

7 per cent., and urea $6\frac{1}{2}$ grains per ounce. There were found three medium-sized hyaline casts.

June 7: Albumin entirely gone, urea 6 grains to ounce, chlorids 9 per cent. The twenty-four hour quantity fluctuated between 50 and 100 ounces, averaging 70 to 80 ounces.

Bacteriologic Examinations.—May 15: Widal test for typhoid, negative. On June 1 a blood examination showed the leucocyte count 20,650 per mm., increase confined to the polynuclear leucocytes. It was an inflammatory leucocytosis, probably septic, not enough to warrant a diagnosis of pus formation. On June 5 a bacteriologic examination of specimens of pus taken from swabs of diseased surfaces at time of operation on previous day showed only the staphylococcus pyogenes aureus.

Dietetic Treatment.—It was realized from the outset that the patient was suffering from a complication of diseases of a most serious nature. The attempt was made, therefore, to secure the assimilation of a sufficient variety and quantity of food to fully support the vital forces. To do this without overworking the renal organs added to the difficulties. The patient was supplied with, and perfectly assimilated from 2,000 to 4,000 calories of mixed foodstuffs during each twenty-four hours of his sickness. During the entire period he had but two dejections containing undigested particles in sufficient quantity to be determinable by macroscopic examination. He was so well nourished as to occasion approving comment of the professional friends who saw him.

Medicinal Treatment.—The first day he had, in divided doses, two grains of calomel, which acted mildly. On the second day he had one grain of elaterium, divided in eight doses. This was followed by free watery dejections, with immediate relief from uremic symptoms. On the fourth day he had one dose (by rectum) of 20 grains of chloral hydrate and 60 grains sodium bromid, which was followed by several hours of refreshing sleep. At this time and continuing for several days, he had 20 grains of potassium citrate every four hours, alternating with one-drop doses of a 1 per cent. solution of glonoin. From June 3 to June 10 he had no drugs. From this date until the end he had strychnin sulphate, $1/40$ to $1/30$ grain hypodermatically at three and four-hour intervals.

The surgical conditions and treatment have been given in detail in the history of the case.

THE BLOCKING OF AURICULAR EXTRASYSTOLES.

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The newer methods of analyzing the venous pulse have enabled us to differentiate clinically a number of forms of cardiac arrhythmia that could not otherwise be properly classified. So far as I am aware, arrhythmias due to a blocking of extrasystoles on their passage from the auricles to the ventricles have not been described. I am able to report such a case here through the kindness of Dr. T. W. Huntington.

Patient.—P. C., clergyman, aged 56, entered the Lane Hospital for operation on a left inguinal hernia.

History.—His family history was excellent and his past history contained little of note. He had been twice poisoned by eating mussels, the last time five years ago, but had never had a severe illness. He habitually drank a cup of coffee in the morning, a cup of tea at noon, and an occasional glass of wine at meals; he did not use tobacco. He first noticed some irregularity of the heart three months previously. He did not know what brought it on nor what made it worse, and was conscious of his heart action only when considerably excited. He had no shortness of breath on exertion; no swelling of the feet; no attacks of dizziness; no cough, etc.

Examination.—This showed a rather nervous, fairly well-nourished man. The right pupil was slightly larger than the left; both reacted to light and on accommodation. Lungs, abdomen, and reflexes were negative.

Heart and Vessels: The point of maximum impulse was in

the fifth intercostal space, 2 cm. outside of the mammary line and 12 cm. to the left of the median line. The area of cardiac dullness extended from the upper border of the third rib to 12 cm. to the left and 3 cm. to the right of the median line. The heart sounds were clear, the first being of a booming quality at the apex; the second at the pulmonary area was accentuated and reduplicated. The radial arteries were markedly thickened and contained irregular hard plates. The temporal arteries were also thickened and tortuous. Pulse: The rhythm was interrupted at times by small premature beats, followed by longer compensatory pauses; at other times there was simply a pause in the regular succession of beats without any palpable premature pulse. These pauses were sometimes so frequent as to produce for a short time a slow and perfectly regular rhythm (Fig. 2). One might have suspected that he was dealing with an ordinary case of extrasystoles, some of which produced a radial pulse, while others did not. Auscultation over the apex, however, showed complete silence during most of the intervals when no extra pulse was felt; whereas, as is well known, extrasystoles affecting the ventricles invariably give rise to a well-marked first sound.

Sphygmography.—In Figure 1 are seen tracings of the radial pulse, the apex beat, and the jugular pulse during a comparatively normal period. The duration of the normal pulse interval is about 0.85 second. The jugular tracing shows the three characteristic waves: *a*, due to the contraction of the auricle; *c*, the transmitted carotid pulse; and *v*, following the contraction of the ventricle. The regular

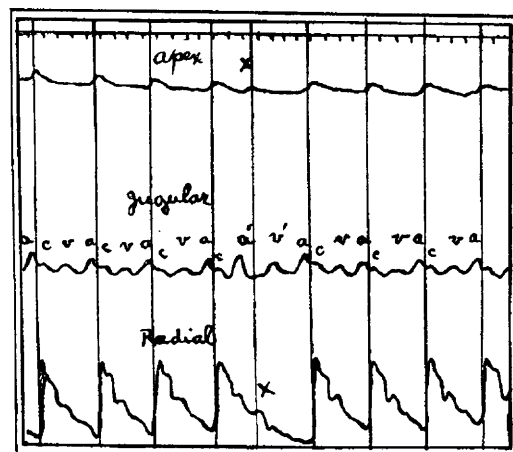


Fig. 1.—Normal pulse rate, interrupted at *x* by an extrasystole, which starts in the auricles and spreads to the ventricles.

rhythm is interrupted by an extrasystole at *x*. This extrasystole shows slightly on the rather poor apex tracing, and slightly also on the radial tracing. On the jugular tracing it is indicated by an unusually high wave *a'*, which begins a little earlier than the *v* wave, which should follow the preceding normal systole. The early occurrence of this wave and its height lead us to believe that it is due, at least in part, to a premature auricular contraction, and possibly in part to the *v* wave from the preceding normal systole. The carotid pulse following this extra-auricular contraction is so small as not to be visible on the tracing from the neck, but the radial tracing shows that a small extra pulse was present. The apex tracing and the occurrence of a *v'* wave on the jugular tracing likewise bear witness to the occurrence of an extrasystole of the ventricle. This tracing shows, therefore, a normal pulse rhythm interrupted at one point by an extrasystole. The latter originates in the auricles and spreads thence to the ventricles.

The radial pulse in Figure 2 might lead one to suspect a slow and regular heart action were it not that the apex tracing and a closer inspection of the radial tracing also demonstrate the presence of extrasystoles, *x*. It is possible to have ventricular extrasystoles interpolated between the normal beats of a slowly beating heart without there being any compensatory pauses.¹ On such an assumption the normal pulse interval in this tracing would be 1.5 seconds; but from Figure 1, which is a continuation of Figure 2, on the same piece of paper, we have

1. Pan (O.); Deutsch. Arch. f. klin. Med., lxxviii, 128.

already learned that the normal interval is 0.85 second. From a comparison with this earlier tracing, it also becomes apparent that those extrasystoles of Figure 2 involving the ventricles are exactly the same as the solitary extrasystole of Figure 1. They all originate in the auricles and spread thence to the ventricles. In two places on Figure 2, however, no extra-ventricular contractions are interposed between the normal beats. This appears not only from an inspection of the apex tracing, but from the complete absence of heart sounds previously referred to. A study of the jugular pulse shows the cause of this absence. The ordinary extrasystole in this patient shows itself on the jugular pulse by a well-marked a' wave, due to the premature contraction of the auricle, and also by the c' and v' waves, due to the contraction of the ventricles. During these intermissions, however, the high a' wave alone appears. The auricles contract prematurely, but no contraction of the ventricles follows.

The reason why the ventricle fails to contract may be studied in Figure 3, which by a fortunate coincidence shows on a rapidly moving paper the three main features of this pulse, viz.: The normal pulse, the extrasystole crossing to the ventricle, and the extrasystole that affects the auricles only. The interval between any a wave (contraction of the auricle) and the succeeding c wave (carotid pulse), the a - c interval, as it is called, has been taken by Mackenzie to measure the rate of transmission of the cardiac contraction wave from the auricles to the ventricles. Its normal duration is 0.2 second or less; and if it

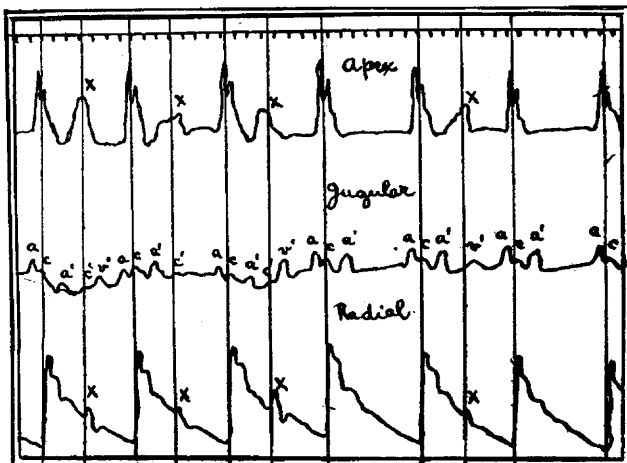


Fig. 2.—Each normal pulse is followed by an auricular extrasystole. In those marked x the extrasystole spreads from the auricles to the ventricles; in the remaining two the contraction wave does not affect the ventricles.

be longer than this, Mackenzie considers that the conductivity across the His bundle is impaired. The a - c interval for the normal beats in my patient was 0.18 second, or about normal. The a - c interval for the extrasystoles on the other hand was 0.35 second, a very considerable prolongation (0.17 second beyond his normal); yet the prolongation here was due only in part to a loss of conductivity. A comparison of the apex and jugular tracings shows that the interval between the beginning of the a wave on the jugular tracing and the beginning of the ventricular contraction on the apex tracing is 0.13 second in the normal beat and 0.21 second in the extrasystole; a lengthening of 0.08 second. The remainder of the lengthening of the a - c interval is due to the fact that an abnormally long time elapses between the beginning of the ventricular contraction and the beginning of the carotid pulse (prolongation of the period of ventricular tension). Thus in Figure 3, the interval between the beginning of the ventricular systole and the beginning of the normal carotid pulse is 0.05 second, while the same interval for the extrasystole is 0.14 second, a difference of 0.09 second. At least two factors tend to lengthen the period of tension, both of which depend on the early occurrence of the abnormal contraction. In the first place, the aortic pressure is highest just after any systole so that the earlier the succeeding contraction the longer it will take for the intraventricular pressure to reach such a height as to exceed the aortic pressure and

to open the semilunar valves. In the second place, the contractility of the ventricle is reduced after a systole and it seems reasonable to assume that a weakened ventricle should take a longer time than a rested one to produce a certain pressure.

How are we to explain the complete absence of certain ventricular contractions? Two possibilities present themselves: 1, A lack of conductivity from the auricles to the ventricles; 2, a lack of contractility on the part of the ventricles. We have just seen that where the ventricles take part in the extrasystoles both conductivity and contractility are diminished. The diminution of conductivity is apparent from the lengthening of the interval between the contraction of the auricles and the contraction of the ventricles. The diminution of contractility is evidenced by the prolongation of the period of tension. A still further reduction of either of these might lead to a falling out of the ventricular systole; but it is difficult to decide which is actually most at fault in this patient. One fact mitigates against its being a lack of contractility. In the many times I listened to the irregular heart action I have never heard an extrasystolic first sound that was not followed by a second sound, which means that the ventricular contractions, when present, were always of sufficient force to send blood into the aorta or pulmonary artery. Had the falling out of the extrasystoles been due to a lack of contractility we should have expected to find transition systoles of such force that, although the ventricle contracted, the

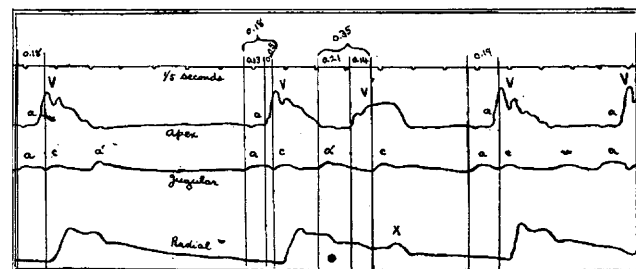


Fig. 3.—Record on a rapidly moving piece of a paper. First a normal contraction followed by an extra-auricular contraction, but not ventricular contraction; second, a normal contraction followed by an ordinary auricular extrasystole; third, a normal pulse without an extrasystole.

semilunar valves were not opened. The apparent absence of such extrasystoles causes us to favor the view that the falling out of ventricular contractions was due, not to a lack of contractility, but to a failure of impulses to cross the auriculoventricular junction.

The slowing or failure of the conduction during the extrasystoles is due to the occurrence of the latter so immediately after the preceding normal beats. The interval was not sufficiently long for the conductivity of the His bundle to return to its normal level. A similar observation has been made on hearts whose conductivity was but slightly diminished. In such hearts every contraction wave may pass from the auricles to the ventricles so long as the rate is a slow one; but if the rate becomes accelerated, one or more waves are blocked at the auriculoventricular junction.

Syncope in Biliary Colic.—Maggard, in the *Journal of the Kansas Medical Society*, states that the syncope which sometimes attends biliary colic is best treated by atropin, strychnin, normal salt solution and brandy. Ether is a valuable heart stimulant and should be injected deeply when given hypodermatically in order to prevent the severe pain which it produces when injected superficially.