of gonorrhoea at 24 and the onset of heart symptoms 20 years later, with the single exception of gastric ulcer four years before the latter. Hence we cannot date the onset of the endocarditis; but it is certain that it must have been present for some time and have become fairly extensive before symptoms were produced. Subsequent to this the evidence shows that the inflammatory process was present in varying degrees of activity over a period of 13 years, now advancing and causing complete block, now receding and allowing normal impulses through; and finally, post mortem we find a small microscopic area in the muscle still in a state of active subacute inflammation, and one or two fresh vegetations on the valves. The clinical story, coupled with these pathological findings, certainly suggests an infection which, though now active and now relatively quiescent, had never died out, but persisted for 13 years.

Secondly, it is remarkable that during the last 12 years, when the patient was under frequent observation, sometimes with complete block, sometimes free from block, he was never actually found to be in the transitional stage of partial block. Considerable variation in the pulse-rate was found at different times, but irregularity was never noted subsequent to the occasion when he first consulted a doctor about his heart 13 years before death. Further, the ventricular rates observed at times when complete block was present are noteworthy. Two electrocardiograms taken in 1915 show ventricular rates of 51 and 49 per minute with complete block. In 1920 after auricular fibrillation had set in, three electrocardiograms show ventricular rates of 60 per min., although the perfect regularity of the ventricle proves that all impulses from the auricle were completely blocked; and the clinical records at this time show that an average ventricular rate of 73 per min. was maintained for three weeks, a maximum of 88 being attained on three occasions.

Now these rates are so high for an idioventricular rhythm that they compel speculation concerning the site of origin of the stimuli to which the ventricle was responding. It was conjectured, during life, that the lesion must have produced functional section of the conducting tissues across the A.-V. node, leaving sufficient intact nodal tissue below it to serve as the pacemaker to the ventricle. Post mortem it was hoped that microscopical examination might give information on this point, but unfortunately, as stated above, the changes in the conducting tissues were found to be diffuse, and no particular level can be selected as the most severely affected. The A.-V. node and bundle, lying as they did quite at the periphery of the main septal inflammatory focus, have suffered very much less than the tissue immediately subjacent to them. Their conducting function must have been interfered with in two ways—namely, (1) by transmitted pressure due to inflammatory tension in the underlying focus, and (2) by direct implication in the inflammatory process. Of these two factors we are inclined to think that the first—transmitted pressure—was the more important in producing the clinical events of the case.

If actual inflammation of the conducting tissues themselves had been the direct cause of the complete block in 1908, 1910, and during most of 1915 and 1916, we should have expected to find a degree of scarring of these tissues, not only much more extensive than is seen in these sections, but such as would entirely have prevented any resumption of the conducting function after 1916. We should also have expected the period during which such inflammation was subsiding to have been marked by a state of partial heart-block of such duration that it could hardly have been overlooked. For these reasons we regard actual implication of the junctional tissues themselves in the inflammatory process as having been of minor importance in the production of the heart-block in the earlier stages, and consider that transmitted pressure was the predominant factor. Both, however, must at different times have varied much in degree, and probably also in level. The pathological changes found do not permit us to assert that the variations in ventricular

rate recorded on different occasions when complete block was present were due to alteration in the level at which the junctional tissues were being attacked, but we consider this to be the most probable explanation. In the same way we cannot prove that the relatively rapid rate of the ventricle when block was complete, in particular during the final stages, was due to activation from the A.-V. node, but we think that this is far more probable than that the ventricle itself was originating impulses at such a rapid rate.

In conclusion, we wish to express our indebtedness to Sir Arthur Keith for his advice concerning the histological examination, and for his kindness in personally removing the tissues for this purpose. We also feel it only right to record our appreciation of the spirit of the patient, who left directions that we were to be given the opportunity to examine his heart after death.

EIGHT EXAMPLES OF BUNDLE BRANCH LESION IN THE HEART.*

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SINCE presenting to the New York Pathological Society 20 typical examples of bundle branch electrocardiograms which were published in the proceedings of the Society, I have collected eight additional examples from about 500 consecutive patients. This gives a frequency of 1.6 per cent., which is about the same as in the previous group of 20 cases. One of the eight patients is particularly interesting, in

Electrocardiograms of Cases F.H.H.D. (typical) and F.O.P.S. (irregular).

that the records taken two and a half years previously showed a normal electrocardiogram. The clinical picture is about the same in the two groups. Some patients had very definite histories of severe attacks of pain, lasting several hours, followed by gradual recovery; in others the onset of pain did not appear so clearly in the history.

The records of these eight people fall into two groups. In the first group of four cases the records are like the electrocardiogram of Case F.H.H.D., and show the typical characteristics necessary to diagnose a disease of one bundle branch which completely destroys the function. The R-waves or S-waves are large and wide and show notches, and the T-waves are large and downward in Lead 1.

The four remaining records are the same as the electrocardiogram of Case F.O.P.S., and are not so typical of this condition. However, they all show marked abnormality in the notching or in the width

* Read before the meeting of the New York Pathological Society, Feb. 8th, 1922.

ANALYSIS OF FINDINGS OF EIGHT EXAMPLES OF BUNDLE BRANCH LESIONS IN THE HEART.

Case.	Age.	Sex.	Probable duration of disease in years.	Onset.	Pain and dyspnœa.	Œdema.	Rhythm.	Blood pressure.	Murmurs.	Response to digitalis.	Size of heart.	Diagnosis.	Electro-cardio-gram.
F.H.H.D.	76	M.	10/12	Pain ex.	Pain.	No.	I.	90-180	S.	Good.	M. E.	Arterio-sclerosis.	T.
S.H.B...	48	M.	4	Pain referred to epigastrium.	Vice-like feeling. Dy. Mod. ex. Pain. Dy. ex.	V.s.	N.	160-200(S.)	S.	Fair.	E.	„	T.
F.B.B.I.	64	M.	3	Pain and discomfort	„	No.	I.	110-160	S.	St.	E.	Advanced cardiorenal disease.	T.
F.I.I.P.	58	M.	2	Dyspnœa ex.	„	Yes.	I.	170-200(S.)	None.	Good.	Large.	Arterio-sclerosis.	T.
F.O.P.S.	61	F.	3/12	Pain ex.	„	No.	F.	80-190	None.	Good.	Sl. E.	Cardio-sclerosis, hyperthyroidism.	AT.
F.I.O.S.	59	M.	6/12	Pain and discomfort.	„	No.	R.	100-140	S.	Not given.	Not E.	Arterio-sclerosis.	AT.
F.D.O.O.	81	M.	8 days	Sudden dy. and pain.	„	Yes.	I.	100-150	S.	Good.	Sl. E.	Senile arterio-sclerosis.	AT.
F.S.M.L.	63	M.	1	Palpitation.	Nervous feeling. Dy. ex.	No.	R.	100-150	S.	Not observed.	E.	Arterio-sclerosis.	AT.

Ex. = On exertion. Dy. = Dyspnœa. V.s. = Very slight. I. = Irregular. R. = Regular. S. = Systolic. N. = Normal. F. = Attacks of fibrillation. St. = Using strophanthus habitually. M. = Moderately. Sl. = Slightly. E. = Enlarged. T. = Typical. AT. = Atypical.

of the R-waves, so that the lesion must be considered a serious one, if not complete.

The four patients with typical records complained of severe pain over the heart, and also showed greatly increased blood pressure. In the other four there was no increase in blood pressure of any extent, and there was a history of pain in only one person. The orthodiagrams of the hearts of those with typical records all revealed markedly enlarged hearts of the aortic type. Only two of the patients with less typical histories showed this enlargement, the other two having enlarged hearts of the vertical type.

As the table shows, only one patient was under 50 years of age in this series, he being 48. All but

one of the eight were males. There was no uniformity in duration of disease, one man having a normal tracing two and a half years prior to the appearance of a typical bundle branch lesion. Dyspnœa and præcordial pain were present in each record, the præcordial pain being most marked in those with definite bundle branch tracings. These attacks, which were intermittent, came on usually by exertion. In one person it was the chief complaint which brought him for examination. Only two of the typical bundle branch cases were bothered with cough—brought on, no doubt, by some congestion in the lungs. The heart valves were not primarily involved in one-half of the patients.

CARDIOSPASM

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If the process of deglutition is observed radio-scopically in the normal individual, delay at the cardiac sphincter is only just sufficient to be perceived. The condition known as cardiospasm implies the overaction or too persistent action of this sphincter, resulting in derangement of the mechanisms of swallowing, and eventually gives rise to various pathological changes in the œsophagus.

Ætiology.

The ætiology of the condition is doubtful. It was at one time regarded as a manifestation of hysteria, but it does not appear to be especially common in the neurotic, and certainly cannot be cured by treatment of purely suggestive value. A history of vague dyspepsia frequently precedes its onset, and in about one-tenth of the cases gross disease of the stomach, such as carcinoma or ulceration, is found to occur coincidentally. It is possible to discover an analogy between this chronic spasm of the cardiac sphincter and the acute spasms of other parts of the alimentary canal observed in certain abdominal conditions—in both cases a protective mechanism which retards the progress of food towards the site of a lesion. On the other hand, one might conjecture that an interruption in the wave of contraction of the œsophageal musculature could be caused by quite a small island of sclerosis, as happens in the muscle of the heart in heart-block.

Stages of the Disorder.

Three stages of this disorder have been described. Discomfort—seldom amounting to actual pain—during a meal is the first symptom. It is referred to

the lower part of the sternum, and may occur several times during a single meal and then pass completely away for days, or weeks, or months, tending to recur at shorter and shorter intervals. This phase represents either the painful overaction of the sphincter and muscular coats of the œsophagus or indicates some lesion, possibly quite trifling, of the lower end of the gullet. Characteristic of the second stage is the sudden and explosive vomiting of small quantities of food during meal-times. This also is very variable in its incidence, occurring perhaps two or three times during a single meal and then ceasing for several days or longer. In this stage the gullet, unaccustomed to the retention of food, precipitately returns one or two mouthfuls when difficulty in their transmission arises. In the third stage the œsophagus becomes more tolerant of retaining food which it can no longer successfully propel. Vomiting is less spontaneous and less frequent, but when it occurs a larger amount of non-acid material is brought up. This may be retained for many hours, and examination of the vomit may show the retention of fragments of some particular meal for many days.

At this third stage the peristaltic action of the gullet is ineffective, and the passage of food through the sphincter depends largely on the weight of the column of food collected. When this pressure exceeds the resistance of the sphincter the food tumbles into the stomach and for the moment relief is felt. Radio-scopically the altered shape and function of the gullet can be very easily observed. Instead of a tube patent only when food passes down it, the œsophagus has become a bag—an accessory stomach, as it were, never entirely empty, with walls hypertrophied, so far as the muscular element is concerned, and exhibiting peristalsis only in their upper part. The movements communicated from the pulsation of the heart can be observed. In extreme cases the dilatation may rise above the xiphisternum, while in its lower part it