CASE II.

H. D., female, aged 45. Two previous attacks of rheumatic fever. Admitted with all the signs of failing compensation—general oedema, ascites, orthopnoea, and cyanosis. She complained of sharp pain in the chest and left upper arm as far as the elbow.

Physical Signs in Heart.—H.A.B. fifth space, 5½ in. from the middle line. Impulse irregular and not very forcible. At apex both sounds heard, and, in addition, there is a loud, rough, systolic murmur conducted outwards to the axilla. At the base both sounds are heard and the second sound is accentuated at the pulmonary cartilage. There is no murmur at the base.

CASE III.

M. G., female. Aged 31. Four attacks of rheumatic fever. Complains of shortness of breath, palpitation, and pain. The latter is in various parts of the chest, but especially in the precordium, and often radiates down the left arm.

Physical Signs.—H.A.B. in fifth space, 3½ in. from the middle line. The impulse is slapping in character, and preceded by a well-marked presystolic thrill. At the apex a rough presystolic murmur is heard, followed by a short first sound. The second sound at the pulmonary cartilage is accentuated. The pulse is irregular, and the liver cannot be felt.

The occurrence of such cases is sufficient to cast doubt on the theory of the "mechanical" origin of cardiac pain. And a more detailed consideration of the conditions confirms the necessity of abandoning this theory. Thus, two possible methods of mechanical stimulation of the afferent nerve endings in the heart wall suggest themselves (a) by increased strain as a result of undue distension of the chamber; (b) by increased pressure as a result of excessive strength of contraction of the cardiac wall. As already mentioned, the former of these alternatives is apparently the one adopted by Head.³

If it were true, then the ideal condition for the production of heart pain would be the overfilling of a cardiac chamber during diastole. In other words, pain should be a prominent symptom in all cases of heart disease, with marked dilatation and insufficient emptying of the chambers. Now this condition is met with to an extreme degree in the common case of "double mitral disease" with failure of compensation; yet a patient afflicted in this manner not uncommonly exhibits all the physical signs of complete failure of compensation—cyanosis, orthopnoea, general anasarca—without at any time suffering any pain referable to the heart. In the series of 50 cases two well-marked examples of this condition occurred, both in young subjects, and both going to a fatal issue; yet the patients never manifested either pain or tenderness. Such cases would be quite inexplicable were the mechanical effects of cardiac dilatation the chief factor in the production of pain. The converse argument tells in the same direction—many of the severest cases of heart pain occur in patients in whom no dilatation can be observed either clinically or at autopsy.

The second alternative—increased pressure on the nerve endings as a result of excessive force of contraction—hardly calls for consideration. The condition of great muscular hypertrophy without the occurrence of any pain is so commonly met with, in athletes, in renal disease, etc., that this possibility could never be seriously urged as a fitting explanation.

It seems, then, that no mechanical theory is capable of offering a satisfactory explanation of the origin of the "painful" stimulus. But there is one factor which may be presumed to be present to some degree in all cases of heart disease, namely, some *chemical* change in the myocardium. The suggestion presents itself that the stimulus to the nerve endings may be a chemical one, some product of the disordered metabolism of the heart muscle. On consideration a number of facts can be adduced which fit in well with this suggestion. Thus, there is a general consensus of opinion that those heart affections are most characterized by pain in which, at autopsy, most evidence of myocardial degeneration is present. Again, the paroxysmal nature of the pain in many cases, without any corresponding change in the physical signs in the heart, is easier to explain on the assumption of a chemical stimulus than of a mechanical one.

No striking originality is claimed for this view; in fact, it is in some degree foreshadowed by Mackenzie's opinion, quoted above, that the pain often results from exhausted contractility, for there can be practically no doubt that the phenomena of muscular exhaustion must rest on a chemical basis. In the present state of biochemical knowledge it is impossible to suggest what actual com-

pound or compounds could be invoked as an adequate stimulus. But it is quite within the bounds of probability that competent chemical investigation of the myocardium of patients whose clinical history was known might throw considerable light on this question.

Further, this conception of the mechanism of cardiac pain tends to bring it into line with other painful conditions—notably, "cramp" of voluntary muscles and the "lightning pains" of tabes dorsalis. Mott and others believe that the latter are caused by gradual liberation of chemical irritants from the degenerating posterior root neurons. This view has displaced the earlier one—that the pain was due to pressure from sclerosis of the roots. It affords a ready explanation of the clinical observation that the pains often cease when ataxia develops, and that in those patients who never become ataxic pain continues to be a prominent feature throughout the course of the disease.

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- ¹ O. May, *Archives of Middlesex Hospital*, October, 1909. ² G. A. Gibson, *Nervous Affections of the Heart*, 1905, p. 38. ³ H. Head, *Brain*, vol. xix (1896), p. 153 et seq. ⁴ W. Einthoven, *Quarterly Journal of Experimental Physiology*, vol. i, p. 244. ⁵ J. Mackenzie, *Lancet*, 1895, p. 16 et seq. ⁶ J. Mackenzie, *Diseases of the Heart*, 1908, p. 187.

INFECTIVE ENDOCARDITIS TREATED WITH ANTIDIPHThERIAL SERUM.

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IN the absence of bacteriological proof, some authorities state¹ that the cure of infective endocarditis cannot be accepted as such.

Time and the many exigencies of general practice make it almost impossible for the practitioner to make blood cultures, take opsonic controls, and indulge in the ultra-refinements of the bacteriological laboratory. The following notes are based purely on clinical observation, and their publication would not have been sought had not the case in question been seen by one whose authority goes without saying.

On August 14th, 1909, I was called to see a man, J. T., aged 30, and was told that he had just had "a severe shivering attack."

Previous History.

The patient, as far as he could remember, had always enjoyed good health, the only trouble being a "running ear," which sometimes discharged a little, but for long periods was quiescent; the condition of the ear he dated back to childhood. His habits had always been regular, and he had always been most abstemious. No history of rheumatism or injury could be elicited.

State on Examination.

I found the patient sweating profusely, the pulse was forcible, and the respirations hurried; he was pale, complained of headache, and had just had an attack of vomiting; the tongue was furred, dry, and tremulous; the temperature was 104° F. For some few days he had not felt quite as well as usual, but did not attach much importance to this condition. The provisional diagnosis made was some atypical form of typhoid.

Progress of the Case.

The pyrexial curve and absence of other typhoid symptoms dispelled the idea of typhoid fever, and I then regarded the case as one of septic infection, and by a process of exclusion infective endocarditis was surmised.

The patient, in spite of ordinary treatment, continued to have severe rigors, averaging three attacks in the twenty-four hours, with an intermittent pyrexia ranging between 97° and 104° to 105°, accompanied by delirium and followed by profuse sweats.

Examination of the heart gave at first indefinite results, but a systolic murmur could be heard over the aortic area on the third day; and on the fourteenth day a well-marked mitral systolic murmur could be heard carried up into the axilla.

The spleen was enlarged and tender. The urine contained albumen, but no casts. Examination of the left ear revealed a perforation of the tympanic membrane, with a bead of pus in the middle ear.

On the eighth day of the disease the condition of the patient was so grave that I deemed it necessary to call in further advice, and Professor Dixon Mann saw the patient with me. The diagnosis was confirmed, but the prognosis was hopeless.

Treatment.

At first the patient was put upon large doses of quinine and arsenic, but they had practically no influence upon the pyrexia; and, having to deal with a case of a pyaemic nature, whose blood stream was no doubt charged with infective bacteria, I decided to treat the patient as much as possible on germicidal lines.

Regarding the septic ear as a possible channel² for the infecting micro-organism, this was treated with instillations of hydrogen peroxide, followed by irrigations of a mild antiseptic lotion. The alimentary canal and the renal secretion were likewise treated antiseptically; for the former salol and benzonaphthol were administered three-hourly, and for the latter 15 grains of urotropine was given night and morning. To maintain the patient's strength I allowed as much as he could take of an easily assimilative diet. Stimulants were not resorted to, since the patient had never taken any.

In spite of the above treatment, the rigors still persisted, so that my mind turned to vaccines and antitoxins. Two cases were recently brought to my notice where, even after blood cultures were made, and in the one case a suitable emulsion of killed micro-organisms made, and in the other an appropriate serum prepared, yet the result was disappointing.

Although antidiphtherial serum so far is regarded as a specific in neutralizing the toxins of the Klebs-Loeffler bacillus, yet cases are not wanting in which it has been of service in other directions, consequently I decided to inject an initial dose of antidiphtherial serum (Parke, Davis and Co.) of 2,000 units into the abdominal parietes. To my surprise and to the gratification of the patient, only one rigor took place during the next twenty-four hours, and for the following five days the morning temperature was slightly subnormal and the evening temperature just above the normal. On the evening of the fifth day after the first injection I was hurriedly summoned and found the patient in a severe rigor, with a temperature of 104.2°; the rigors continued for the following three days, but in a milder form than formerly, and were not as frequent.

The patient derived so much benefit from the first injection that he begged for another, and I therefore gave him a second injection of 4,000 units in the early morning; the same evening he had a slight rigor, and on the following day the temperature dropped to 101°; after this the temperature kept more or less to the normal, and the patient made an uninterrupted recovery. The patient remained in bed six weeks; three weeks he recruited in a convalescent home, and on examination on November 28th the heart sounds were quite clear, his anaemia much less marked, and his weight had increased 8½ lb.

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HENOCH'S PURPURA OR ANGIO-NEUROTIC OEDEMA?

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IN connexion with Dr. Leigh Day's case of Henoch's purpura, the following cases, which occurred within a few weeks of one another, may be of interest:

CASE I.

C. M., male, aged 12. First seen on February 9th, 1906; had complained for four days of severe abdominal pain, accompanied by diarrhoea. "The motions looked like blood." On both legs and the backs of both hands there had been white swellings, "the size of a walnut," which developed rapidly, lasted a few hours, and then disappeared.

The boy was pale but not anaemic; the temperature was 100°, the pulse 80 and irregular. The abdomen was slightly retracted, nowhere tender, and nothing abnormal was felt. There were no spots nor any trace of the swellings described. There were no objective signs in the chest.

On February 10th the temperature was 99.6°, the pulse 74, and he looked very ill, with sunken eyes, and much abdominal pain. During the night he had passed six loose motions, containing much bright red blood but no mucus or pus. Nothing abnormal was felt on rectal examination. Several purpuric spots were observed around the elbows.

On February 11th he appeared better, the temperature was normal, the pulse 78, and he had had only one motion, normal in appearance, in the last twenty-four hours, but the urine was scanty and contained a large amount of blood. The skin of the buttocks and sacrum was covered with large purpuric spots, some the size of a shilling.

On February 12th the general condition was better and the pain was neither so frequent nor so severe. He had slight diarrhoea, but the motions contained only a small quantity of blood. The urine had increased and the quantity of blood had diminished.

Up to February 19th his condition remained much the same, when there appeared on the back of the left hand a patch of solid oedema the size of a bantam's egg. The movements of all the joints in the hand were unattended by pain. The patch lasted twelve hours, and then subsided rapidly, leaving no trace.

From February 19th to 28th his general condition steadily improved, pain ceased, and the blood disappeared from motions and urine.

On February 25th he had a fresh crop of purpuric spots on the buttocks which lasted two days only. By March 1st he was quite convalescent.

CASE II.

G. L., male, aged 4½. Was seen on March 12th, 1906. For a week he had complained of severe abdominal pain, coming on specially at night; otherwise he did not appear to be ill. The boy was well nourished and not anaemic; he did not complain of pain at the time of examination, and the temperature was normal. Careful examination of abdomen revealed nothing abnormal. There were no objective signs of disease.

For the next few days the attacks of pain were frequent and severe, but I failed to detect anything to throw light on the condition.

On March 20th I examined him during an attack of pain. The pulse was 100, regularly intermittent, and there was most noticeable rigidity and retraction of the abdominal muscles, but nowhere did pressure reveal any tender spot. Around the wrists and on the extensor aspects of the legs were a large number of circular spots, resembling urticaria, but the hyperaemic border was a darker and more dusky red than in ordinary urticaria. There was no irritation. These spots lasted four days, and left a stain like a bruise.

On March 21st his condition was about the same. On the backs of both hands were patches of solid oedema of an area of about 2 sq. in. There was no pain on movement of any of the joints of the hands. These swellings lasted forty-eight hours and then rapidly disappeared, leaving no stain. He complained of pain in the dorso-lumbar region.

On March 22nd a patch of solid oedema appeared on the back, over the spine of the last dorsal vertebra. It lasted less than twenty-four hours, leaving a well-marked bruise, which covered an area of 4 sq. in.

On March 23rd the eyelids were oedematous and the site of urticarial spots becoming purpuric. The child was very ill; the pulse was 120 and intermittent, the temperature normal; he was taking very little nourishment.

On March 24th he vomited during an attack of pain; urticarial wheals appeared on the cheeks, replaced two days later by purpuric spots.

On March 25th the child was exceedingly ill and vomited frequently; on the next day he was much collapsed, the face livid, the eyes sunken, and the pulse 120, small and very soft. He had vomited incessantly all night, but had had no attacks of pain.

On March 27th the condition was greatly improved; he had only one attack of pain, and there had been no vomiting. On March 29th large purpuric spots appeared on the elbow and backs of forearms; and from March 30th to April 3rd several fresh crops of spots appeared. On the arms many of the spots coalesced, causing large areas of discoloration. On April 3rd a few spots were noticed on the hard palate. He complained of pains in both knees; there was solid oedema above the left patella, and the knee-joint was hot, swollen, tender, and contained excess of fluid. The pulse for the last few days had been about 70.

From April 4th to 30th he improved steadily, although he had several fresh crops of spots and patches of oedema. The attacks of pain occurred at longer intervals, and were less severe. By the middle of May he was convalescent.

Perhaps the most interesting feature common to both cases was the occurrence of solid oedema which appeared and disappeared very rapidly. This is not a condition generally met with in Henoch's purpura, but angio-neurotic oedema is characterized by severe gastro-intestinal crises, and Osler has pointed out that Henoch's purpura has an interesting connexion with angio-neurotic oedema. In neither case was the spleen felt. The temperature in the first case was slightly raised at the onset, but in the second it was normal throughout; in both at times the pulse was unduly slow, and for some time in the younger boy it was regularly intermittent. There was no involvement of the joints in the first case, and a very slight and transient swelling of one joint in the second. The changes in the younger boy's general condition were very rapid. One morning he would appear to be in extremis, and the next his illness seemed to be slight. Conty regards the disease as of nervous origin, affecting the vasomotor nerves, and certainly in these two cases the symptoms might be explained by the presence of a toxin acting mainly on the sympathetic nervous system.

THE Metropolitan Asylums Board has arranged for further lectures and demonstrations in hospital administration for the benefit of candidates for the diploma in Public Health. There will be two simultaneous courses covering the same ground, the one being conducted at the North-Western Hospital by Dr. J. MacCombie, and the other by Dr. F. M. Turner at the South-Eastern Hospital. Each course lasts three months, that at the North-Western Hospital commencing at 11 a.m. on Monday, January 10th, and continuing on each consecutive Monday thereafter. The South-Eastern Hospital course begins at 2 p.m. on Wednesday, January 12th, continuing at the same hour on the following Wednesdays. The fee is £3 3s.