acid is present, especially in excess, that ulceration is simple. If hydrochloric acid be absent, it is malignant.

I think, from trial of numerous cases, these microscopic and chemical investigations do help in the diagnosis between ulcer and early cancer, though I am far from suggesting they do not err.

In the majority of cancer cases the free hydrochloric acid is absent, at any rate after a proteid meal, and in the few where it is present it may be that malignant disease is grafted on the seat of an old ulcer.

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Remarks

ON

THE PATHOLOGY OF BRADYCARDIA.

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THERE is a form of bradycardia termed "paroxysmal" or "intermittent." Two varieties are described: (1) In which the pulse is permanently infrequent, and in which there are sudden accesses of greatly-diminished frequency; (2) where the pulse is of normal frequency between the attacks.

SUMMARY OF CASE.

The case which I shall now relate belongs to the first variety. The following are the important points to be noted in reference to it:

- (a) Marked bradycardia, the pulse frequency ranging from 17 to 30 per minute, "a pouls lent permanent."
- (b) Transient loss of consciousness, associated with cessation of the pulse at the wrist.
- (c) Auricular contractions occurring twice or thrice as frequently as the ventricular, the cause of the bradycardia being "heart block."
- (d) An enormous heart, the autopsy revealing no cause for the hypertrophy and dilatation.

I give a brief summary of the case:

R. M., aged 25, was healthy till May, 1898, when he suddenly lost consciousness. Some time later he was admitted to the Royal Infirmary under the care of Dr. Barr, to whom with Dr. Raw I am much indebted for permission to publish this case. On admission he complained of "breathlessness on exertion and attacks of unconsciousness." On January 8th, 1900, he entered Mill Road Infirmary suffering from extreme cardiac failure. He died on January 15th, 1900.

When he was in the Royal Infirmary I noted the following points:

1. The apex beat was situated well outside the nipple line, slow, heaving, and forcible in character, 20 beats per minute.
2. Pulsations or small beats, numbering 50 or 60 per minute, occurred regularly over the third left interspace, just to the left of the sternum.
3. Synchronous pulsations were observed in the veins of the neck.
4. Definite systolic retraction in the third, fourth, and fifth left spaces.
5. Cardiac dullness (deep) extended 7 in. to the left, 2 in. to the right of the middle line.
6. A systolic murmur at all areas, loudest and harshest over the conus of the right ventricle. It was conducted to the left and well heard along the vertebral border of the scapula.
7. No sounds were audible accompanying the small pulsations mentioned above.
8. Pulse varied from 17 to 30 per minute.

Fig. 1 is a pulse tracing. A cardiogram was taken simultaneously with a record of the venous pulse in the neck. The

Fig. 1.—Pulse tracing taken by Dr. Barr. Rate, 20 per minute.

diagram (Fig. 2) is an accurate representation of the tracings. The cardiogram demonstrates the slow rhythm of the ventricle and the more frequent rhythm of the auricles. When the contraction of the auricles coincides with that of the ventricles it is masked by the record of the ventricular

Fig. 2.—v = wave caused by ventricular systole; a = wave caused by auricular systole. At a' the auricle contracted, but its record is masked by the larger record of the ventricular systole, which occurred at the same moment.

contraction. The waves in the venous pulse tracing are synchronous with those of the cardiogram. The tracings show, therefore, the two independent rhythms, auricular and ventricular.

The pulsations visible over the conus of the right ventricle were due, in my opinion, to the strong right auricle forcing blood through into the right ventricle, the ventricle being in diastole and permitting the impact of blood to make itself evident by a slight movement of the chest wall over the conus.

Later, when admitted to Mill Road, it was noticed that the cardiac dullness (deep) had increased to the right by 1 in., that the only murmurs persisting were a marked tricuspid regurgitant and a faint mitral regurgitant, and that the frequency of the pulse had reached 40 to 80 per minute.

NECROPSY.

The necropsy, to my astonishment, gave little assistance. There was some cyanosis of the lips and ears, slight general jaundice, and a little puffiness about the ankles. The chart of the deep cardiac dullness, dated January 14th, was mapped out on the chest, and long needles were inserted at right angles to the surface. To the right the needle was found to have entered the pericardial sac without touching the heart; at the apex the needle just entered the edge of the left ven-

Fig. 3.—Diagram drawn by Professor Keith. It compares the heart of the patient with a normal heart, and demonstrates the condition present at the auricular canal. (Drawn to scale.)

Fig. 4.—Simultaneous tracings of the radial and jugular pulses, showing that the a-c interval (space A) is twice the normal period (two-fifths of a second in place of one-fifth), indicating a delay in the passage of the stimulus from auricle to ventricle. It shows also that only every other systole of the auricle is followed by a systole of the ventricles. x = murmur due to ventricular systole, mitral regurgitation; y = murmur produced by auricular systole discharging blood into an almost empty ventricle. The time marking here represents one-fifth second. The space z indicates the period of ventricular systole during which the semilunar valves are open—the sphygmic period.

tricle, thus demonstrating that the deep cardiac dullness as elicited *ante mortem* was correct.

The pericardium was normal, and contained 1 oz. of clear fluid. The heart weighed 29 oz., and showed marked hypertrophy and dilatation. The valves were all translucent, and of a normal appearance. The tricuspid and mitral valves were incompetent. The lungs were tough and doughy, and comparatively pale and dry. There were old pleural adhesions, and there were two pulmonary apoplexies in the right lower lobe. The liver was engorged and nutmeg. The kidneys weighed 15 oz., and on microscopical examination were found to be normal, except for signs of back pressure. There was no evidence of arterio-sclerosis.

Some weeks ago I sent the heart to Professor Arthur Keith, of the London Hospital, for his opinion. With exceptional kindness he has drawn a diagram showing its condition, and more particularly that of the auriculo-ventricular junction. His method of examining hearts may be as new to others as it was to me. I therefore reproduce the diagram (Fig. 3). From his report I quote the following:

"The two marked changes in your specimen (besides the hypertrophy and dilatation of the chambers) are: (1) Stretching of the auricular canal (the part normally within the auriculo-ventricular groove); (2) separation, or rather attenuation, of the bond between the bases of the valves (tricuspid) and base of the ventricle." These points are well shown in the diagram.

REMARKS.

Here is an undoubted case of "heart block." Normally the stimulus to contraction is initiated in the remains of the sinus venosus, consisting of the musculature at the mouth of the great veins and adjoining portion of the right auricle. The wave of contraction spreads over the auricle and through the auricular canal on to the ventricle "with varying speed, quickest over the tissue which has become modified so as to approach more nearly in its properties to ordinary striated muscle—namely, the reticulated bulged portion of the auricle and ventricle; more slowly over those parts which retain a more embryonic character—namely, the auriculo-ventricular muscular ring and the bulbus arteriosus."¹

Normally the ventricle responds to a stimulus spreading down to it from the auricle; if by any means the ventricle is cut off from this normal periodic stimulus it tends to initiate its own stimulus and to take on a rhythm different from and independent of the auricle. Gaskell demonstrated this in the frog's heart by ligaturing the auriculo-ventricular groove so that no stimulus could cross from auricle to ventricle. The term "heart block" is applied to this condition.

A heart in which the auricle dominates the rhythm as a rule beats with normal frequency. When the stimulus for ventricular action arises in the ventricle itself there is a slow rhythm. The result of "heart block" is therefore a bradycardia. Such a condition was present in this case; the block in all probability occurred at the attenuated portion of the ventricular wall situated at the base of the ventricle at its junction with the auricular canal.

An interesting question arises as to the cause of the great hypertrophy and dilatation of the heart. It is too great to be accounted for satisfactorily by the strain of work. It was not due to chronic nephritis; there was no sclerosis of the valves. The incompetency of the mitral and tricuspid must be in some degree responsible, but this pertains rather to the later stages than to the period of comparative compensation, when, as I have already noted, there was a transverse cardiac dullness of 9 in. I venture the following suggestion:

Since during each ventricular diastole the auricles contract twice or thrice, the resulting condition of the ventricles would be one of over-distension, and they would during their systole have an excessive amount of blood to deal with. The strain

on the ventricular walls due to the forcible pumping in of blood by the auricles would produce dilatation, and the large volume of blood dealt with would necessitate hypertrophy.

The discussion of this case would be incomplete if it did not include a brief reference to the "attacks of unconsciousness" of which the patient complained. Twice on March 23rd, 1899, it was noted that during an attack of dizziness his pulse became imperceptible, returning gradually to its normal rate of 20 a minute. I believe that this explains the loss of unconsciousness, and brings this case into line with the more pronounced forms of the "Adams-Stokes syndrome."

In this case such attacks were infrequent and not at all pronounced; convulsions in relation to them were not noted at any time, but, with the notes before us, I think I am justified in assuming that the dizziness and fainting were due to a temporary cessation of ventricular contraction.

A case, somewhat similar but much more marked, is recorded by A. Webster.² His patient complained of taking what he called "weak turns associated with palpitation, sensations of giddiness, and occasional loss of consciousness." The record is illustrated by a series of most beautiful tracings demonstrating the disappearance of the radial pulse coincident with the dizziness and loss of consciousness.

We may take it, then, that in the case I have described the bradycardia was due to a condition of heart block so complete that the auricles and ventricles beat independently of each other.

There are other cases, however, in which the block in the auricular canal is not complete, and where the ventricles respond to every second contraction of the auricles, or even to every third or fourth contraction. Quite recently I met with such a case in which the bradycardia was caused by a severe depression of conductivity, the ventricle failing to respond to every second contraction of the auricle. Two similar cases are described and figured by Mackenzie³ in his recent excellent paper in the *BRITISH MEDICAL JOURNAL* of March 11th, 1905.

The patient I refer to is 62 years of age. His occupation up to the present illness was that of a baker. This entailed the carrying of heavy loads up and down stairs. He denies syphilis or alcoholic excess.

The present illness began three years ago with a sudden loss of consciousness. Since then he has occasionally suffered from "mazy bouts." The doctor in attendance tells me that when he first saw him eighteen months ago he noticed that the pulse was 20 a minute; there was then some bronchitis, with considerable dyspnoea; the extremities were cold, but there was no cyanosis or oedema.

Since that time his pulse has remained persistently infrequent, and the bradycardia probably dates from then.

I was surprised by the healthy appearance presented by the man. He had a good colour, and was well nourished, but I noticed signs of deficient aëration of the blood in the hands, which were very cold, and, according to the patient, became livid and numb on the slightest exposure. He felt perfectly well with the exception of great dyspnoea on exertion. The "area of cardiac response" was very limited. There was marked emphysema of the chest. The radial and temporal arteries were uniformly thickened and tortuous. The pulse was 32 to the minute, regular in force and frequency, of large volume and good tension. I noticed two beats in the neck to each radial pulse, and took tracings to demonstrate this (Fig. 4).

On analysing these records it will be seen that the waves due to auricular systole occur at regular intervals, and that every other such wave is followed by the carotid pulse. It is almost impossible to record the venous pulse in the neck without at the same time recording the carotid. This is rather an advantage than otherwise, as it gives a fixed point in the analysis of the tracing. In the record of the jugular pulse the distance from the beginning of the contraction of

the auricle to the first appearance of the carotid beat corresponds roughly to the time between the auricular and ventricular contraction. This interval is called the *a-c* interval, and is normally about one-fifth of a second in duration. Where, however, there is depression of conductivity, and the stimulus finds greater difficulty in passing from auricle to ventricle, this *a-c* interval is lengthened. It is so here; instead of being one-fifth of a second it is two-fifths of a second. An examination of the tracing shows, first, an auricular contraction *a*, followed by the carotid pulse *c*, and that the *a-c* interval is double the normal duration; then we meet with another wave *a*, due to a second contraction of the auricle, but this time there is no sign of a ventricular response. The stimulus has not been able to reach the ventricle. This sequence is repeated without variation in all the tracings.

Fig. 5.—Diagram constructed to show the action of the heart when Fig. 4 was taken.

To demonstrate the action of the heart when Fig. 4 was taken I have constructed the diagram Fig. 5. It is built on the plan adopted by Wenckebach and Mackenzie. The down strokes in the upper division, *A S*, represent the auricular systoles and correspond with the waves "*a*" in Fig. 4, while the down strokes in the lower division, *V S*, represent the ventricular systoles, and correspond to the radial and carotid pulses in Fig. 4. The short, thick lines connecting these down strokes represent the passage of the stimulus through the fibres joining auricle and ventricle. In this case they are two-fifths of a second in duration—that is, double the normal length. The absence of this line indicates that the stimulus has failed to pass these fibres, and hence a ventricular systole drops out.

I also obtained a tracing combining a cardiogram and a sphygmogram, which demonstrates that in this case every ventricular contraction is represented by a pulse wave in the peripheral arteries. The heart is little, if at all, enlarged, the deep cardiac dullness measuring $1\frac{1}{2}$ in. to the right and 4 in. to the left of the middle line. On auscultation I found that it was difficult to hear the heart sounds at the base owing to the emphysema. At the apex I made out a wheezy systolic bruit, followed by a clear second sound, and then during the diastole of the ventricle, and synchronous with the extra pulsation in the neck I heard a short, whiffing murmur, due, I take it, to the auricular systole forcing blood into the partially-filled ventricles.

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- ² A. Webster, Cardiac Arrhythmia in Relation to Cerebral Anaemia and Elliptiform Crises, *Glasgow Hospital Report*, 1901.
- ³ J. Mackenzie, New Methods of Studying Affections of the Heart, *BRITISH MEDICAL JOURNAL*, March 11th, 1905.

British Medical Association.

CLINICAL AND SCIENTIFIC PROCEEDINGS.

BIRMINGHAM BRANCH.

Thursday, October 12th, 1905.

Sir THOMAS CHAVASSE in the Chair.

Slough from the Stomach.—Dr. SHORT showed a large slough, measuring 7 in. by 5 in., which had been vomited from the stomach of a woman aged 30 eight days after she had swallowed about an ounce of sulphuric acid. The slough was so long that part of it might have come from the lower part of the oesophagus, but the width made it evident that the greater part must have come from the interior of the stomach, and appeared to have stripped the mucous membrane and part of the muscular coat. Fortunately perforation had not occurred, and the woman seven weeks after the accident was up and about. The stomach was now dilated, and there was evidence of some obstruction to the free emptying of the viscous, with great diminution if not complete destruction of its digestive powers.

Resection of Carcinoma of the Caecum.—Mr. MORRISON showed a man, aged 44, from whom he had removed, on September 15th last, in the Queen's Hospital, a cancerous caecum, together with adjacent portions of ileum and ascending colon—8 in. of bowel in all. The patient had been previously under the care of Dr. Power of Atherstone, who had attended him for several attacks closely simulating appendicitis. The presence of a painful lump in the right iliac fossa confirmed this view, and removal of the appendix was advised. On opening the abdomen, however, a malignant growth was found in the caecum close to the appendix, and there were enlarged infected glands in the mesentery. Local peritonitis had glued together two points in the ascending colon several inches apart, the intervening portion of gut forming a loop resembling the sigmoid flexure. The diseased area having been resected, the colon was opened laterally above this adhesion, and junction with the cut end of ileum effected by means of a Murphy button. Infected glands were removed. The free end of the large bowel was closed by a double row of fine silk sutures. Provision having been made for drainage of the extensive raw space left by the operation, the rest of the wound was closed. The button was passed on the twentieth day—October 4th. The growth, which was a columnar-celled carcinoma, was also shown.

Endemic Dysentery.—Professor SAUNDY read a paper on endemic dysentery, or so-called ulcerative colitis, in which he drew attention to the occurrence from time to time of sporadic cases of dysentery. These cases were frequently overlooked, as was pointed out by Dr. Hilton Fagge, on account of the absence of marked symptoms; but in other cases they were called ulcerative colitis, a term which had received acceptance by the profession without due ground. Dysentery had always been recognized as a disease of this country; and although improved sanitary conditions had tended to make it rare at the present day, it undoubtedly occurred at times. The opinion that there were two forms of dysentery, one belonging to temperate climates and the other confined to the tropics, would not stand examination, while the doctrine of amoebic dysentery, upon which the supposed specific difference of tropical dysentery rested, had not been strengthened by the observations of the last few years. Modern researches had tended to show that the bacillus discovered by Shiga in the dysentery of Japan, was the cause of dysentery all over the world, as it was found by Flexner in the Philippine Islands, by Kruse in Germany, and by Vedder and Duval in the United States. The bacteriology of faeces presented great difficulty, and therefore it was not altogether surprising that the researches made into the bacteriology of asylum dysentery by Goodliffe and by Durham did not arrive at any definite result. Professor Saundby had been equally unsuccessful in several of his cases, but at length Mr. Hewetson had found the bacillus Shiga in the stools of a case of dysentery recently under the care of Professor Saundby at the General Hospital. The paper was illustrated by preparations and photographs showing the state of the intestine, and by microscopic specimens, photographs, and culture tubes showing side by side the bacillus isolated by Mr. Hewetson and the bacillus Shiga. This case was believed to be the first in which the bacillus Shiga had been identified in this country. Professor Saundby pointed out that as the bacillus Shiga gave the serum reaction (Widal reaction) when brought in contact with a drop of blood from a patient suffering from dysentery, it afforded a means of identifying the disease which ought to aid in the early diagnosis of these cases. In the discussion that followed, Drs. NEWTON, MALET, KAUFFMANN, VOKES, and HEATH took part; and Dr. SAUNDY replied.

The Roentgen Rays in Diagnosis.—Mr. HALL-EDWARDS then read a paper on Recent Advances in the Application of the Roentgen Rays to Diagnosis and Therapeutics, under the following heads: The use of the diaphragm and compressor in the diagnosis of calculi and of diseases of the hip-joint; the measurement of dosage in the treatment of various diseases; the treatment of ringworm; the action of the rays upon the blood; the early diagnosis of phthisis; the treatment of eczema, acne, and malignant disease. The paper was discussed by Drs. SHORT and HEATH, and Messrs. MORRISON and HEATON, and Mr. HALL-EDWARDS replied.

REQUESTS TO MEDICAL CHARITIES.—The late Miss Mary Elizabeth Wilson, of Calverley Park, Tunbridge Wells, whose will has now been proved, bequeathed £100 each to the Eye and Ear Hospital, the Homoeopathic Hospital, and to the General Hospital, all in Tunbridge Wells.