PAROXYSMAL TACHYCARDIA.*

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SINCE 1867, when paroxysmal tachycardia was first described by Cotton,[6] the chief clinical manifestations of this disorder have been well recognised, and a quarter of a century ago all that was known regarding the subject was fully discussed by A. Hoffmann.[14] With the advent of the electrocardiograph further information regarding paroxysmal tachycardia was revealed, and for more than a decade it has been clearly differentiated from auricular flutter and paroxysmal auricular fibrillation. More recently the varieties of paroxysmal tachycardia have been differentiated one from another, and fresh attempts made to analyse the nature of the disorder.

The varieties of paroxysmal tachycardia are classified according to the site wherein the stimulus for the paroxysmal beats is considered to originate, namely the auricles, the auriculo-ventricular node and the ventricles. We can therefore differentiate ventricular from supraventricular paroxysmal tachycardia, the latter term including auricular paroxysmal tachycardia and nodal paroxysmal tachycardia.

Of 138 cases, 26 (18·8 per cent.) were auricular, 40 (28·9 per cent.) nodal, 26 (18·8 per cent.) of undetermined supraventricular origin, and 46 (33·4 per cent.) ventricular. Of the 14 cases which form the basis of this communication, 1 was auricular, 1 nodal, 8 ventricular, and 4 of undetermined origin.

Etiology.—The causes, both predisposing and immediate, of paroxysmal tachycardia are still obscure. Fully one-half of the patients, when examined, are between the ages of 40 and 59; few are younger than 30, but paroxysms may begin early in the second decade.[1, 12, 13, 26] The ratio of females to males is approximately 2 : 3. In many cases there is evidence of either antecedent or still active rheumatic infection of the heart. Six of the present series of 14 cases presented evidence of rheumatic infection. Nevertheless paroxysmal tachycardia

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is one of the rarest manifestations of rheumatic carditis. Subacute infective, and likewise malignant, endocarditis are exceptional. In only 2 of my 14 cases was there evidence of syphilis. The age incidence of paroxysmal tachycardia indicates that the disorder is not intimately related to arterio-sclerosis, and in none of my cases was there evidence of any causal metabolic disorder. Toxæmia, whether endogenous or exogenous, can seldom be invoked as a factor, but one of my patients who had experienced twelve attacks between the ages of 46 and 51 had only recently reduced his weekly allowance of tobacco from seven ounces to four. In a few cases digitalis has been held to be responsible,[30] but in Reid's cases the quantity of digitalis administered was in excess of that indicated by the Eggleston method of calculation.[28] Acute gastro-intestinal disorders and physical or emotional strain are likewise exceptional. Of the nervous origin of the disorder there is little evidence. It is not due to loss, or diminution, of vagal control. Vagal stimulation never lessens the paroxysmal rate. It is true that some cases of paroxysmal tachycardia are promptly arrested by compression of either the right or left vagus in the neck. All such cases are probably examples of auricular paroxysmal tachycardia; in the nodal and ventricular forms of the disorder vagal stimulation never arrests a paroxysm. Well authenticated instances of paroxysms being induced by emotion, atropine or adrenaline are most exceptional. In one case,[12] attacks were provoked by emotion, and a paroxysm of undefined nature at a rate of 215 per minute was excited by injection of 1·0 mgrm. atropine; in another,[31] paroxysms were induced by slight exertion, by atropine and by adrenaline; but in a third,[29] a paroxysmal rate of 180-200 was uninfluenced by atropine. The disorder may originate and recur repeatedly in young individuals in whom, between the paroxysms, no cardiac affection can be recognised; in persons of middle age who, though known to be affected with organic heart disease, have good cardiac reserve power; and in others who are suffering from grave heart failure and who may even be at the point of death.

During the intervals between paroxysms single extrasystoles may occur not infrequently. They are usually of constant form, whether auricular, nodal or ventricular, in any one individual, and as a paroxysm is virtually a rapid series of extrasystoles, usually of the same form as that of the single extrasystoles, no

rigid distinction can be drawn between the briefest paroxysm constituted by two extrasystoles (Fig. 1) and longer paroxysms lasting for seconds, hours or days. Hence, a clue to the nature and origin of paroxysms may perhaps be derived from a study of single extrasystoles, always bearing in mind, however, that single extrasystoles form the basis of one of the most frequent forms of cardiac arrhythmia, whereas paroxysmal tachycardia is one of the rarest. An extrasystole is usually considered to be the heart's response to a stimulus of ectopic origin, and the latter may be the outcome of a functional, *e.g.* nervous, disorder or may be due to structural changes in the myocardium. It has been suggested, however, that some extrasystoles may be due

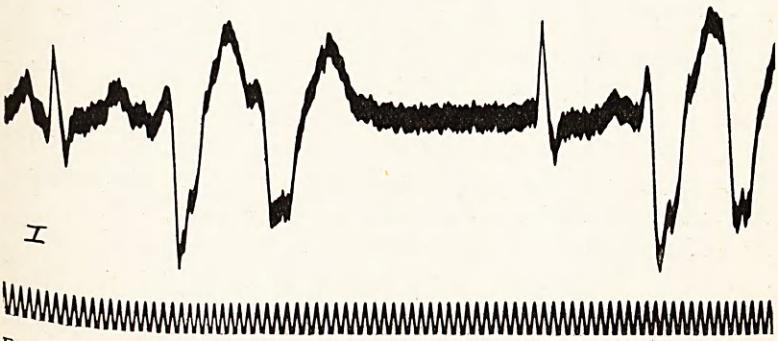

FIG. 1.—Two pairs of ventricular extrasystoles in Case IX; three weeks later ventricular paroxysmal tachycardia, terminating by death on the ninth day. In all the electrocardiograms the time marker record is 28·57 per second.

to circus movement, and the possibility of a series of extrasystoles, namely a paroxysm of tachycardia, being a manifestation of circus movement has been discussed by Lewis[22] who, after weighing the evidence for and against this hypothesis, concludes that we are not yet in a position to form a final judgment. Whatever be the fundamental nature of the disorder, it is apparently one that can undergo frequent and rapid fluctuation. Were it not so, short paroxysms, each constituted by two to ten or more beats, would not occur at brief intervals.

The **morbid anatomy** is not yet well defined, for there have been autopsies in only a few cases. The first observations of real value were those which drew attention to acute inflammatory lesions of the auriculo-ventricular node in cases of nodal rhythm.[7] Extensive disease of the interventricular septum[4]

and dystrophic fibrosis of the area supplied by the circumflex branch of the left coronary artery, the vessel being thrombosed and having a gummatous lesion in its wall,[29] have been described in two cases of ventricular paroxysmal tachycardia. Similarly, ventricular paroxysmal tachycardia has been provoked in the dog by ligation of the right coronary artery.[21] But in other clinical cases the coronary arteries were not diseased or occluded.[30] Lesions in the neighbourhood of the sinus node have been found in two cases of supraventricular paroxysmal tachycardia.[4] Neither in site nor in character, however, are the lesions distinctive, and though it is generally believed that ventricular paroxysmal tachycardia is usually associated with grave disease of the heart,[37] further anatomical and histological observations are much to be desired. Malignant endocarditis of the aortic and mitral valves was found in Case XI, a man aged 41, who died five days after a paroxysm, during which the rate was 133 per minute (Fig. 8).

In Case VIII all the cavities of the heart were dilated; the only valvular abnormality was patchy chronic thickening of the anterior mitral cusp and of the aortic cusps. On the left side of the interventricular septum in the region of the left branch of the bundle, there was a patch of subendocardial fibrous thickening about 1 cm. in diameter. The only naked-eye lesion of the coronary arteries was a calcareous patch of atheroma in the descending branch of the left coronary artery. Microscopic examination of the walls of the ventricles revealed no pathological changes. Case VII presented chronic sclerosis of the mitral and aortic valves with stenosis of the respective orifices, but neither the heart muscle nor the coronary arteries showed any naked-eye changes.

Symptomatology.—The chief symptoms and clinical signs are common to all three forms of the disorder. In each, the paroxysmal acceleration starts abruptly (Figs. 2, 5), the rate of the ventricular contractions and of the arterial pulse is greatly accelerated; during a paroxysm other symptoms and signs may develop. After lasting for a variable period of time, a paroxysm ends as abruptly as it began; then after a brief post-paroxysmal pause, the heart regains its normal rhythm (Figs. 2, 5), or the paroxysm may be the immediate precursor of ventricular fibrillation and the death of the patient (Fig. 14).

The initial symptom is usually severe palpitation. It begins

suddenly, is usually referred to the præcordia, and lasts throughout the paroxysm. In one instance the sensation within the chest was compared to that "when the engines reverse." In exceptional instances the chief initial complaint is that of throbbing in the head. If a paroxysm lasts for more than a few seconds the patient suddenly feels dizzy, faint, breathless, and exhausted; often he has a sense of death impending, and consciousness may be lost, as in one of my patients, Case XIV, aged 58, who for fifteen years had suffered from occasional attacks of tachycardia, each lasting for fifteen minutes to an hour and a half. Cerebral manifestations were noted in 15 of 104 cases.[2] All these symptoms are indications of the cerebral anæmia which ensues as an immediate result of the very rapid and abnormal cardiac action in a paroxysm. A sensation of oppression of the chest, or of "tightness" in the throat, and

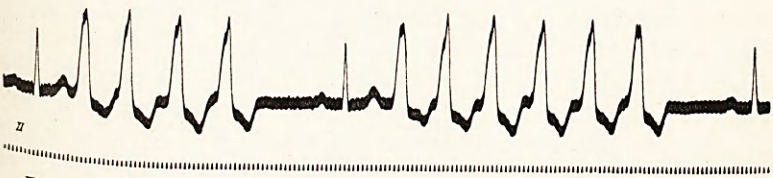

FIG. 2.—Ventricular paroxysmal tachycardia. Two brief paroxysms at a rate of 142·8 per minute. Case IV. ($\times \frac{5}{11}$.)

anginal pain referred to the præcordia, arms or neck are unusual symptoms, but were noted in three of my cases. A brief attack may be unaccompanied by any symptoms. In a man aged 38 (Case XII) the ventricular rate rose from 96 to 156 after an exercise tolerance test; when he lay down the rate was found to be 214, and when he again stood up the rate suddenly fell from 228 to 108 per minute. During the paroxysm he had no palpitation, dyspnœa or distress.

The intensity of symptoms is usually proportionate to the degree of tachycardia and the duration of the paroxysm, but is apt to be more urgent in ventricular than in nodal or auricular forms of paroxysmal tachycardia. Thus auricular paroxysmal tachycardia at a rate of 160 may be associated merely with palpitation and dizziness (see Case I), whereas ventricular paroxysmal tachycardia at the same rate may be the immediate cause of the patient's death (see Case VII). This difference is probably due (1) to the ventricular contrac-

tions in auricular and nodal paroxysmal tachycardias being of supraventricular origin, and therefore of normal character, whereas those of ventricular paroxysmal tachycardia are fundamentally abnormal in respect of the site at which the stimulus originates and the sequence in which the various parts of the ventricular musculature are activated; and (2) to the greater severity of the pre-existing cardiac disease in the ventricular form of paroxysmal tachycardia.

Clinical Signs.—At first the *face* is pale, and has an anxious look. The pallor is soon succeeded by cyanosis, which begins in the ears, lips, and nose, and beads of sweat may break out on the forehead. The combination of pallor and cyanosis, which is most evident in the ventricular form of paroxysmal tachycardia, may give place to a general dusky hue. Meanwhile the *breathing* has become embarrassed, and if the patient had previously been recumbent he sits up gasping for breath. If the arterial pulse can be felt it is found to be fast, at a rate usually of 160-200, and regular; but it may be so rapid and feeble as to be imperceptible. Twelve hours after the start of a paroxysm in Case IX the radial pulse at a rate of 226 was just perceptible, but twenty-four hours later, when the ventricular rate was 230, the patient was "pulseless." If the pulse waves can be felt they may seem to be equal in volume, but graphic records usually reveal an alternating pulse. In rare instances the volume of successive pulse waves is inconstant because the rhythm of the ventricular beats is not absolutely regular (Fig. 5). When the ventricular rate is high, a trivial degree of ventricular arrhythmia is associated with obvious irregularity in the volume of the pulse waves. Towards the end of a long continued paroxysm of ventricular origin, which is proving fatal, the ventricular rate may gradually lessen. In Case VII it fell from 160 to 106, and death ensued on the fourth day; in Case IX it fell from 230 to 140 before death supervened on the ninth day of the paroxysm. Gradual slowing of ventricular rate from 172 to 124 during a period of five days without change in the cardiac mechanism has been recorded,[23] and in another case a paroxysmal rate of 190 "slowed to 120 beats per minute a few hours before death."[29]

The *arterial blood-pressure* falls. In Case I it fell from 148/62 to 110/64; in a case of auricular paroxysmal tachycardia, with a ventricular rate of 235, it fell from 130/90 to 84/76;[37]

in one of ventricular origin, with a paroxysmal rate of 160, the systolic blood-pressure fell from 110 to 85;[31] in two cases of undefined nature it fell from 140-150 to 90, and from 150 to 110.[14] In one of Vaughan's cases[34] the blood-pressure was 98/80; in Case IX the blood-pressure fell to 74/66 on the sixth day of a paroxysm. Figures lower than this may be expected in the gravest cases of all, but in them a precise determination of the arterial pressure becomes impossible. The fall of arterial pressure may be ascribed to several factors: loss of contractile power by the ventricular muscle consequent on shortening of each diastolic phase; synchronous contraction of auricles and ventricles, a phenomenon to which Wenckebach drew particular attention;[35] and in ventricular paroxysmal tachycardia the abnormal character of each ventricular contraction entailing a lessened output per beat.

The *jugular pulse* in the neck, and more especially that on the right side, is usually of large size during a paroxysm. Both

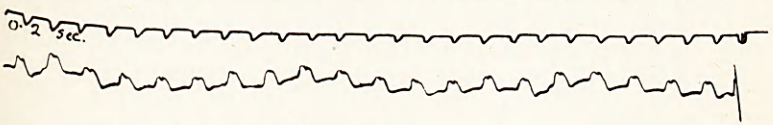

FIG. 3.—Ventricular paroxysmal tachycardia. Apical impulse; rate 237 per minute. Case IX.

this phenomenon, described nearly fifty years ago by Bensen[3] and the ventricular form of the jugular pulse as recorded graphically, are due to simultaneous contraction of auricles and ventricles, and may be seen in all three varieties of paroxysmal tachycardia. The moment a paroxysm ends, the jugular pulse lessens in size or may even cease to be visible, and the auricular wave of the pulse reappears.

The *cardiac impulse* is forcible and diffuse, an invariable phenomenon when the auricles contract simultaneously with the ventricles. The apparent strength of the contractions is in sharp contrast with the feebleness of the arterial pulse. Wenckebach[35] held that the heart does not dilate during a paroxysm, but dilatation of 1 cm. was recorded by Vaughan[34] and of 2·5 cm. by Wilson.[37] The *cardiac sounds* become shorter, sharper and feeble; the two sounds approximate to one another in character, and when the diastolic pause is curtailed to such a degree that the interval after each sound is approximately identical, the cardiac sounds acquire the tic-tac character of

those of the fœtal heart. Any endocardial murmurs that had previously been heard become obscured or inaudible.

A precise determination of the cardiac rate can seldom be made without instrumental aid. If the patient is pulseless, a graphic record may yet be obtainable from the apex-beat of the heart (Fig. 3); and in exceptional instances an electrocardiograph is available. Failing these, auscultation is the best means of determining the cardiac rate. The paroxysmal rate varies in individual cases, but is usually more or less constant in all the paroxysms of any one patient. It is often 160, 170, 180 or 200, but may be as high as 250 or 260. The latter figure is seldom exceeded, but rates as high as 265 and 267 in paroxysms of ventricular origin,[17, 1] and as low as 129, 128, and 100 in paroxysms of auricular origin have been recorded.[11, 15, 26] From the rate alone we cannot define the variety of paroxysmal tachycardia with which we may be dealing. The less frequent rates, for example 140, 150, or 160, may be observed in each of the varieties of tachycardia; and a rate of 200 or more, though commonly occurring in the ventricular variety, has been observed in auricular paroxysmal tachycardia.[1, 4, 37]

During a long paroxysm, œdema develops in the dependent parts, and signs of venous congestion are usually discovered in relation to the lungs, liver and kidneys. Vomiting and tympanites were observed in Case VI of the present series, and in one of Butterfield and Hunt's cases.[4] Intractable hiccough, nausea, persistent vomiting and anuria were noted in Case IX. Transient hemiplegia and dry gangrene have also been recorded.[37]

Electrocardiograms.—In the diagnosis of paroxysmal tachycardia instrumental methods of examination are unnecessary, but an electrocardiogram is almost essential for defining the particular variety of paroxysmal tachycardia with which we are dealing. In auricular and nodal paroxysmal tachycardias the form of each ventricular complex, being usually identical with that yielded by the ventricular beats before and after the paroxysms, indicates that the ventricles are contracting in response to stimuli of supraventricular origin (Figs. 4 and 5). The rhythm is extremely regular in auricular paroxysmal tachycardia; in nodal paroxysmal tachycardia it may be regular or irregular (Fig. 5). In some supraventricular cases definite auricular deflexions are observed. If they are upright, and the

P-R interval is longer than the normal 0·15 to 0·18 second, the condition is one of auricular paroxysmal tachycardia. If the auricular deflexions, *P*, are inverted, and the *P-R* interval is

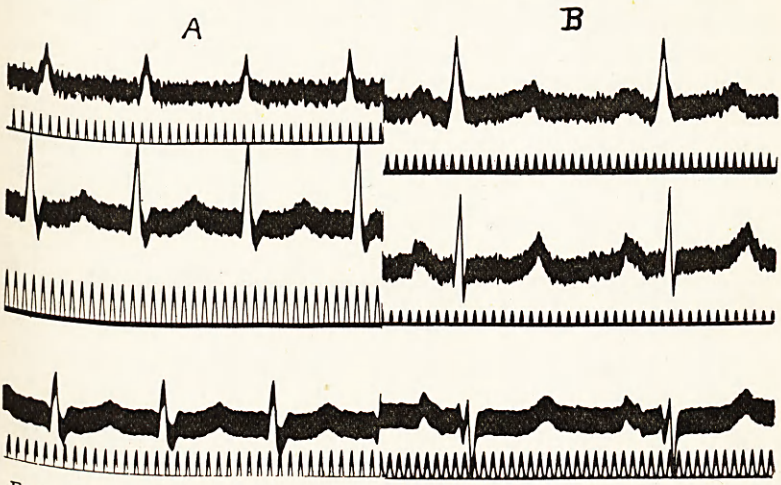

FIG. 4.—Auricular paroxysmal tachycardia. A, Electrocardiograms by leads I, II, and III during a paroxysm at a rate of 160 per minute; B, between paroxysms. Case I.

shorter than normal, the diagnosis is that of nodal paroxysmal tachycardia (Fig. 5). In other cases, when the auricular deflexions are ill-defined and obscure in all three leads from the limbs and in direct chest leads, we can only say that the tachycardia is of

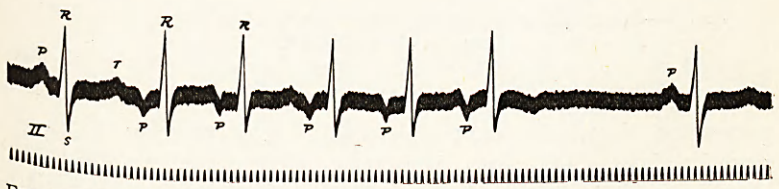

FIG. 5.—Nodal paroxysmal tachycardia. The first beat is a normal one; the next five beats form a brief paroxysm of nodal origin. At the end of the record the post-paroxysmal pause is followed by a normal beat. Case II. (× ⅔.)

supraventricular origin; on the electrocardiographic evidence alone we cannot be certain whether it is auricular or nodal. Anomalous ventricular complexes of supraventricular origin, indicating defective conduction in one branch of the bundle and simulating complexes of ventricular origin, have been recorded by

Lewis [22] and other writers. In such cases the clue to the solution of the problem as to whether the complexes are of supraventricular or of ventricular origin is to be sought in the presence or absence of auricular deflexions notching the ventricular complexes, or in the form of the electrocardiogram either at the start, or end, of the paroxysm.

In ventricular paroxysmal tachycardia each ventricular complex has the form of a ventricular extrasystole, originating usually in the right (Fig. 2) or the left ventricle (Figs. 6A and 7). In some cases, however, the electrocardiographic record

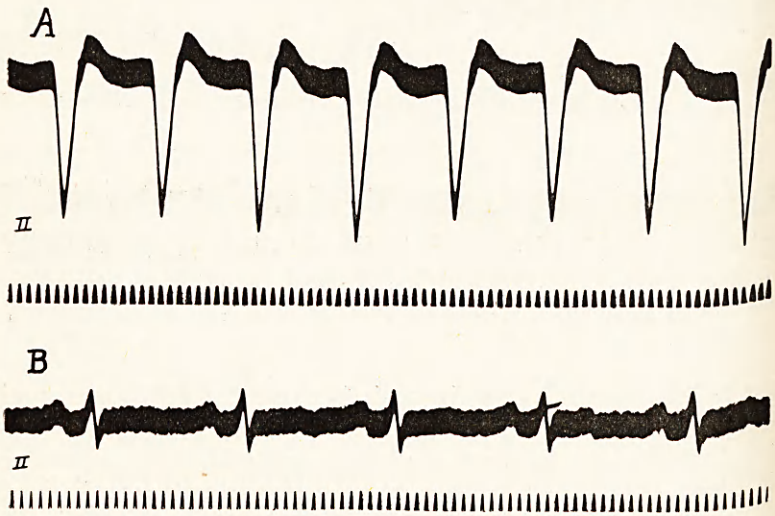

FIG. 6.—A, ventricular paroxysmal tachycardia, at a rate of 174 per minute; B, normal rhythm one week later. Case VI.

of the ventricular paroxysm differs from this. Case XI had presented regularly recurring single extrasystoles (Fig. 8), and in Case VIII there had been single and paired extrasystoles yielding deflexions of large amplitude, characteristic of left apical extrasystoles. In neither of these cases had the extrasystoles any electrocardiographic resemblance to the deflexions subsequently recorded during the paroxysms (Figs. 8 and 14D). In such cases the single extrasystoles and the subsequent paroxysm do not appear to have originated at the same site in the ventricular wall. The ventricular rhythm is usually most regular. Exceptionally it is irregular, as in two cases recorded by Strong and Levine,[33] in another

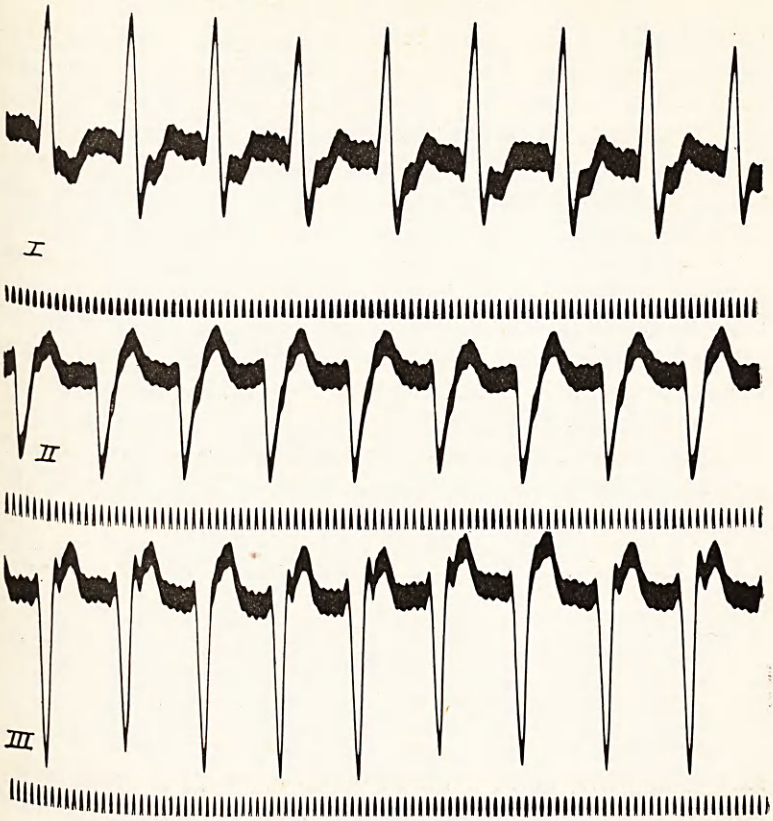

FIG. 7.—Ventricular paroxysmal tachycardia. Leads I, II, and III. Ventricular rate 160·1 per minute. Case VII.

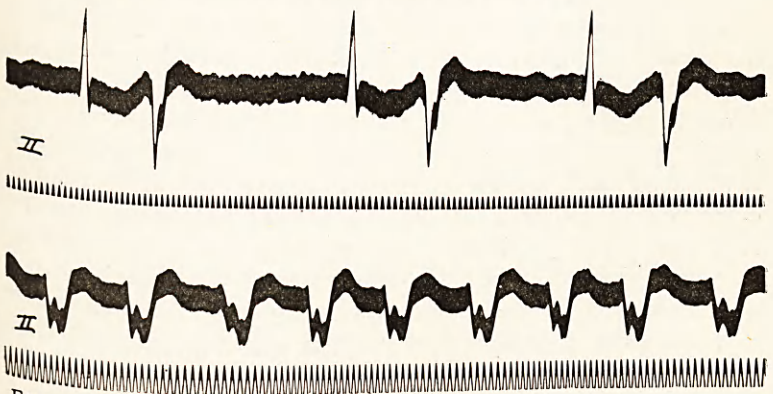

FIG. 8.—The upper record shows a regularly recurring ventricular extrasystole; the lower record, a paroxysm of ventricular origin at the rate of 133 per minute. Case XI (see p. 202).

by Barker,[1] and in one case shown to me by Dr R. A. Fleming. In rare instances the sequence of rhythmic and uniform ventricular complexes is interrupted now and again by that of a ventricular beat originating in the other ventricle,[9] or ventricular complexes of different form alternate with each other, suggesting that the impulse "travels alternately and regularly through each ventricle."[11] Meanwhile the auricular beats may be of normal rate and of sinus origin; less often they are of retrograde origin, and each ventricular impulse may then evoke an auricular response or there may be retrograde heart-block; still more rarely the auricles are in fibrillation as in Cases III and VII, and in three cases recorded by Wolferth and McMillan.[39]

Duration of Paroxysm.—A paroxysm may be a momentary event of a few beats, as in Cases II (Fig. 5), III (Fig. 12), and IV (Fig. 2). In other instances ventricular extrasystoles occur

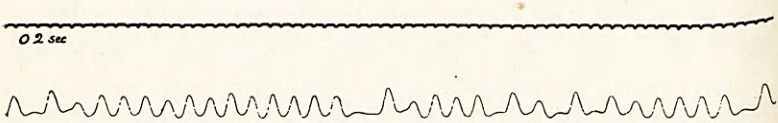

FIG. 9.—Brief paroxysms of ventricular tachycardia. Brachial pulse tracing. Case V.

singly, in pairs, or in frequent series of three or more beats. A portion of a record lasting little more than one minute presented thirteen single and four paired extrasystoles, two series of 3 beats, one of 4, three of 5, one of 7, two of 9 and one of 13 beats (Fig. 9). In other patients the rhythm of the heart is uniformly regular except when interrupted by a paroxysm which may end in a few minutes, or last for several days, as in Case I. In the present series of cases the longest paroxysm was one of eight days (Case IX), ending in the patient's death. Four paroxysms of auricular origin occurring at intervals of from one year to eighteen months in a man aged 38, lasted for five, eight and a half, ten and five days respectively.[37] In a man aged 58 a paroxysm of ventricular origin lasted for eleven days; about a week later it recurred and ended fatally eight days thereafter.[29] In a lad aged 16 a paroxysm at a rate of 192 lasted for eight weeks.[26] * The longest paroxysm on record is one of supra-

* Dr Parkinson informs me that the paroxysm was of ventricular origin.

ventricular origin and of fifteen months' duration in a man aged 22.[39] With the lapse of time paroxysms tend to last longer.

The Frequency of Paroxysms is most inconstant. Brief paroxysms, each of a few seconds or some minutes' duration, may recur frequently for a few days (Case V): in other patients intervals of days, months or years may elapse between paroxysms (Cases I, VIII, IX, XIII and XIV).

Arrest of Paroxysms.—The end of a paroxysm is always abrupt (Figs. 2, 5, and 10); then ensues a brief post-paroxysmal pause comparable to a post-extrasystolic pause, and thereafter the normal cardiac rhythm reasserts itself. Numerous methods of arresting paroxysms have been tried. No methods are of avail in the nodal and ventricular forms of the disorder. Attempts to arrest auricular paroxysmal tachycardia meet with more success, and such measures as deep breathing, holding the breath, drinking of cold water, straining while the glottis is closed, or firm compression of the abdomen succeed in some cases. All these measures, if successful, probably act as does ocular compression, by reflex vagal stimulation. A lawyer aged 51 (Case XIII) who had suffered from twelve paroxysms in five years, was seized with pain at the heart and down both arms while playing golf after lunch. He finished the round, and drove twenty-five miles to consult his doctor, who found him looking gravely ill and the pulse uncountable. Three minutes after an injection of heroin, gr. 1/12, the pulse-rate suddenly fell to 86. The patient then vomited a quantity of beefsteak, and immediately felt perfectly well.

Direct compression of the right or left vagus in the neck is undoubtedly the most effective means of arresting auricular paroxysmal tachycardia. So far as I am aware, the first successful result of vagal compression as a means of arresting paroxysmal tachycardia was that recorded by Bensen in 1880.[3] Successful results were subsequently recorded, but in none of these instances was the precise nature of the tachycardia recorded, nor was it stated which vagus was compressed.[16, 27, 35] Hoffmann[15] recorded successful left vagal compression in two cases of undefined paroxysmal tachycardia. More recently, successful arrest of undoubted auricular paroxysmal tachycardia by vagal compression has been recorded by several writers.[5, 18, 34, 37] In Case I of my series, an example of auricular paroxysmal tachycardia, compression of either right or

left vagus was repeatedly and promptly successful in arresting a paroxysm (Fig. 10). In no case of nodal or ventricular paroxysmal tachycardia have I found a paroxysm influenced by compression of either right or left vagus (*e.g.* Cases VII, VIII, and IX), and similar unsuccessful results have been recorded by others.[29, 31, 34] When either direct or reflex vagal stimulation succeeds in arresting a paroxysm, we are dealing with one of auricular origin.

Diagnosis.—The sudden onset of palpitation with an arterial pulse-rate of 140 or more always suggests the possibility of paroxysmal tachycardia. If the rhythm of the arterial pulse, and of the ventricles as ascertained by auscultation, is wholly irregular, the condition is almost certainly that of paroxysmal auricular fibrillation, a disorder by no means rare. If the

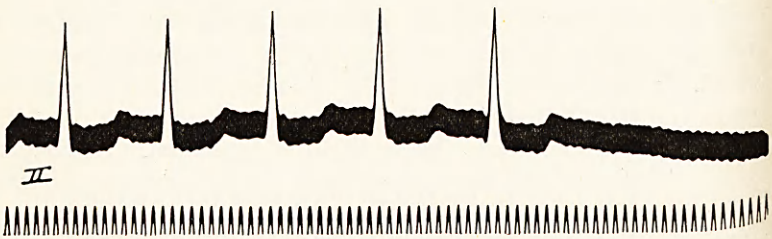

FIG. 10.—Auricular paroxysmal tachycardia. Arrested by compression of the left vagus. Case I.

ventricular rhythm be regular, however, the possibility of auricular flutter has to be borne in mind. In auricular flutter the auricular rate is usually 250-300, and the ventricular rate 140-150, the auriculo-ventricular ratio being 2 : 1. A ventricular rate exceeding 150 is therefore more likely to indicate paroxysmal tachycardia than auricular flutter. Difficulty arises in those exceptional cases when the rate of auricular flutter rises to 350 or more—the fastest auricular flutter rate I have observed was 380, with a ventricular rate of 175—or when the auriculo-ventricular ratio becoming temporarily 1 : 1 the ventricular rate rises to 250, 260 or more per minute. This latter contingency is, however, exceedingly rare. McMillan and Sweeney,[24] reporting a case of this nature, could find records of only ten cases in the literature. In auricular flutter the jugular veins are not so greatly distended, nor do they pulsate so forcibly as in paroxysmal tachycardia. The effects of vagal

compression are in some degree distinctive. If vagal compression causes transient slowing of the ventricles, and if this effect passes off rapidly after the vagal compression is relaxed, we may be certain that we are dealing with a case of auricular flutter. If vagal compression causes the ventricular rate to fall suddenly to normal, *e.g.* from 160-180 to 80, and the latter rate is maintained after withdrawal of pressure from the vagus, the attack was undoubtedly one of auricular paroxysmal tachycardia. In nodal and ventricular forms of paroxysmal tachycardia, the ventricular rate is not influenced by vagal compression. An electrocardiographic examination is, however, the most reliable and sure means of determining the diagnosis. Accuracy of diagnosis is not of mere academic interest, because the prognosis varies according to the variety of paroxysmal tachycardia with which we are dealing. Moreover, if paroxysmal tachycardia be mistaken for auricular flutter, we may be tempted to administer digitalis or strophanthus, because of the undoubted efficacy of these two drugs in the latter disorder. Their administration in cases of paroxysmal tachycardia of auricular origin is attended by no benefit, and in those of ventricular origin is fraught with danger.

The **prognosis** is usually more favourable in young persons than in those who are advanced in years. It depends, as has been shown by Willius and Barnes,[36] on the degree of integrity of the cardiac muscle and valves rather than on the rate or duration of the tachycardia. If there is no organic heart disease, and if the heart's efficiency between paroxysms is good, or if paroxysms recur at long intervals, are of short duration and do not cause serious cardiac embarrassment, the prognosis is relatively favourable and the patient may lead an active life for many years; whereas when paroxysmal tachycardia supervenes in cases of organic disease of the valves or myocardium the prognosis is more grave. Indeed a paroxysm which recurs frequently or lasts for many hours or a few days, and is accompanied by gross cardiac failure, is almost certainly the immediate herald of the patient's death (see Cases VII, VIII, and IX). Paroxysms of auricular origin are not incompatible with many years of active, useful life; those of ventricular origin seldom arise apart from grave organic disease of the heart. In ventricular paroxysmal tachycardia slowing of the ventricular rate without change in the cardiac mechanism is, as in Case IX, usually a terminal event.

Treatment. — There is unfortunately little new to add regarding the measures to be taken for the arrest or prevention of paroxysms. During a paroxysm the patient should be kept absolutely quiet in the recumbent posture and with the head low. Attempts should be made to arrest the paroxysm by compressing the right or left vagus in the neck. In paroxysms of auricular origin this will often succeed promptly (see p. 205). Methods of indirect vagal stimulation, to which reference has already been made, are not likely to succeed if direct vagal compression has failed. Fortunately the vast majority of paroxysms end spontaneously.

Digitalis and *strophanthus* are seldom, if ever, of therapeutic value. They neither arrest nor mitigate the severity of an attack, nor do they prevent its recurrence. In Case IX three drachms of digitalis tincture given within a period of four hours, and followed five hours later by $\frac{1}{100}$ gr. of strophanthin

FIG. 11.—Ventricular fibrillation in a dying heart. Case X.

intravenously, were without effect. In Case VIII $\frac{1}{250}$ gr. of strophanthin intravenously, however, lowered the ventricular rate from 200 to 96, from 193 to 90, and from 194 to 78 in three successive paroxysms. But both these patients died within a few days thereafter.

When a case ends fatally death is sudden and is usually, if not always, due to the onset of ventricular fibrillation. The mode of death is described in the record of Case X (see p. 216). Ventricular fibrillation is known sometimes to supervene in the dying human heart,[19] and may likewise be the terminal event in cases of auricular fibrillation having a ventricular extrasystole recurring regularly after each physiological beat of the ventricles as a result of digitalis or strophanthus poisoning. Further, there is abundant evidence, as pointed out by MacWilliam,[25] that ventricular tachycardia may develop into ventricular fibrillation. Digitalis and strophanthus, whether by direct action on the myocardium or by indirect action through the vagi, often convert an auricular action from flutter into fibrillation and may

likewise induce ventricular fibrillation. The general conclusion may therefore be drawn that in paroxysmal tachycardia the drugs of the digitalis group are not only valueless but may even be dangerous. The risk attending their administration appears to be greatest in paroxysmal tachycardia of ventricular origin.

Quinine is an old remedy in these cases, and the success attending the administration of *quinidine* in some cases of auricular fibrillation and of flutter has led to a fresh enquiry regarding the effects of these two drugs in paroxysmal tachycardia. The results recently recorded are disappointing. Quinine bihydrochloride, 3 gr. intravenously, arrested the paroxysms in six of nine cases;[32] quinidine was successful in two auricular cases,[18] and in one ventricular case the paroxysm invariably terminated within half an hour after the oral administration of 0·4 grm. of this drug.[31] But quinidine had "little or no effect" in another series of six cases.[26] In Cases VIII and IX of the present series, the paroxysm in both cases being of ventricular origin, quinidine was valueless. But a further study of the action of the drug in paroxysmal tachycardia is desirable.

The use of *heroin* was apparently successful in Case XIII (see p. 205), but repeated doses of morphia were of no value in Case IX, in which bromide, pituitrin and ice-bags likewise proved valueless. During a long-continued paroxysm all measures appropriate to the relief of cardiac failure and of intractable vomiting may have to be employed.

Case I.—Auricular paroxysmal tachycardia at a rate of 140 to 180, controlled by vagal compression.

A shale miner, aged 58. Aortic incompetence and an aneurysm of the aortic arch had been recognised five years previously, when the Wassermann reaction was strongly positive. When re-admitted to hospital in June 1925 he had been complaining for six weeks of sudden attacks of palpitation and severe throbbing in the occipital region, recurring every few days. The attacks had lasted from a few minutes to about a day and a half, and each attack had stopped suddenly. Examination revealed aortic incompetence, a large aneurysm of the aortic arch, a normal cardiac rhythm without any extrasytoles (Fig. 4 B), thickened arterial walls and a blood-pressure of 175/48. Thirty paroxysms were recorded in thirteen weeks. During the paroxysms the ventricular rhythm was regular: the rate was usually 160 (Fig. 4 A) but in five paroxysms the rate was 140 or less; the maximal rate was 180. During a paroxysm the veins of the neck became greatly swollen and

pulsated freely, and the arterial blood-pressure readings changed from 148/62 to 110/64. In the intervals between paroxysms the ventricular rate was usually between 80 and 90 per minute, the cervical veins were not turgid and only a slight degree of pulsation could be seen in them. The ninth paroxysm lasted for twenty-seven and a half hours; like previous paroxysms it ceased spontaneously. Three days later a paroxysm, which had lasted for an hour and forty minutes, was promptly arrested by compression of the left vagus. Subsequently there were twenty paroxysms, the shortest lasting for five minutes, the longest for sixteen hours. Two were arrested by compression of the right vagus, three by compression of the left vagus (Fig. 10), fourteen ceased spontaneously, and one ceased ten minutes after subcutaneous injection of pilocarpine nitrate, $\frac{1}{40}$ gr. On only one occasion did vagal compression fail to arrest an attack. Even the longest paroxysm, lasting for twenty-seven and a half hours, was not attended by any signs of cardiac failure. Two months after the patient's discharge from hospital he still had frequent paroxysms, but had been able promptly to stop them by compressing the right vagus.

Case II.—Brief paroxysms of nodal paroxysmal tachycardia.

A clerk, aged 36, had good health until January 1921, when he first began to complain of occasional sudden attacks of dizziness, each of which passed off gradually. Four years later he presented no signs of organic disease of the heart; the Wassermann reaction was negative; frequent nodal extrasystoles occurred singly, in pairs or in groups of 3, 4, 5, or 6, at a rate of 139 per minute (Fig. 5). Occasionally a single nodal extrasystole was followed by a ventricular extrasystole. During the paroxysms the cardiac rhythm was not absolutely regular, and the coincident pulse-waves were markedly unequal. A similar, though slower, nodal rhythm with marked irregularity of the arterial pulse-waves was recorded in a former publication.[8]

Case III.—One brief paroxysm of ventricular origin at a rate of 150 in a case of chronic rheumatic, mitral valvular disease with auricular fibrillation.

A paperworker, aged 37, had suffered from acute rheumatism at the age of 15, and from "rheumatics" at the age of 28. He had complained of shortness of breath, faintness and dizziness for six or seven years before his first admission to hospital on 13th February 1922, when he was found to present mitral incompetence and stenosis, with auricular fibrillation but without dropsy. Quinidine sulphate in doses of 0.4 grm. thrice daily failed to restore the normal rhythm. The Wassermann reaction was negative. Nine months later he was slightly cyanosed. 20.4 grm. of quinidine sulphate, administered in the course of eleven days, again failed to arrest the auricular fibrillation. On the sixth day of

administration of quinidine, a short paroxysm of ventricular tachycardia, represented by six beats at a rate of 150, was recorded (Fig. 12). Except during this paroxysm, the ventricular rate was always infrequent, seldom rising above 90 per minute. During April and May 1923 the ventricular rate varied from 50 to 90, and no further paroxysm was recorded. Thereafter his health slowly deteriorated, and eventually he became bedridden. In April 1924 he developed a left hemiplegia and died a week later.

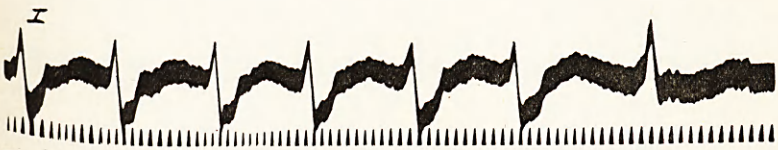

FIG. 12.—Ventricular paroxysmal tachycardia, at a rate of 150 per minute. The last beat of the record is of sinus origin. Case III.

CASE IV.—Brief ventricular paroxysmal tachycardia at a rate of 142.8 per minute.

A married woman, aged 45, who had frequently suffered from "muscular rheumatism," and who had complained of breathlessness and intermittent dropsy of the legs since an abortion ten years ago, and of anginal pain, was admitted to hospital on 28th April 1924. She was stout, orthopnœic, and dropsical at the ankles. The heart was a little enlarged; its sounds were pure. The regular cardiac rhythm was interrupted by frequent right ventricular extrasystoles occurring at first singly, in pairs or in series of three. The blood-pressure was 164/90. The lungs, liver, kidneys and other organs did not present any signs of disease. The Wassermann reaction was negative. The dropsy speedily vanished and the blood-pressure fell to 130/90. A month after her admission a right ventricular extrasystole recurred regularly after each physiological beat, and on the 5th June a brief ventricular paroxysmal tachycardia, consisting of six beats at a rate of 142.8 per minute was recorded (Fig. 2). The administration of digitalis tincture, ten minims thrice daily, and of quinidine sulphate, 0.2 grm. twice or thrice daily, was then begun and continued for three weeks. At the end of that period the extrasystoles had ceased, and three weeks later the patient was discharged.

CASE V.—Brief paroxysms of tachycardia of ventricular origin, in a case of subacute infective endocarditis.

A wireworker, aged 44, had been treated for "kidney disease" for a year previously. Four months before admission he developed an abscess of the jaw, and two months later all teeth were extracted, after which the face and feet again became œdematous. Four days before

admission he developed signs of a large infarct in the lower lobe of the right lung. He was found to have pronounced aortic incompetence, moderate enlargement of the spleen, clubbing of the finger-tips, and slight intermittent hæmaturia. The Wassermann reaction was negative. Blood cultures were sterile. A fortnight after admission he had repeated short paroxysms of ventricular tachycardia, each usually consisting of 4 to 13 beats (Figs. 9 and 13) at a rate of 142 to 153 per minute. Thereafter the cardiac rhythm again became wholly regular.

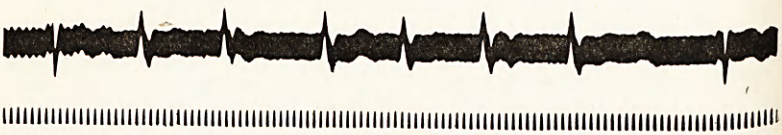

FIG. 13.—Paroxysm of six beats of ventricular origin, at a rate of 142 per minute. Case V. The first and last beats are of sinus origin.

CASE VI.—Acute rheumatism; ventricular paroxysmal tachycardia of several hours' duration, ten weeks before death from progressive heart failure.

A warehouseman, aged 46, who had suffered from five previous attacks of acute rheumatism, the first occurring at the age of 25. He had never fully recovered from a sixth attack of acute rheumatism which began four months previous to his admission on 4th June 1924. He was pale, the heart was enlarged, the pulse was rhythmic and varied in rate from 110 to 128. The urine contained albumin, granular tube-casts and red blood corpuscles. The Wassermann reaction was negative. On the fourth day after admission he wakened at 1.15 A.M., complaining of fluttering of the heart, and was mildly delirious. Six hours later he began to vomit frequently, the abdomen was distended and tympanitic, and the pulse was still uncountable. An electrocardiogram showed rhythmic ventricular paroxysmal tachycardia at a rate of 174 per minute (Fig. 6 A). That night the patient slept well after morphia, $\frac{1}{12}$ gr., and no further paroxysm was recorded. A record of the heart, taken a week after the paroxysm, showing normal rhythm, is reproduced in Fig. 6 B. Three weeks later, he developed pleurisy with effusion, fresh arthritis, hæmatemesis, progressive œdema and asthenia, and died ten weeks after the paroxysm of tachycardia. Permission for an autopsy was refused.

CASE VII.—Ventricular paroxysmal tachycardia at a rate of 160 per minute in a case of chronic rheumatic valvular disease with auricular fibrillation. Death on the fifth day after the paroxysm began.

A domestic servant, aged 47, who had suffered from several attacks of rheumatism, a perforated gastric ulcer at the age of 20, and

"influenza" a month before admission on 22nd December 1925. She was breathless, cyanosed and dropsical, presented signs of mitral and aortic valvular disease and of auricular fibrillation with a ventricular rate of 90 to 118. The urine was scanty and albuminous; the urea concentration was 2.7 per cent., the phenol-sulphonephthalein excretion was 60 per cent. The Wassermann reaction was negative. The administration of Guy's pill was followed by temporary improvement after digitalis, digitaline, diuretine and mestarine had failed.

Early in the afternoon of the 21st January a paroxysm of tachycardia, at a rate of 160 per minute and with a weak though regular pulse and increasing cyanosis, dyspnœa and dropsy, began suddenly and persisted. Digitalis was discontinued on the second day. In electrocardiograms taken on the third day the ventricular rate was 160.1 per minute (Fig. 7). That afternoon she was unconscious. Quinidine sulphate 0.8 grm. was given eight hourly, and at 10 P.M. the ventricular rate had fallen to 128 per minute. On the fourth day she regained consciousness, the ventricular rate fell to 106-116, the rhythm remaining regular. She died suddenly at 1.30 A.M. on the fifth day. The total dosage of quinidine was 3.2 grm. *Post-mortem* examination revealed pronounced stenosis and shrinking of the mitral and aortic valves and chronic interstitial nephritis, but no evidence of recent endocarditis or naked-eye changes in the myocardium.

CASE VIII.—Three attacks of ventricular paroxysmal tachycardia at a rate of 190 to 200 per minute. Sudden death on the fourth day after the initial paroxysm.

A farm labourer, aged 69, was admitted to hospital on the 11th March 1925, complaining of breathlessness and of swelling of the legs for three weeks, and of occasional giddiness lasting for a minute or two. He had orthopnœa, slight Cheyne-Stokes breathing, and some œdema of the ankles. The heart was hypertrophied and dilated, its sounds were pure but feeble; its rhythm was normal, but this was frequently interrupted by left ventricular extrasystoles, and the pulse was alternating. On one occasion paired ventricular extrasystoles of this type were recorded. The blood-pressure varied from 150/110 to 190/110. Radiographically the aortic shadow was prominent and dense. The liver and bases of the lungs presented signs of chronic venous congestion, and the urine contained a trace of albumin. The Wassermann reaction was negative. Digitalis induced a copious diuresis.

The ventricular rate was usually between 40 to 56 per minute, and seldom exceeded 60. The initial ventricular complex by Di was small, upward and notched; by Dii and Diii a downward broad deflexion, lasting for 0.105 second. Although *T* was poorly developed in all three leads, it was directed downwards in Di, and upwards in Dii and

Diii. When the patient was discharged from hospital on 6th July 1925 he was still feeble but no longer dropsical.

A few days before re-admission, on 23rd October 1925, he had lost consciousness for a few minutes. He was orthopnœic, cyanosed and

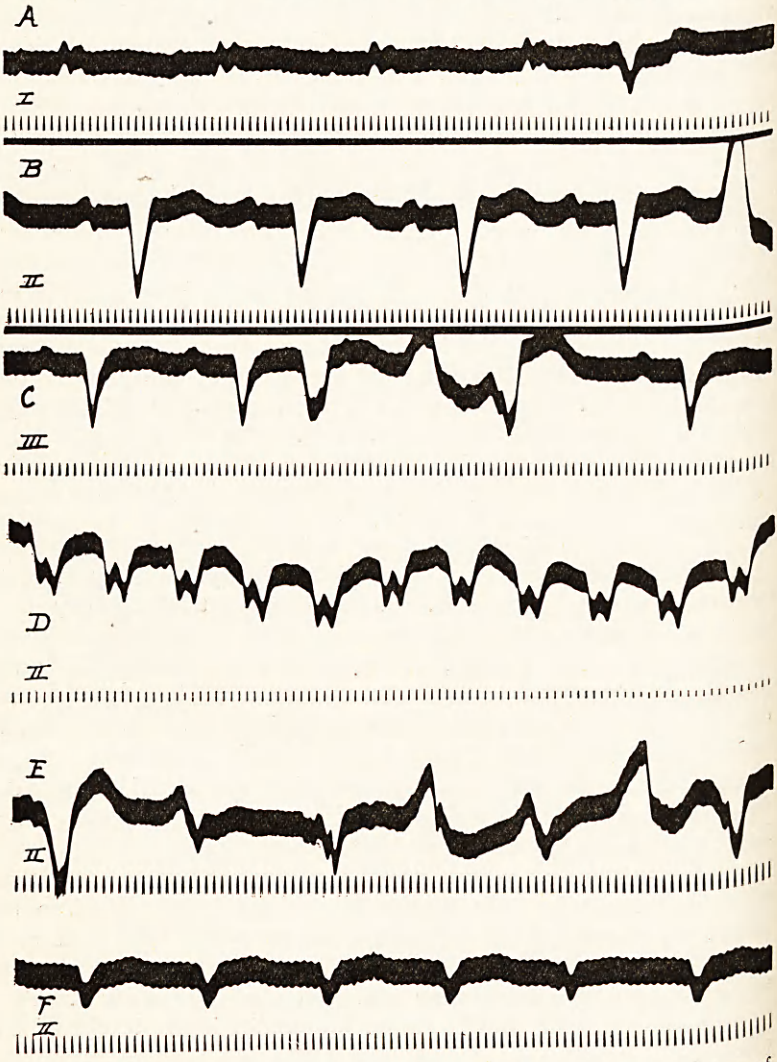

FIG. 14.—Ventricular paroxysmal tachycardia. A, B, and C show portions of records taken by leads I, II, and III respectively on 29th October 1925. The sinus rhythm is interrupted by occasional ventricular extrasystoles. D shows the cardiac mechanism during a paroxysm of ventricular origin, in which the rate was 190·4 per minute. In E, taken seven minutes after D, the ventricles were verging on fibrillation. F was taken twenty-five minutes after E. Case VIII.

markedly dropsical. The dropsy lessened after the administration of theobromine sodium salicylate. Two days after admission he had a "giddy turn," and was breathless for a few minutes, and on the following day the scanty sputum contained bright red blood. The ventricular rate was now 85 per minute; its regular rhythm was, as before, interrupted by occasional ventricular extrasystoles, each followed by an alternating pulse. On 29th October, electrocardiographic records were obtained of ventricular extrasystoles, which were single, in pairs, or in groups of three, originating from different foci (Figs. 14 A, B, and C). At 9 P.M. the following day there suddenly developed a paroxysm in which the ventricular rate was 200 per minute; the rhythm was regular. At 9.45 P.M. the rate was still 200, and the patient was almost pulseless. Both right and left vagal compressions failed to arrest the paroxysm. Strophanthin, $\frac{1}{250}$ gr., was then given intravenously; 25 minutes later, the ventricular rate had fallen to 96 per minute, the pulse was stronger and again irregular. Two days later a second paroxysm started at 7.15 P.M. The ventricular rate rose to 190.4, and in one record attained a rate of 193.2 per minute; the rhythm was regular. Half an hour after the paroxysm began, $\frac{1}{250}$ gr. of strophanthin was given intravenously; in half an hour the pulse-rate fell to 90. A third paroxysm, in which the ventricular rate was 194, began at 10.15 the following morning. At 10.45, $\frac{1}{250}$ gr. of strophanthin was given intravenously. The heart's action at 11.37 is shown in Fig. 14 D; the rhythm was regular; the rate was 190.4 per minute; the upward notch on each downward ventricular deflexion is probably an auricular deflexion. At 11.44 A.M. the ventricles were verging on, if not actually in, fibrillation (Fig. 14 E). At 12.19 P.M. the ventricular contractions were again rhythmic at a rate of 111 per minute, but no auricular deflexions are visible (Fig. 14 F). At 12.33 P.M. the heart's rate had fallen to 78, and the form of the ventricular deflexions was almost identical with that of Fig. 14 B, though the auricular deflexion was less well defined. The paroxysm had ceased. During the afternoon two doses of quinidine sulphate, each of 0.2 grm., were administered; at 7.30 P.M. the patient suddenly became more breathless and died a minute later.

The morbid anatomy of the heart has been referred to on p. 196.

CASE IX.—Recurring attacks of paroxysmal tachycardia for fourteen years. Final paroxysm at a rate of 226 to 240, terminating by death on the ninth day.

A ploughman, aged 29, who had been subject to paroxysms of tachycardia for fourteen years, the longest lasting for four days, was admitted to hospital in December 1923. The heart was much enlarged, a mitral systolic murmur was audible, the regular cardiac rhythm at a rate of about 90 was interrupted by single or paired ventricular extra-

systoles (Fig. 1); there was no cyanosis, dropsy, or other sign of heart failure. The Wassermann reaction was negative. During the second week of January there were four paroxysms, each lasting from eight to twelve hours, in which the ventricular rate was 175 to 185, and the rhythm regular. Quinidine (0.4 grm.), given twice daily for four days, failed to ward off the paroxysms. The final paroxysm wakened him at 11 P.M. on 4th February. The following morning the ventricular rate was 226, the rhythm was regular, both right and left vagal compressions were ineffective. Vomiting became frequent and distressing. On the third day the face was dusky, no pulse could be felt at the wrists, the cardiac impulse was forcible, the ventricular rate was 230, the ventricular rhythm was still regular; the jugular pulse was small, the liver was not enlarged or tender, there was no œdema. That night he had only two hours' sleep despite $\frac{1}{4}$ gr. of morphia. On the fourth day the extremities were cyanosed and cold, he was still vomiting frequently and still pulseless; the ventricular rate was 237 to 240 (Fig. 3). On the fifth day the ventricular rate had fallen to 220 and the vomiting had lessened, but abdominal pain and intractable hiccough had supervened. Next day the patient was less dusky and the extremities were warm; the ventricular rate had fallen to 207; the blood-pressure was 74/66. On the seventh day the ventricular rate had fallen to 160 to 168, and the blood-pressure had risen to 86/80, but the ankles had become œdematous, and the abdomen was distended and tympanitic. On the eighth day the ventricular rate had fallen to 140, the base of the right lung was œdematous, the urine was scanty and albuminous, the hiccough still persisted. The following day the patient died suddenly, 194 hours after the start of the paroxysm.

Case X.—Auricular fibrillation with subsequent regularly recurring ventricular extrasystoles and terminal ventricular fibrillation.

A housewife, aged 55, with a history of acute rheumatism at age 12, had been ailing for six years, and had suffered from symptoms of cardiac failure for three months. When admitted on 5th January 1926, she was cyanosed, orthopnœic and dropsical, with signs of mitral stenosis and incompetence, aortic incompetence, auricular fibrillation and right ventricular extrasystoles. The Wassermann reaction was negative. She was given Guy's pill, 3 grs., once a day, and theobromine sodium salicylate, 10 grs., thrice daily, had a copious diuresis, and by the end of the third week had lost all but a trace of dropsy. On the twenty-sixth day there was an intermittent "coupled rhythm" of the ventricles, and though the digitalis was stopped this coupling was still pronounced on the succeeding day. On the twenty-eighth day, while the patient was lying in her bed, she suddenly sat up in a general tonic convulsion, lasting for a second or two, then fell backwards and became livid. The heart was found

to have ceased beating, but the breathing continued for about half a minute.

Meanwhile the patient was connected with the electrocardiograph, and the oscillations of the fibre were being watched. At first each normal ventricular beat was followed by a ventricular extrasystole. A few seconds later the fibre began to oscillate widely and rapidly in the form of large diphasic deflexions, which apparently represented a brief paroxysm of ventricular tachycardia or the initial phase of ventricular fibrillation. The diphasic deflexions gradually became smaller, and ten or twelve seconds later, when the shadow of the fibre had been steadied on the plate, a record showed irregular deflexions indicative of ventricular fibrillation (Fig. 11). The deflexions gradually decreased in amplitude until the fibre was at rest.

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Discussion.

Dr Lambie said—Naturally, in a condition which is irregular in its onset and which undergoes spontaneous arrest, it is difficult to judge of the effects of remedies. We do know, however, that in cases of auricular paroxysmal tachycardia, vagus stimulation will, in the large majority of cases, arrest an attack. Thus, when searching for drugs which will not merely arrest a paroxysm, but prevent further attacks, one naturally thinks of those which bring about vagus stimulation. Drugs of the digitalis series, for example, do this. Digitalis increases vagus tone by stimulating the vagus centre, but we know from experience that it is ineffective in the majority of cases (although there is difference of opinion upon this point), and that is natural because digitalis not only brings about stimulation of the vagus, but it increases the irritability of the heart muscle and may itself induce extrasystoles or even auricular flutter and fibrillation.

There is another drug, however, which has not received sufficient attention—namely pilocarpine—and, in the small number of cases which I have observed, it has seemed to me that patients have benefited by its administration. It brings about vagus stimulation in a different

way. It stimulates the vagus endings in the heart, and it appears to possess the advantage of having less tendency to increase the irritability of the cardiac muscle. In one case the patient had frequent attacks of auricular paroxysmal tachycardia. The attacks could be cut short by compressing the vagus in the neck. I then tried if they could be arrested by the administration of pilocarpine. One or two trials were successful, by hypodermic injection. It was then necessary to exclude the possibility that this might be due to reflex inhibition from the prick of the needle. I failed to arrest an attack by the injection of sterile water, although, when pilocarpine was injected, the attack stopped after a short time. This patient was then given pilocarpine by the mouth, and, so long as he was under its influence, there were no attacks, though they recurred when the administration of the drug was intermitted. I suggest, therefore, that pilocarpine is worthy of further study in the treatment of auricular peroxysmal tachycardia.

Dr A. Rae Gilchrist (introduced) said—I have had the opportunity, in Professor Murray Lyon's wards, of seeing about half a dozen cases of ventricular tachycardia during the last two years, and one thing that has come out from the study of these cases is that several of the attacks were related to the administration of digitalis. The digitalis was given by the method of massive dosage suggested by Eggleston. The dose of digitalis was not in excess of the usual therapeutic dose, and yet these patients showed ventricular tachycardia on several occasions immediately following the administration of the drug. One man had three doses, separated by sixteen-day intervals, and on the first occasion on which he had the dose the first attack came on two days after the dose was given. On the second occasion, when he had another dose of digitalis, sixteen days after the previous one, he had two attacks, the first on the day the dose was given, the second on the day following. A fortnight later he had a third dose, and the attack started after 1 grm. of the powder had been given. This suggests that digitalis is only the provoking cause of the condition, and that there are other factors present. Two of these patients who developed the condition after digitalis were in an advanced stage of cardiac failure, and this suggests, that with progressing failure, the heart muscle becomes more susceptible to the drug. Now the patients did not receive a poisonous dose of the drug and showed no general toxic symptoms—no vomiting, no nausea, no anorexia—and yet an attack came on with regularity after the drug had been given. I think, therefore, that there are other factors besides digitalis, in the causation of the condition, and one of these is undoubtedly the state of nutrition of the heart muscle. In this connection also, it is interesting to notice that in patients with advanced heart failure, with normal rhythm,

digitalis may induce auricular fibrillation. This question has been studied recently in America by Resnik, and he came to the conclusion that the second factor was anoxæmia of the heart muscle. It appears to me that with digitalis inducing auricular fibrillation in the one case and ventricular tachycardia in the other, anoxæmia may be a provoking cause in these cases where tachycardia has followed on digitalis administration. It also suggests that ventricular tachycardia, in that way, is analogous to circus movement in the auricle. Some of these patients were suffering from auricular fibrillation at the time. It is interesting to think that they may have had two circus movements, one in the auricle and one in the ventricle, at the same time.

Dr Ritchie replied.