OCCURRENCE OF MYOCARDITIS IN PARALYTIC POLIOMYELITIS

BY

J. F. GALPINE, M.D., F.R.C.P.Ed., D.P.H.
Consultant in Infectious Diseases, Coventry

AND

W. C. MacC. WILSON, M.D., B.Sc., D.P.H.
Group Pathologist, Coventry

Out of 141 cases of paralytic poliomyelitis admitted to the isolation unit in Coventry during 1957, there were six deaths in the acute stage and one death in the earlier post-acute stage. Of these seven fatalities, five (all adults) came to necropsy, and all showed microscopical evidence of myocarditis. Histological reports of British cases of poliomyocarditis are not numerous, being confined, so far as is known, to individual cases in children. A description of the five cases follows.

Clinical Summaries and Pathology

Case 1.—A man of 28 was admitted to hospital on May 15, 1957, with pyrexia and meningeal signs. The cerebrospinal fluid (C.S.F.) contained 190 white blood cells per c.mm. (60% polymorphonuclears) and 75 mg. of protein per 100 ml. Next day the paralysis spread rapidly to the limbs and respiratory muscles, and he was placed in a cabinet respirator. On May 21 his general condition deteriorated and tracheotomy was performed, followed by change-over to positive/negative-pressure ventilation through a cuffed tracheotomy tube. *X*-ray examination of the chest on May 22 showed collapse of the right upper lobe ; this was later followed by collapse of the right lower lobe with increasing mediastinal shift to the right unaffected by bronchoscopic toilet, suction through the tracheotomy, and antibiotics. His condition slowly deteriorated, and he died on July 10 in the ninth week of illness. *Diagnosis :*—Paralytic poliomyelitis, confirmed at necropsy. Basal pneumonia was present. The heart, containing much agonal clot, showed no gross abnormality, but histology revealed poliomyocarditis changes of moderate severity.

Case 2.—A housewife aged 25 was admitted on July 23, 1957, in the fourth week of the puerperium, with neck and back rigidity, nasal speech, and weakness of the left arm. The C.S.F. contained 150 polymorphonuclears and 75 lymphocytes per c.mm. and 250 mg. of protein per 100 ml. On July 24 the paralysis spread to all limbs, respiratory power became affected, and the patient was placed in a cabinet respirator. Next day her swallowing was somewhat impaired, but the pharynx remained dry and cabinet respiration was continued. On July 31, when the respirator was opened for nursing, she became cyanosed, complained of ill-localized pain in her chest, and died in about 10 minutes. *Diagnosis :*—Bulbo-spinal poliomyelitis, confirmed at necropsy. The C.N.S. changes were of considerable severity. The heart was somewhat dilated, but otherwise unexceptional. A large pulmonary embolus extended well into the pulmonary tree on both sides. *Histology of heart :*—Slight to moderate poliomyocarditis changes present.

Case 3.—A housewife of 32 was about 28 weeks pregnant when admitted on August 17, 1957. Paralysis was rapidly spreading in the trunk and limbs ; soon dyspnoea was obvious, and she was placed in a cabinet respirator. She vomited repeatedly, and, when a Ryle tube was passed, hourly suction produced amounts varying from a few cubic centimetres to some ounces of blackish fluid, a specimen of which gave a positive benzidine reaction. Intravenous feeding was started, and was continued until death on August 23. On this day her condition deteriorated sharply, and tracheotomy was performed without avail. *Diagnosis :*—Bulbo-spinal poliomyelitis, confirmed at necropsy. C.N.S. changes were marked. The trachea and bronchi contained blood-stained fluid, and the lungs showed extreme congestion and oedema. There was pronounced dilatation of the stomach and small bowel. The uterus contained a normal male foetus. Macroscopically, the heart showed no definite abnormality. *Histology of myocardium :*—Changes in the muscle fibres were slight, but there were small scattered foci of interstitial inflammatory cellular infiltration.

Case 4.—A spinster aged 30 was admitted on September 10, 1957, with pyrexia (101° F. ; 38.3° C.), marked neck rigidity, and rapidly spreading paralysis. On the 11th she was put in a cabinet respirator, and later the same day, when swallowing became impaired, tracheotomy was performed and pressure-breathing through a cuffed tube begun. On September 12 her temperature had risen to 104° F. (40° C.) and numerous extrasystoles were noticed, which an electrocardiogram indicated to be of auricular origin. Her blood-pressure was then 150/85. Digoxin, 1 mg. intravenously, was followed by a reduction of pulse rate from 130 to 100, but irregularity persisted. On September 13 the fast irregular pulse continued, and the blood-pressure was 160/90. Her condition remained unaltered until her sudden death on September 14. *Diagnosis :*—Bulbo-spinal poliomyelitis, confirmed at necropsy. C.N.S. changes were well marked. The left lung showed long-standing collapse with fibrosis. The right lung was well aerated, but had a marginal strip of collapse. The heart showed some hypertrophy of the left ventricle and a thickened and scarred mitral valve. *Histology of myocardium :*—Some interstitial fibrosis, presumably due to old rheumatic carditis, with no evidence of recent activity. Poliomyocarditis changes of some severity were present.

Case 5.—A man aged 25 was admitted on September 17, 1957, with pyrexia and a nasal voice associated with left palatal weakness but no meningeal signs. Next day he had slight nuchal rigidity, impaired swallowing, a hoarse voice, and weakness of the left masseter. On September 20 he was completely aphonic, and some weakness had appeared in both arms. The same afternoon his breathing had a periodic rhythm reminiscent of Cheyne-Stokes respiration. His temperature fell to 97.4° F. (36.3° C.), with a regular pulse of 103 and blood-pressure 160/140. His extremities were cold, clammy, and blue, and his trunk mottled and livid. An electrocardiogram showed supraventricular tachycardia. Later the same day the systolic pressure began to fall and the diastolic became difficult to estimate. His pulse became periodic, swinging regularly every half-minute between 85 and 120 ; he also developed a coarse eye-flicker. At 11.30 p.m. and at 12.30 a.m. (September 21) he had copious coffee-ground vomits, and a blood transfusion was started. He died at 1.55 a.m. *Diagnosis :*—Bulbo-spinal poliomyelitis, confirmed at necropsy. C.N.S. changes were well marked. The heart showed numerous subepicardial and a few subendocardial petechiae. The myocardium was very dark and congested-looking. The stomach contained thick mucoid material with an area of severe ecchymoses on the lesser curvature. *Histology of heart :*—Florid poliomyocarditis changes present (Figs. 1 and 2). Sections of the ecchymotic area in the stomach showed intense engorgement of dilated vessels in the submucosa, with interstitial haemorrhages.

Review of the Histology in the Coventry Cases

The morbid histology in poliomyocarditis has been summarized as hyperaemia, structural distortion probably due to oedema, fragmentation of myocardial fibres, and infiltration, both perivascular and diffuse, by lymphocytes and large monocytic cell elements

(Nordenstam, 1956). These features were present in the Coventry cases, but our experience coincided with that of Fox *et al.* (1953) in that polymorphonuclear leucocytes were prominent in the cellular infiltration, and we found the same wide variation in intensity from small scattered and easily overlooked foci to florid and fairly diffuse changes. There did not appear to be anything specific in the distribution of the lesions or in the degenerative or reactive changes in affected muscle fibres, but the mixed nature of the inflammatory cellular infiltration did seem to be characteristic; in places the intensity of infiltration approached that of an acute pyogenic lesion, but cellular pleomorphism was still as evident as in the small scattered and inconspicuous foci.

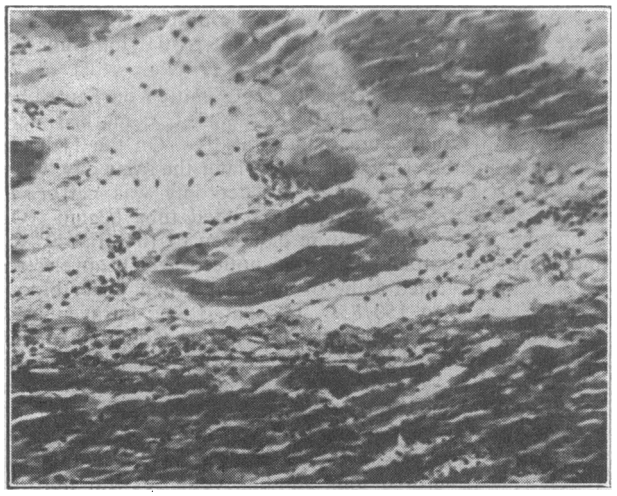

FIG. 1.—Inflammatory cellular infiltration of myocardium, both perivascular and diffuse. (Haematoxylin and eosin. ×100.)

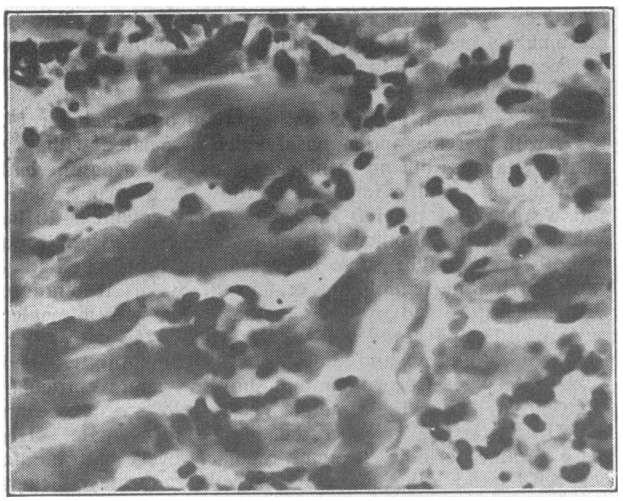

FIG. 2.—Heavy pleomorphic cellular infiltration with necrosis and fragmentation of muscle fibres. (Haematoxylin and eosin. ×400.)

Discussion

Myocarditis in poliomyelitis has become well documented since Robertson and Chesley (1910) described swelling of muscle fibres, indistinctness of striation, and oedema of interstitial tissue. Saphir and Wile (1942) showed myocarditis to be potentially not uncommon by recording histological changes, including foci of perivascular cellular infiltration, in six out of seven fatal cases. Since then reports of larger series have appeared: for example, Fox *et al.* (1953), in America, found histological evidence of myocarditis in 38 out of 70 fatal cases; Vimtrup *et al.* (1956), in Copenhagen, in 43% of 112 cases; and Nordenstam (1956), in Stockholm, in all of 26 necropsies. Involvement of the myocardium does not correlate with any particular clinical type of paralytic poliomyelitis (Spain *et al.*, 1950). Poliomyelitis virus has been isolated from the myocardium—first by Jungeblut (1950)—and invasion is thought to occur during viraemia in the early stages of illness. There has been speculation—for example, by Fox *et al.* (1953)—concerning the variation in cardiotropism among the poliomyelitis viruses, which, if it exists, might explain why it was not until 1957 that poliomyocarditis was found in this unit, though search had previously been made for it in five fatal cases occurring between 1953 and 1956. Similarly in another fatal bulbo-spinal case in November, 1958, no histological evidence of myocarditis was found. Strains of Coxsackie virus, a group whose enteric habit and physical properties are similar to the poliomyelitis and E.C.H.O. viruses (*Brit. med. J.*, 1958), have been isolated in cases of encephalomyocarditis neonatorum (Gear, 1958).

British reports of poliomyocarditis include those of Hertz *et al.* (1913), who described a case of polio-encephalitis in a boy of 12½ years in whom concomitant myocarditis was diagnosed clinically on the basis of a systolic murmur at the apex and cardiac dilatation; of Wright and Owen (1952), who described poliomyelitis in a mother and her newborn infant and found myocarditis in the baby, who succumbed; of McConnell (1952), who described 10 cases of poliomyelitis in infants under 6 months, with three deaths, occurring in Northern Ireland in 1950, and found myocarditis at the post-mortem examination made in one case; and of Pugh (1952), who described a case of fatal bulbar poliomyelitis with myocarditis complicating Still's disease, and in mentioning the difficulty of assessing the relative importance of bulbar involvement and myocardial changes in causing death inclined to the former.

A number of authors have studied the electrocardiographic (E.C.G.) changes in poliomyelitis. Weinstein (1957) believes that such studies indicate greater frequency of myocarditis than is shown by anatomical studies, and considers that, while dysfunction of the nervous system and abnormal ventilation may account for minor E.C.G. changes, major and more persistent ones are related to myocarditis. Bjerre-Christensen (1956) picked out, in retrospect, arrhythmia, P-wave changes, and conduction disturbances as suggestive of myocarditis. Gefter *et al.* (1947) thought clinical findings less instructive than E.C.G. abnormalities; these increased with severity of case, though myocardial derangement could only be inferred. Rose (1952) found non-specific abnormalities, as did Fox *et al.* (1953), whose comment seems to express a common view—"the myocarditis produced by poliomyelitis does not seem to give rise to characteristic electrocardiographic changes." In the E.C.G. recordings taken in two of the five cases reported above, one showed auricular extrasystoles and the other auricular tachycardia.

Pulmonary oedema, often found in fatal cases of poliomyelitis, was present in its gross form at necropsy in only one of the five cases. Though a number of factors, including cardiac involvement, have been suggested in its pathogenesis, Weinstein (1957), along

with others, believes that "destruction of the medulla is the keystone of this phenomenon." Case 3 supports this view, as C.N.S. changes were well evident, while myocarditis was minimal.

The clinical diagnosis of myocarditis in poliomyelitis leaves much to be desired, though tachycardia without obvious cause is a possible clue (Fox *et al.*, 1953). Gefter *et al.* (1947), in describing 11 fatal cases, with myocardial pathology in five out of six necropsies, stated that they showed little clinical evidence of cardiac disease. Standard works do not mention cardiac signs in their accounts of poliomyelitis. Sudden death in poliomyelitis may have an association with myocarditis, as it was found in all of 14 subjects dying suddenly in a total of 28 fatalities (Shutkin, 1951). Weinstein (1957) points out that it is difficult to distinguish the origin of cardiovascular dysfunction among the competing factors arising from possible involvement of myocardium, lungs, and medulla. Among the welter of neurological and ventilatory disturbance in our five cases it would have been hard to say that clinical evidence of myocarditis existed. In the absence of satisfactory clinical or E.C.G. evidence the diagnosis of myocarditis has usually been a posthumous histological discovery. As only the severest cases come to necropsy no generalization is possible regarding the myocardium in all cases (Spain *et al.*, 1950). It seems safe to say, however, that no evidence has appeared which gives myocarditis any clinical importance in non-life-threatening paralysis, but in the severe cases it may be an unsuspected contributor to death.

Summary

Five fatal cases of paralytic poliomyelitis are described. All showed histological evidence of myocarditis. The severity of the myocardial lesions varied greatly from case to case, but the pleomorphic nature of the inflammatory cellular infiltration is probably characteristic of poliomyelitis. The difficulties in clinical diagnosis of poliomyocarditis and assessment of its importance are discussed.

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ADULT PYLORIC OBSTRUCTION DUE TO A MUCOSAL DIAPHRAGM

REPORT ON TWO CASES

BY

DIGBY CHAMBERLAIN, Ch.M., F.R.C.S.

Professor of Clinical Surgery, University of Leeds

AND

N. V. ADDISON, F.R.C.S.

Senior Registrar, the General Infirmary at Leeds

Intrinsic diseases of the stomach can cause pyloric obstruction, and include neoplasms, ulcer, congenital and acquired hypertrophy of the pylorus, spasm, tubercle, syphilis, and foreign bodies. The commonest cause of pyloric stenosis is a duodenal ulcer, either in the active state with associated oedema or in the chronic stage with cicatrization.

Obstruction can also be due to a mucosal diaphragm, which may occur either proximal to or at the level of the pylorus, with no visible or palpable cause on external examination of the stomach. This diaphragm consists of a double layer of mucous membrane, with a small aperture, usually central in position, there being only submucous tissue and muscularis mucosae between the layers (Fig. 1).

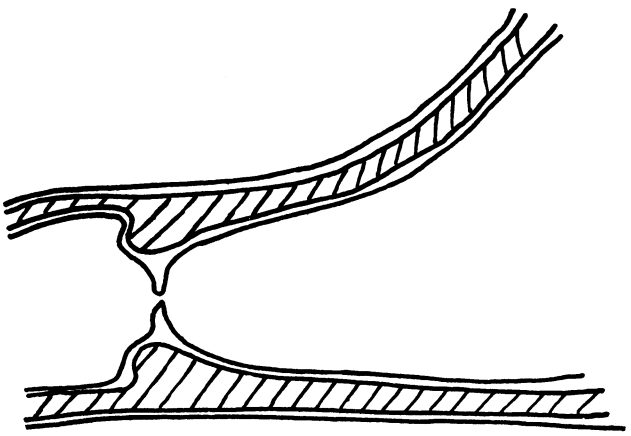

FIG. 1.—Diagrammatic representation of a mucosal diaphragm at the pyloric ring, consisting of two layers of mucous membrane, with a central aperture.

At the time of writing only four cases have been described in the world literature, but Rhind (1959) has since reported further cases.

We record two case histories of adult pyloric obstruction due to mucosal diaphragms and discuss the aetiology and differential diagnosis.

Case 1

A housewife aged 55 attended hospital on September 2, 1958, with an eight-months history of fullness after meals. During this time she had lost just over 2½ st. (15.9 kg.) in weight. Her appetite was variable, and sometimes she had eaten reasonably well. Before her symptoms started she had some mental worry, and she thought they might have been due to this cause. She made herself vomit at least once a day, and during the previous two months had been vomiting more often, but not large amounts. Bowels and micturition were normal. On examination she looked reasonably well, and did not appear to have lost weight