A CASE OF INDEPENDENT VENTRICULAR ACTIVITY OCCURRING DURING ACUTE ARTICULAR RHEUMATISM *

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History.—S. P., male, aged 20, entered the Har Moriah Hospital, Feb. 4, 1913. He had measles when 3 years old and typhoid when 10; otherwise there was no history of any previous illness. He was not addicted to tea, coffee, tobacco or alcohol. Five days before admission he developed a typical attack of acute articular rheumatism involving the ankles, wrists, knees and elbows. The attack was accompanied by fever; there were no chills or gastric disturbances.

On admission, except for swelling and redness of the inflamed joints, the physical and neurological examination revealed nothing abnormal. There was no urethral discharge. The complement-fixation test for gonococci was negative. The cardiac outline was normal to percussion, the apex beat was in the fifth interspace, 8.5 cm. from the midsternal line; the heart sounds were normal; the pulse was rhythmical. The systolic and diastolic blood-pressures were within normal limits. The temperature ranged between 101 and 103. There was no dyspnea. The patient did not appear very ill; sodium salicylate in moderate doses was given for two days.

Two days after admission a transient pulse irregularity appeared. Six days thereafter, it recurred once in about fifteen beats and clinically resembled extrasystoles; no tracings were made at that time. February 12, the irregularity occurred every third or fourth beat. From that day frequent polygraphic and later electrocardiographic tracings were taken. February 14, for the first time a rough blowing systolic murmur was heard at the apex. Occasionally there were runs of from three to twelve stronger thumping beats accompanied by the murmur; studies of the tracings showed that these beats were due to simultaneous action of auricle and ventricle. Four days later the arhythmia was very infrequent, the systolic murmur had almost entirely disappeared. The patient left the hospital feeling well.

Polygraphic and Electrocardiographic Tracings.—In Figure 1, *a, b, c* are continuous parts of polygraphic tracings taken February 12. In Figure 1 *a*, with the exception of three rhythmic beats (5, 6, 7 in the venous curve of Figure 1 *a*) independent ventricular action is present, as is evidenced by the carotid wave, *c*, falling with or preceding the auricular wave, *a*; one ventricular extrasystole (*r'*) is also present. Figure 1 *b* shows a similar arhythmia with varying *c-a* intervals and occasional simultaneous action of auricle and ventricle. The rhythm again becomes normal in the last section of the tracing (Fig. 1 *c* at *n*). The average pulse-rate throughout is sixty per minute, though the beats which inaugurate the normal rhythm (Figure 1 *c*, X' and X") are somewhat more rapid. The entire tracing is typical of those taken on the days when the arhythmia was marked (for instance, Fig. 2, *a* and *b*). One week later the pulse was rhythmical except for an occasional independent ventricular contraction (Fig. 3 at *1*), the rate about 57 per minute. February 26, the rhythm was normal, the rate 70. Subsequently, pressure on the vagi was practiced to study its effect on the rhythm. Left vagus pressure showed a transient increase of pulse rapidity (Fig. 4). During right vagus pressure (Fig. 5) there was slight temporary slowing of the pulse-rate. Neither right nor left vagus pressure had any effect on the normal auriculoventricular sequence. After sufficient time

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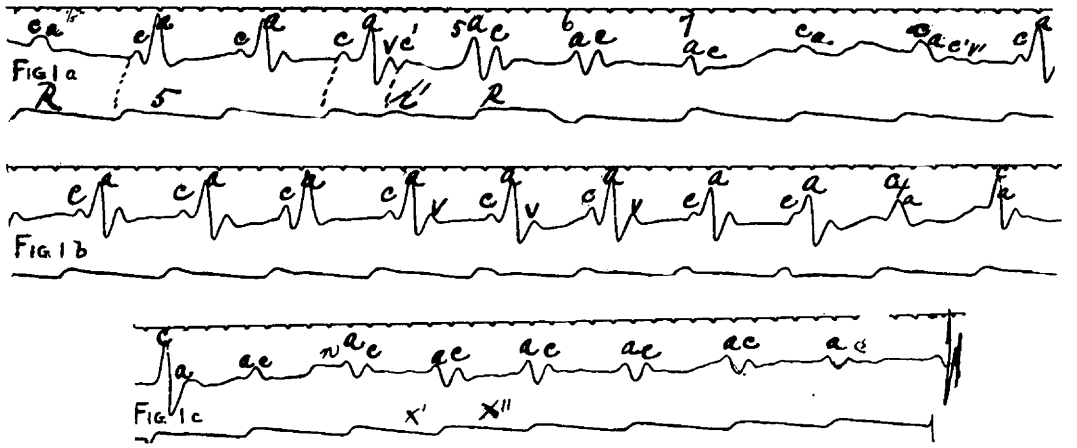

Fig. 1.—Continuous polygraphic tracing taken February 12. In Figure 1 *a*, except beats 5, 6 and 7 (venous tracing), independent ventricular beats are present; the carotid wave, *c*, precedes or falls with the auricular wave, a. R = radial pulse; r′ = ventricular extrasystole. Average pulse-rate is 60 per minute. In Figure 1 *b*, ventricular automatism with varying *c-a* intervals and occasional simultaneous action of auricle and ventricle (superposition of *a* and *c*) are present. Figure 1 *c*, rhythm becomes normal at *n*. X′ and X″ and somewhat more rapid beats which inaugurate the normal rhythm.

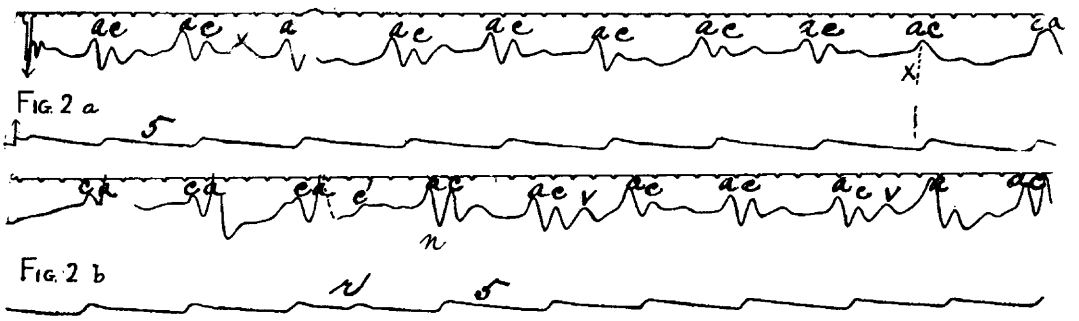

Fig. 2.—Continuous tracing taken February 13. The automatic beats begin at X. The rhythm is again normal at *n* (Fig. 2 *b*). The numerals on the radial beats represent their lengths in fifths of a second. Except for extrasystole (r′) the rhythm has been normal, the rate 60 per minute.

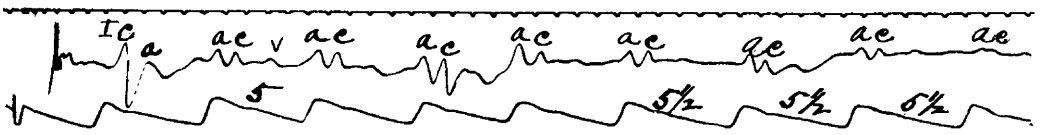

Fig. 3.—Section of a long polygraphic tracing taken February 19. It shows one independent ventricular contraction at I.

had been allowed for the heart to recover from vagus pressure, atropin sulphate, 1/30 grain, was injected subcutaneously and a continuous polygraphic tracing lasting one hour was taken, the important sections of which are reproduced (Fig. 6, *a* and *b*). They show that ten minutes after the injection ventricular automatism was occasionally present; the rate throughout was 60 per minute. Immediately preceding this arhythmia, the rhythm had been normal with an occasional increase of rate to 75 per minute. At the end of the atropin experiment the pulse-rate was 100, the beats sequential.

Many electrocardiograms were taken, two of which are given (Figs. 7 and 8). The rhythm is normal in the first lead (Figure 7, *a*). In the first section of

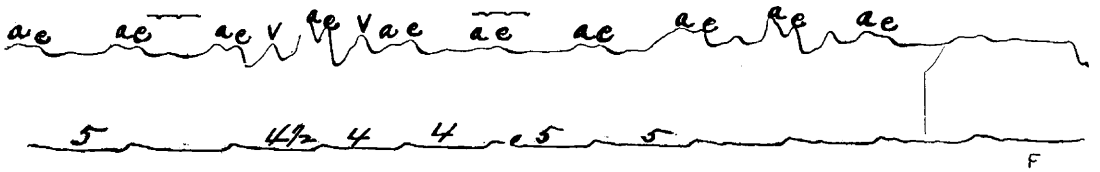

Fig. 4.—Left vagus pressure. It shows transient increase of the pulse-rate. The venous curve is somewhat distorted by the digital pressure on the vein.

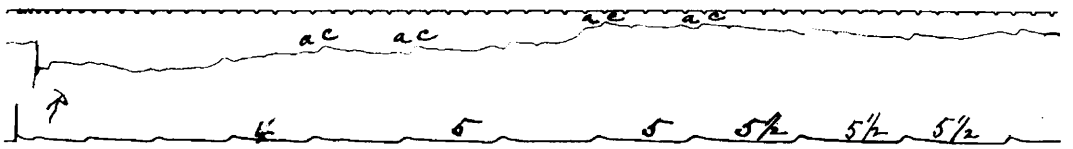

Fig. 5.—Right vagus pressure. It shows slight transient slowing of the pulse-rate; later the rate increased.

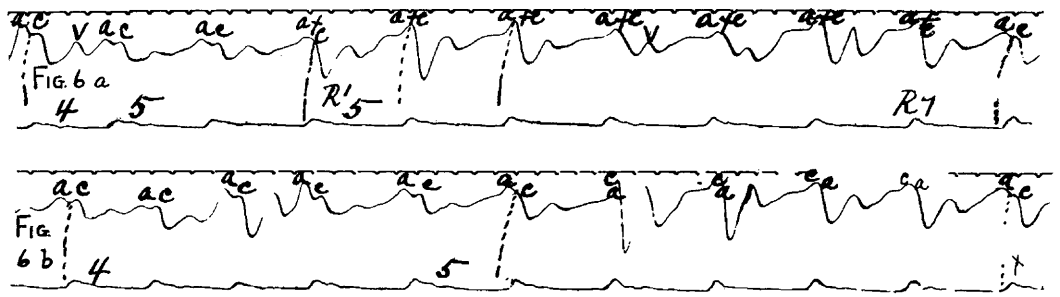

Fig. 6 *a*.—Ten minutes after subcutaneous injection of atropin sulphate, 1/30 grain. The incidence of *c* in the jugular tracing is shown by the dotted lines. Ventricular automatism is present from R^1 to R^7. The ventricular rate is 60 per minute. Fig. 6 *b*.—Fifteen minutes after atropin injection. It shows several automatic beats; the ventricular rate is 60 per minute.

the second lead (Figure 7, *c*), there is a slight difference in the length of the diastolic pauses, an arhythmia apparently of sinus origin. There are several automatic beats (Fig. 7, *c*, R^2, R^3, R^4) in the second part of the same lead. The beats are again sequential in the third lead (Fig. 7, *d*). Three days later, February 25, an electrocardiogram, second lead only (Fig. 8, *a* and *b*), was taken, ten and fifteen minutes, respectively, after the subcutaneous injection of 1/30 grain of atropin sulphate. With the exception of slight sinus arhythmia,

the beats are rhythmical, the ventricular rate about 60 per minute. Twenty minutes after the injection, independent ventricular activity with approximately the same ventricular rate is present (Fig. 8, *c*). In the later tracings (Fig. 8, *d, e*) normal auriculoventricular sequence is reestablished. It is important to note that the idioventricular and sequential complexes are identical throughout. Subsequent electrocardiographic tracings, the last taken three months after the onset of the arhythmia, show that the latter has not recurred.

COMMENT

Abnormal cardiac mechanisms similar to this have been ascribed to various causes. When auricles and ventricles beat at such rates that

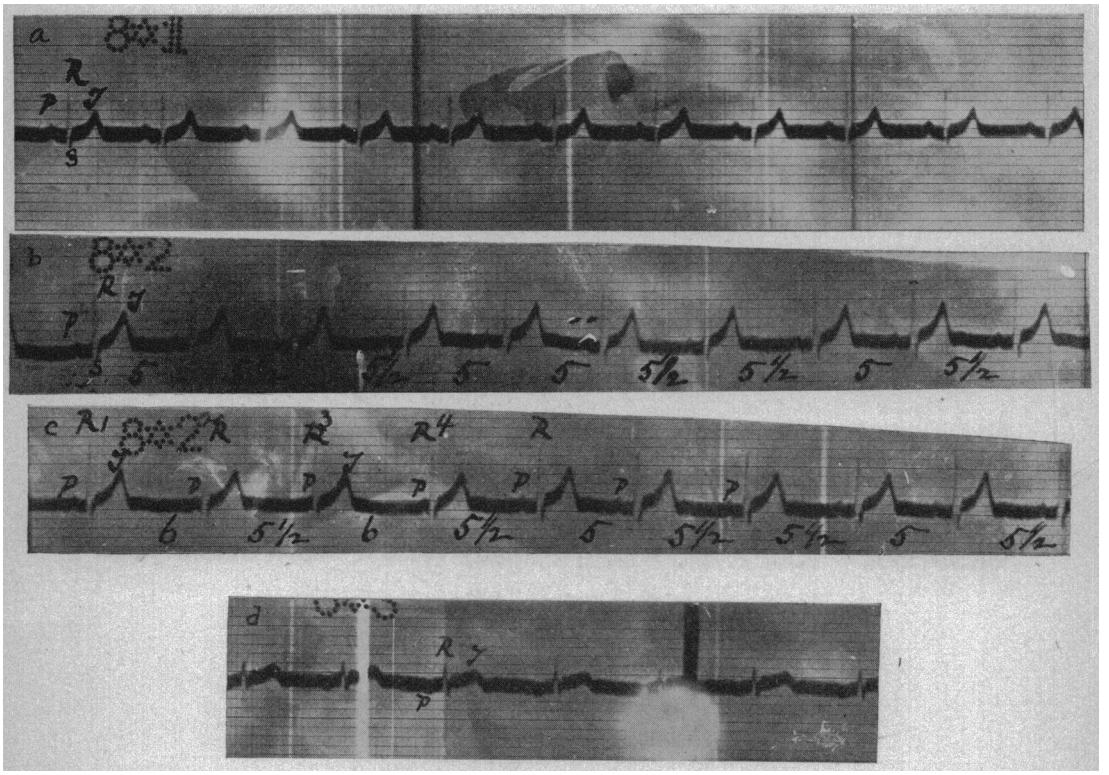

Fig. 7.—Electrocardiogram taken February 22. P = auricular deflection, R, S, T = ventricular complex. Fig. 7, *a*.—Lead 1 shows normal rhythm. Fig. 7, *b* and *c*.—Continuous parts of Lead 2. The numbers beneath the complexes represent the lengths of the beats in fifths of a second. Fig. 7, *b* shows slight sinus arhythmia. Fig. 7, *c*.—R^2, R^3 and R^4 are automatic ventricular contractions as shown by the varying P-R intervals. Slight sinus arhythmia is also present. Fig. 7, *d* (Lead 3).—The rhythm is again normal.

their waves and deflections in the tracings are regularly superimposed, their origin in or near the auriculoventricular node has sometimes been assumed; these are called nodal extrasystoles.[1] Such simultaneous

1. Lewis: Quart. Jour. Med., 1912-1913, vi, 221. Laslett: Ibid., vi, 210. Cowan, Fleming and Kennedy: Lancet, London, 1912, i, 207.

action is seen in parts of the tracings (Fig. 1, *a* and *b;* Fig. 8, *c*), but it apparently depends on *transient* identical auricular and ventricular speeds, for as the latter vary, varying *a-c* and *c-a,* or P-R intervals soon occur. Besides, nodal beats are usually either regularly interpolated in the normal rhythm, or when premature, are followed after longer or shorter compensatory pauses by the normal dominant beat,

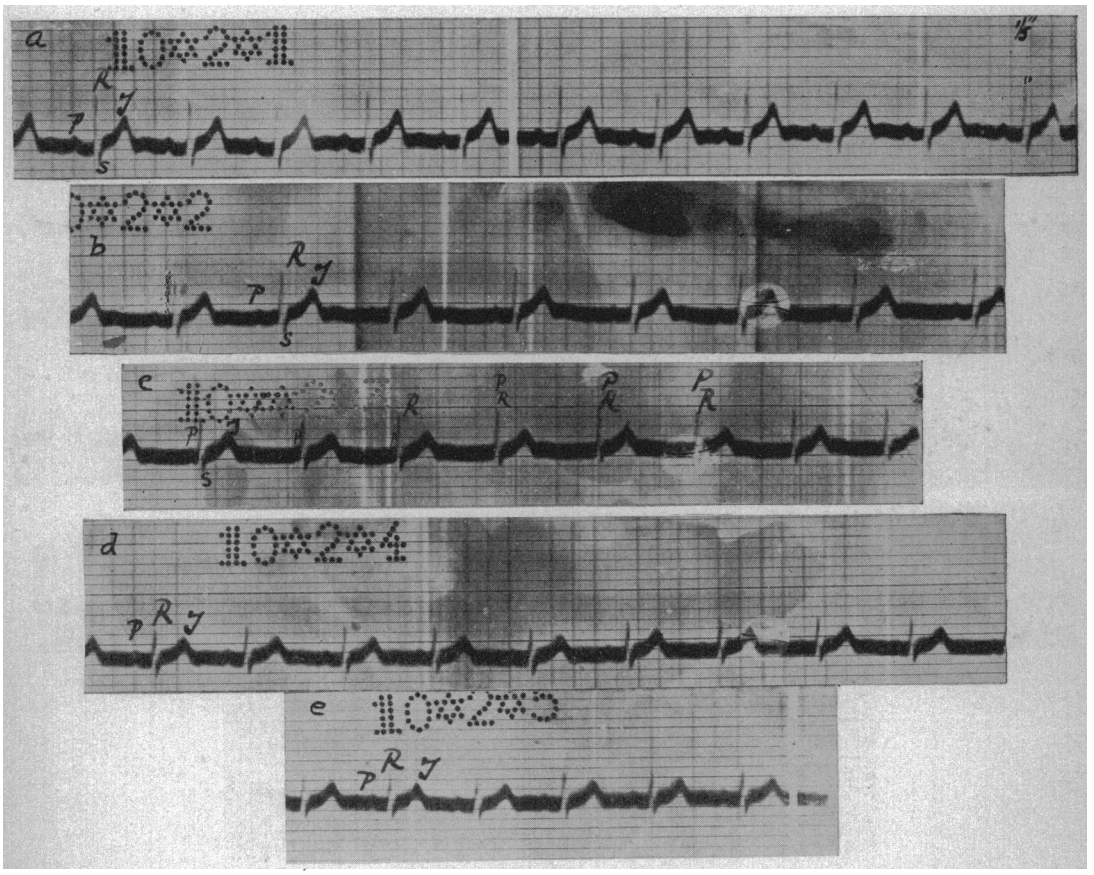

Fig. 8.—Taken February 25. Lead 2; sections taken after the subcutaneous injection of atropin sulphate, 1/30 grain. The time marker measures one-fifth second. Figure 8, *a* and *b*.—Ten and fifteen minutes, respectively, after atropin injection. Slight sinus arhythmia is present; the ventricular rate is about 60 per minute; the auriculoventricular sequence is normal. Fig. 8, *c*.—Twenty minutes after the injection. It shows ventricular automatism, with decreasing P-R intervals and finally simultaneous action of auricle and ventricle (superposition of P and R). Fig. 8, *d* and *e*.—Twenty-five and thirty-five minutes, respectively, after atropin. The beats are again sequential.

assumptions apparently not warranted by the electrocardiograms, all of whose complexes are alike. Retrograde conduction from ventricle to auricle, a rare reversal of the cardiac mechanism,[2] requires consider-

ation as a possible explanation. In the clinical case[3] described, the rhythm when established showed a definite ventriculo-auricular conduction time similar to the normal, and was accompanied by marked ventricular slowing. This conception if applied to my case would necessarily also assume that the reversed mechanism suddenly and irregularly ceased from time to time in such sections of the tracings which do not show a retrograde conduction time (*c-a* or R-P intervals) of less than one-fifth second, and that frequently occasional isolated beats were retrograde — assumptions which seem highly improbable and are not warranted by the tracings. Rihl[4] describes a case of occasional automatic ventricular action produced by vagal pressure. Lewis[5] reports a case of rheumatic mitral stenosis with decompensation; digitalis had been given with consequent ventricular automaticity ("ventricular escape," Lewis). Gallavardin, Dufourt and Petzetakis[6] describe three cases with slow pulses (in one case the rate was 36 per minute) in which there was no clinical evidence of organic cardiovascular disease. Numerous polygraphic and electrocardiographic tracings show the spontaneous occurrence of ventricular automatism in two cases; in all three it was readily evoked by ocular and vagus pressure and by atropin injection. They suggest two main causes for the phenomena: relative retardation of the auricular as compared with the idioventricular rate, or acceleration of the latter beyond the former. Two of their cases had very slow auricular rates occurring either spontaneously or induced by the methods described; the third showed no auricular retardation on vagus or ocular pressure, or after atropin injection. The arhythmia in the first two cases was apparently due to relatively increased idioventricular rapidity beyond that of the sinus. Except for a very slight change in the complexes of the automatic ventricular beats in two of the cases — the absence of a very small S wave — all of the complexes are identical. In digitalis poisoning, Cohn and Fraser[7] have occasionally found either identical auricular and ventricular speeds or ventricles beating more rapidly than auricles with ventricular escape. In my case there is at no time any marked pulse retardation — the lowest rate is 55 — nor is there any evidence of definite auricular slowing, though there is at the periods of ventricular automatism some difference, always slight, between auricular and ventricular rapidity. Except for occasional somewhat slower beats,

2. Cohn, Kessel and Mason: Heart, 1911-1912, iii, 321.
3. Williams and James: Heart, 1913-1914, v, 109.
4. Rihl: Deutsch. Arch. f. klin. Med., 1904, xciv, 286.
5. Lewis: Quart. Jour. Med., 1908-1909, ii, 356.
6. Gallavardin, Dufourt and Petzetakis: Arch. d. mal. du cœur, 1914, i, 1.
7. Cohn and Fraser: Internat. Med. Cong., 1913, Section 6, Part 2, p. 258.

the idioventricular and normal ventricular rates are approximately the same. Slight sinus arhythmia is sometimes present, but is not more marked than is frequently found as a physiological phenomenon.

As possible causes for the production of automatic ventricular action, neurogenic, toxic and organic factors require consideration. A neurogenic factor in the sense of a so-called neurosis due to extracardial conditions (for example, gastric disorders) causing abnormal peripheral disturbances in the centripetal arm of a reflex arc can be here dismissed because of the type of the disease, its course and the definite completion of the arhythmia with the end of the rheumatic attack. Though the action of toxins is an extremely complicated one and to a great extent at present unknown, it seems to depend on their complicated chemical composition and on intricate chemical reactions taking place in the body. It has been pointed out that digitalis poisoning may produce ventricular escape. By analogy, it seems theoretically possible that a rheumatic toxin may also produce a similar arhythmia, though there is no clinical proof for the assumption. Regarding an organic cause for the arhythmia, it is recalled that the patient developed a loud systolic murmur at the apex, one week after the appearance of the arhythmia; the murmur remained for two days, then gradually disappeared. It also disappeared when auricle and ventricle contracted simultaneously, an apparent corroboration that it was due to mitral insufficiency, organic or relative in nature. It is not my intention to discuss cardiac murmurs at any length, the etiology of many of which is not definitely known. Systolic apical murmurs which occur during the course of any febrile disease and then disappear without evidence of an organic cardiac lesion are by no means infrequent. On the other hand, organic murmurs usually increase in intensity and do not disappear. The occurrence of the murmur in conjunction with acute articular rheumatism makes its presence suspicious of some slight, possibly transient, valvular or myocardial involvement. Rheumatic infections cause myocardial inflammation in the form of submiliary myocardial nodules (Aschoff bodies). Healed or healing isolated Aschoff bodies have been found on the interventricular septum in hearts which were the subjects of rheumatic reinfection;[8] during their inflammatory state, if situated close to or even partly involving the bundle of His, before its division, they may conceivably cause sufficient local irritation to produce occasional ventricular automatism with beats of supraventricular origin, and yet the bundle need not be sufficiently compromised to prevent the idioventricular impulse from following its normal course in the conduction system — a fact which probably

8. Thalhimer and Rothschild: Jour. Exper. Med., 1914, xix, 417.

accounts for identical electrocardiographic complexes of all beats, rhythmic and arhythmic.

Right and left vagus pressure had no effect on auriculoventricular sequence. One of the atropin experiments was followed by a number of independent ventricular contractions, with no marked difference between ventricular and auricular rates. This observation does not necessarily exclude the possibility of an organic cause for ventricular automatism because an irritative lesion which does not entirely and permanently compromise the bundle may upset the normal nerve control and mechanism and make it susceptible to atropin poisoning. It would thus seem that the automatic ventricular mechanism was not sufficiently sensitive to respond to vagus pressure, but that atropin poisoning prevented the inhibitory vagus control and permitted ventricular escape.

SUMMARY AND CONCLUSIONS

A case of independent ventricular activity is described. The lowest ventricular rate is 56 per minute, the usual rate is 60 and remains so whether ventricular automatism is present or not. The electrocardiographic complexes of all beats are identical. At one time atropin injection is followed by ventricular escape. The occurrence of the automatic activity during the course of acute articular rheumatism and its disappearance later and a study of the physical signs make it possible that a small transient myocardial inflammatory focus at or near the auriculoventricular connections is the irritative cause of the abnormal mechanism.

Transient independent ventricular activity may occur with no change in the path followed by the idioventricular impulse, with no difference of rate between normal and abnormal beats, and with no marked retardation of the auricular rate.

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