus toxoid is recorded, but is a rare phenomenon, and in this patient was most probably due to sensitization to Witte peptone, a constituent of the medium in which *Cl. tetani* was grown.

My thanks are due to Dr. H. J. Parish of the Wellcome Physiological Research Laboratories for carrying out the necessary intradermal tests and to Dr. H. Stanley Banks, medical superintendent of the Park Hospital (L.C.C.), for permission to record this case.

REFERENCES

- Cooke, R. A., Hampton, S., Sherman, W. B., and Stull, A. (1940). *J. Amer. med. Ass.*, **114**, 1854.
Parish, H. J., and Oakley, C. L. (1940). *British Medical Journal*, **1**, 294.
Whittingham, H. E. (1940). *Ibid.*, **1**, 292.

Clinical Memoranda

Pneumococcal Arthritis complicating Lobar Pneumonia treated with Sulphapyridine : Recovery

This case is of interest in that the presenting symptoms were those of multiple acute arthritis, although the causative condition was lobar pneumonia.

CASE HISTORY

A girl aged 19, who had recently worked as a nursing auxiliary in a medical ward, was admitted to hospital on June 12, 1940, with a diagnosis of "acute rheumatism." Two weeks previously she had had a sore throat, which cleared up in seven days. Five days before admission she had had pain in the chest and back, followed three days later by severe pain in both thighs, legs, shoulders, elbows, and hands, which was unrelieved by full doses of salicylates. Her previous health had been good, and there was no personal or familial history of rheumatism.

On admission she was seen to be a well-nourished girl. She had acute pain in the hands, back, and under the ribs

on both sides, and looked ill and toxic, being in considerable distress. The face was flushed, the tongue dry and furred, the skin moist and hot. The fauces and pharynx showed patches of yellow follicular exudate on an injected background. The tonsillar glands were enlarged. The temperature was 101.2°, the pulse 96 and regular, the respirations 38, and the blood pressure 118/70. There was no obvious anaemia, no cough, and no cyanosis. Pleural friction was heard on the right side of the chest at the lung base; elsewhere the chest was normal to auscultation, and the percussion note was nowhere impaired. No other abnormal signs were found. Signs of acute arthritis, with marked peri-articular swelling, were present in both shoulders, elbows, hands, temporo-mandibular joints, knees, and ankles, and in the cervical and lumbar spine. The skin over the affected joints was hot and tender, and attempts at passive movement caused extreme pain. The left knee-joint contained a little fluid. No rheumatic nodules were found.

Cultures from a throat swab grew almost entirely pneumococci; there were no haemolytic streptococci. The leucocyte counts are shown below.

Date	Leucocytes per c.mm.	Poly-morphs	Small Lymphocytes	Large Lymphocytes	Hyalines	Remarks
June 13	14,200	81%	11%	5%	3%	On admission Bilateral lobar pneumonia After 30 grammes of sulphapyridine
" 20	17,800	80%	17%	1%	1%	
July 4	8,400	42%	54%	2%	2%	

Treatment was as follows: (1) Lead and opium lotion was applied to all painful joints, and both arms were lightly splinted. (2) Sulphapyridine (M & B 693) was given: 1 tablet (0.5 gramme) four-hourly for two days, then 1 tablet four times daily for six days, and 1 tablet three times daily for eight days, a total of 30 grammes. (3) One-third grain of omnopon was required twice daily for the first week to control joint pains.

Clinical signs of consolidation of the right lower lobe appeared on June 14. Pleurisy and consolidation spread to the left lower lobe on June 15. Fever, arthritis, and a clinical picture of lobar pneumonia continued until the end of the third week, when the temperature fell by lysis and the pneumonia began to resolve. The arthritis also subsided at this time, the hands being the last joints to become normal; no suppuration occurred in any joint.

The patient was discharged on July 17. The chest was then clear, and all joints were painless and capable of full active movements. No toxic effects except slight nausea and vomiting resulted from the sulphapyridine.

COMMENTARY

This case was misleading when first seen because the acute painful arthritis, involving especially the small joints of the hands, was the obvious cause of the patient's illness and distress; the throat was inflamed, with patches of yellow exudate, and presented the appearance of a streptococcal infection—a more common cause of acute polyarthritis. The pathological report of a pneumococcal growth coincided with the first clinical signs in the chest. It is noteworthy that several cases of sore throat were observed this summer in which the only organism present on culture was the pneumococcus; sulphapyridine was given promptly in these cases, and no complications ensued.

In the case reported it is probable that the original "sore throat" was a pneumococcal pharyngitis which gave rise to the polyarthritis and to the pneumonia. Although the physical signs were clearly those of a pleuropneumonia of both lower lobes, the temperature chart was by no means typical, partly, no doubt, on account of the preceding and accompanying arthritis, and partly, perhaps, on account of the sulphapyridine.

I should like to thank Dr. G. M. Wauchope for permission to record this case and for much helpful advice in preparing these notes. I also thank Dr. M. Leslie-Smith for the pathological reports.

STELLA INSTONE, M.B., B.S.,
House-physician, New Sussex Hospital,
Brighton.