AURICULAR FLUTTER.

John I. Shepherd,

M.B., Ch.B., Edin.

References.

1. MacWilliam. Jl. Physiol. 1887, VIII, 296.
2. Jolly and Ritchie, Heart, 1910-11, II, 177.
3. (Gibson, Ed. Med. Jl. 1905, XVIII, 9
   (Ritchie, Proc. Roy. Soc. Edin. 1905, XXV, 1085.
4. Hertz & Goodhart, Quart Jl. Med. 1908-09 II, 213.
5. Rihl, Zeitschrift f. exper. Pathol. u. Therapie, 1911, IX, 496.
6. Lewis, Heart, 1911-12, III, 279.
7. Lewis, Heart, 1911-12, IV, 171.
8. Mackenzie, Heart, 1910-11, II, 378, Case 37.
9. Turnbull, Heart, 1911-12, III, 89.
10. Lewis & Schleiter, Heart, 1911-12, III, 173.
11. Ritchie, Ed. Med. Jl. Dec. 1912.
12. Hume, Quart Jl. Med., 1913, VI, 238.
13. Lea, Proc. Roy. Soc. Med., Dec. 1912.
14. Hay, Liverpool Med.-Chir. Jl. 1913, p. 88.
15. Gibson, Brit. Med. Jl. 1906, II, p.1113.
16. Morison, Lancet, 1909, I, pp. 39 - 77.
17. (Lewis, Heart 1911-12 IV. Cases 1, 5, 9, 12.
    (Ritchie, Ed. Med. Jl. Dec. 1912, Cases 2 - 3.
18. (Lewis, Heart 1911-12, IV, Cases 2, 3, 6 & 8.
    (Ritchie, Ed. Med. Jl. Dec. 1912, Case 4.
    (Lea, Proc. Roy. Soc. Med. Dec. 1912, Case 2.
19. Price, Brit. Med. Jl. Mar. 8, 1913, fig. 15.
20. Lewis, Mechanism of the Heart Beat, Shaw 1911, p. 245.
21. Morison, Proc. Roy. Soc. Med., Discussion on 13, above.
22. (7, cases 2, 3, 6.
    (13, case 4.

AURICULAR FLUTTER.

INTRODUCTORY.

Auricular Flutter is a condition now well recognised by those interested in recent methods of diagnosis in morbid cardiac states. The term was first used by Mac William to describe the rapid, regular, apparently co-ordinate contractions resulting from faradic stimulation of the auricles in the lower mammals. (1). It was first applied to a human condition by Jolly and Ritchie (2), who in 1910 published a case in which the rapid action of the auricles was confirmed by means of the electrocardiograph, having been previously inferred from polygraphic records. (3). Hertz and Goodhart (4) also had published a case diagnosed from polygraph records, in 1908. Cases have since been recorded by Rihl (5. three cases), and by Lewis (6). and in 1912 Lewis (7) reported a further series of eight cases, along with those referred to above, and in addition included three previously reported by Mackenzie (8), Turnbull (9) and Lewis and Schleiter (10), in which the true nature of the condition had not been recognised. Ritchie (11) has described three further well-marked cases, along with three where the diagnosis was less well confirmed. A case is reported by Hume (12), and one by Lea (13): Hay (14) refers to four cases under his observation.

Auricular Flutter was probably present in cases

reported

reported by Gibson (15), and by Morison (16). These are not summarised in Lewis's paper. The former was a case of partial heart-block: one venous tracing (fig. 3) showed small waves at about 350 per min., the ventricular rate being 60 to 70. The latter was a case of mitral disease, the radial pulse was 108 to 114: a jugular tracing showed waves at 208-228, and auscultation revealed sounds at that rate; no ordinates are given in the tracing.

The various authors are agreed that the state is probably much commoner than has been generally supposed, and is likely to be missed in most cases which are not carefully investigated by the most modern instrumental means. Clinically the picture presented is considerably varied, as it appears to be compatible with fair health and absence of serious symptoms; frequently it is accompanied by signs of cardiac embarrassment; occasionally it would seem to be transitory. Speaking generally, it is found in the same class of cases as auricular fibrillation; and is probably often mistaken for it; it has frequently been observed to pass into fibrillation.

MEANS OF INVESTIGATION.

The signs of auricular flutter are found in the venous and arterial pulses, and in the electrical changes of the heart; these are recorded by the polygraph and the electrocardiograph, an adaptation of the string galvanometer of Einthoven.

Einthoven. To put it briefly, these show in auricular flutter rapid, regular auricular contractions: in fibrillation contractions varying much in rate, though more rapid than those of flutter, irregular in rhythm and in strength: the waves in the venous pulse resulting from the contractions being usually feeble as compared with the normal.

By means of the polygraph the venous (jugular) pulse is recorded simultaneously with the arterial (radial or brachial) or in some cases with the apex-beat. Unsatisfactory results may be due to the nature of the venous pulse or its being masked by respiratory or local muscular movements or tonicity: given an instrument in good working order, and reasonable expertness in using it and interpreting the records, misleading instrumental *artefacts* are not likely to appear, and variations of volume in the veins of small amplitude and rapid rate are certainly capable of being distinctly recorded. (see p. *11 of Case*). The fault of the instrument is likely to be failure to record distinguishably the weak systoles of the auricle; and in addition the expected waves are broken in upon by the usually non-synchronous (although related) waves due to ventricular systole. The venous pulse may even be imperceptible, especially in cases with good compensation and general circulation. The evidence afforded by the polygraph may therefore be trusted as far as it goes; and as Lewis points out (7, p. 216) the full diagnosis may often be made from the arterial pulse curve alone, which is not

not so inconstant as the venous.

The value of the electro-cardiograph lies in confirmation, or in the elucidation of cases where the other evidence is insufficient. While the diagnosis cannot be scientifically complete without its use, yet it may be reasonably certain; and treatment does not so far depend on an exact differentiation from closely allied pathological states.

RELATION TO ALLIED AURICULAR CONDITIONS.

Auricular Flutter must be differentiated from other pathological states of the auricle, also characterised by abnormal frequency of contraction, (the term contraction not necessarily including systole: see p. //.). The lowest rate of flutter recorded is 200 per min. (5, case 3), and the most rapid confirmed by electrocardiogram 330. (7, case 6). Possibly cases will be found outside these limits which will show all the essential characteristics of flutter, and all cases between 200 and 330 may not be true flutter. But it appears that auricular action between these limits is generally associated with other signs which help to differentiate it from the tachycardias of lower rate and the fibrillation of higher rate.

To contrast it first with <u>auricular fibrillation</u>:- as was pointed out on p. 1, flutter is essentially co-ordinate and regular; fibrillation is well recognised to be

inco-ordinate

inco-ordinate and irregular; this forms the basis for their differentiation by the polygraph and electrocardiograph. In cases of flutter we get at the best a series of regular oscillations in the venous tracing referable to the auricle. In fibrillation irregular vibrations may be recorded of small amplitude and high frequency; more often there is no sign of auricular action at all; the venous pulse is purely of the ventricular type. The electrocardiogram shows in flutter regular complexes due to auricular systole; in fibrillation the complexes due to the auricle are small, very irregular, and at rates from 390 to 522 per min. (2, p. 205). Lewis pointsout (7) that the type of auricular complex is remarkably alike in different cases of flutter, although exceptions occur (7, case 6); Ritchie reports another exception (11, case 3); Lewis (7, p.208) concludes that the general type indicates a definite and specific mechanism of causation, which is not the usual pace-maker in the ~~vene~~ sino-auricular node.

An important difference is also remarked in the arterial pulse. The ventricular action in flutter is timed by a regular mechanism (the auricular impulses) except in complete heart-block, when the rhythm is idio-ventricular. It is rarely however that the ventricles contract in response to each auricular systole at such high rates. Mackenzie (8) reported one such case, Ritchie another (11, case 5), Lewis a third (7, case 6), and cases of so-called

tachycardia

tachycardia are occasionally met with in which the ventricular rate is far beyond the ordinary means of counting. There is practically invariably heart-block as in fibrillation, which may be of any grade from the slightest up to complete, but the resultant effect upon the arterial pulse is very different. Except in complete heart-block, and excluding extra-systoles or intrusive beats, the ventricular rate in flutter always bears a simple fractional ratio (constant over periods which may consist of a few beats or continue for days) to the auricular; the commonest response when the patient comes under observation being to each alternate auricular beat. The ratio is usually expressed as one of auricle to ventricle, as 2-1, 5-1, 8-1. ~~(see p. 14.)~~. In fibrillation the arterial pulse presents complete irregularity, hardly two consecutive beats being alike, and shows no sign of a constant "greatest common measure" in the spacing of the beats. It should be observed that some pulses of flutter simulate this pulse (p. 16.). With complete heart-block the rhythm is here again idio-ventricular.

Flutter and fibrillation are however closely related clinically: the distinction is a recent one, and not absolute, although well-defined cases of each present a considerable contrast. They are often found under the same general clinical conditions, for instance in myocardial disease of rheumatic origin associated with changes

changes in the mitral valve, with some degree of failure of compensation and probably auricular dilatation. Flutter has been reported in at least six such cases (17). The clinical picture in some other cases of flutter is unlike that of fibrillation, being more like that of a tachycardia without definite physical signs except such as may be secondary to the rapid and inefficient heart action; six cases of this kind are recorded (18); in one of these (7, case 6) acute gout was present. Like fibrillation, it may be associated with complete heart-block, as in Jolly and Ritchie's case (2); (where there was a positive Wassermann reaction.)

The presence of some degree of heart-block in both classes of cases has been referred to. In both, too, the action of digitalis is very pronounced in increasing the amount of heart-block; further, under the action of digitalis a number of cases of flutter have passed into fibrillation. Fibrillation has also been observed to pass into flutter (7, case 16).

The study of some venous curves suggests that there may be cases at high and fairly regular rates which present some of the signs held distinctive of fibrillation: e.g. fig. 8 in Jolly and Ritchie's paper (2) may be interpreted as showing in the ~~various~~ venous curves for the most part "c" waves, each followed by a notched "v",

"V", the second half of which is more pronounced; small "a" waves appearing in the hollows, and in the two longest intervals, two being in each case adjacent; these represent a rate of about 400. The arterial pulse appears readily analysable into a succession of definite ratios to the "a" rate, as follows - 6, 5, 4, 4, 5, 5, 4, 4, 6. On turning to the electrocardiograms of the same case (Nos. 30 & 31) a curve typical of fibrillation is found, the auricular waves being small and very irregular, the slowest of them about 480 per min.; but the ventricular waves show a much higher degree of irregularity than those in the polygraph tracing, although the average rate is about 90 in each case, so that there is room for doubt whether the records can be taken as applying to identical auricular conditions. It is known that the auricular condition in some cases may vary within a very short period (11, case 4).

To take another instance, a jugular and arterial tracing published by Price (19) shows a fairly regular series of waves in the venous curve at the rate of about 380 per min., best seen in the long pauses. The arterial pulse is here very irregular, and of the type usually ascribed to fibrillation. It may be suggested that this represents a transitional state. No details of the case are given.

With

With reference to lower but abnormal rates of auricular contraction, and those cases of very rapid action which are more suitably termed tachycardia: while the probability is that these arise essentially in the same manner as flutter (7), and while they show the same failure of the auricles to respond to vagus influence (7 & 11) and to exercise, posture and nerve-stimulation (7), there is an important difference in their reaction to drugs of the digitalis series. The effect of these is in flutter always to produce marked slowing of the ventricles; in ordinary tachycardia they are usually entirely ineffective. In tachycardia, too, (but rarely in flutter) the ventricular rate equals the auricular, i.e. heartblock (if present at all) is so slight as not to reduce the ventricular rate below that of the auricles - all the impulses pass and are responded to. But for the effect of digitalis this might be supposed to be due to the slower auricular rate alone, for it is generally recognised that the more frequent the impulses received by the conducting A.-V. tissues the greater the degree of heartblock in any given case, and this applies even to the short intervals between successive ventricular beats (see p. 15.).

Lewis concludes (20) that "digitalis slowing in auricular fibrillation is due to the effect of the drug in increasing a pre-existing defect in the transmission of impulses from auricle to ventricle." At the time this was written (1911), auricular flutter was not dif-

ferentiated from fibrillation: in view of their very close relationship, the presence of some degree of heart-block in all cases of flutter, and their marked susceptibility to digitalis, it seems reasonable to suppose that this quotation represents the action of digitalis in flutter also.

It may here be remarked that Morison (21) still considers "so-called auricular flutter a true auricular tachycardia, to be distinguished from auriculo-ventricular tachycardia, the rationale of digitalis action being still sub judice."

There is however another aid to distinction, namely that in tachycardia one of the most striking features is the tendency of the attacks to begin with absolute suddenness; and to terminate equally abruptly, with resumption of the normal rhythm, either "spontaneously" or from very individual causes. While auricular flutter is perhaps frequently abrupt in onset, and may be intermittent (22), the usual account is that of a prolonged condition, which under observation has returned to the normal (if at all) through a stage of fibrillation under the action of digitalis.

Before leaving the discussion of the general features and relationship of auricular flutter, it should be noted that as found in the human subject clinically it differs from the experimental state described in the lower mammals

by MacWilliam in at least one important point, namely that the latter was checked or arrested by vagus stimulation. Clinical auricular flutter is, as far as we know, independent of vagus influence.

A reference to experimental fibrillation seems also desirable. The naked-eye appearances are thus summed up by Lewis (20, p. 192): "the walls of the auricle stand in a diastolic position; systole, complete or partial, is never accomplished; the wall as a whole is stationary, but careful examination of the muscle reveals an extremely active condition; it appears to be alive with movement: rapid, minute and constant twitchings or undulatory movements are observed in a multitude of small areas upon its surface. Mechanical records from the tissue have little or no excursion ..... At times the movement is coarser, and then mechanical records usually discover an attempt at regularity ..... the actual transition point from tachycardia to fibrillation, though it may be abrupt, is often quite indeterminable." And on p. 239, "Stimulation of the vagus ..... has one of two effects. If the fibrillation is of short standing it may suppress it. If it is of longer duration it usually fails to do so."

It seems a reasonable conclusion from the above that the venous curves which show attempts at regular waves - at very rapid rates are found when the fibrillation is

of coarser grade; such cases of fibrillation are the less complete, and more akin to flutter.

DIAGNOSIS OF AURICULAR FLUTTER.

The full scientific diagnosis must depend on all the evidence obtained by the polygraph and electrocardiograph. But a reasonably certain diagnosis can often be made without the latter, which is essentially a laboratory instrument. The electrocardiogram alone may be insufficient, as its interpretation may be difficult or impossible without other evidence: on the other hand it may be the only means of explaining the other evidence.

To take the simplest signs first:- it has been remarked that "the full diagnosis may often be made from the arterial pulse curve alone" (7).

The rate of the arterial pulse is seldom that of the auricles (see p. 5.). A two to one a - v ratio is common when the pulse is rapid: all grades of response may occur until the rhythm becomes idio-ventricular in complete heart-block: and different ratios may be intermingled, or succeed one another in stretches of regular beats.

Thus regularity of rhythm frequently appears, for short or long periods: or we find a certain grouping of beats recurs in an irregular pulse: such a curve should suggest flutter, or one in which the intervals between successive beats can be expressed in simple

integral numerical ratios to one another; or again if it is found that a stretch of curve selected with due precautions (see p. 16) is exactly equal to another (so selected) containing a different number of beats or the same number but of differing lengths. These features are not found in the pulse of fibrillation (20, p. 49): they obviously depend on a regular (though concealed) timing mechanism. In those border-land cases where the action of the auricle is not quite regular such analysis of course fails (e.g. 11, case 4).

Although the general diagnosis may be made as above, it does not follow that the auricular rate is directly ascertainable in the same way. Lewis (7) has dealt in detail with the analysis of a number of typical arterial curves, and remarks that in the absence of electrocardiograms certain analysis of tracings consisting only of 2 - 1, 4 - 1, 6 - 1, and 8 - 1 ratios or mixtures of these, is impossible, as they resolve themselves equally well into 1 - 1, 2 - 1, 3 - 1 and 4 - 1 ratios: the presence of an intermediate ratio, or of a 1 - 1 response is of great value in the analysis. He also refers (7, p. 212) to "the remarkable tendency for the ventricle to respond so that the ratio is an even one." A 3 - 1 response only occurred in one case of his series for more than a single beat; even single beats of 3 - 1 and 5 - 1 were rare, and in passing say from a 4 - 1 to a 2 - 1 type the curves showed mixed 4 - 1 and

2 - 1 periods rather than 3 - 1. Ritchie's case 2 (11) appears to confirm this. Hume (12) however reports a case of mostly 3 - 1 ratio, and in Lea's case (13) regular 3 - 1 rhythm was observed "over long periods".

The diagnosis from arterial curves is however by no means so simple as would appear from the above. Even presuming regular auricular action and absence of interrupting irregular impulses, there are several factors which greatly obscure the analysis in many cases. These are, as Lewis has fully stated (7):-

1. Pulsus alternans, which is common at high rates in degeneration of the myocardium.

2. When the ventricular response is frequent, the strength of arterial beats is materially weaker after pauses of less than a certain length in each case.

3. The amount of heart-block varies considerably from beat to beat, so that the conduction-interval between auricular and ventricular systole is relatively less after long pauses and more after short pauses: the effect being that shorter intervals are lengthened relatively to the others, in the sphygmogram.

4. Weak pulse-beats have often an increased pre-sphygmic period, and these being preceded by shorter pauses (2 above) are delayed already (3. above): so that a longer pre-sphygmic period is superposed upon $A_S - V_S$ lengthening to further delay the weak beat and consequently exaggerate the shorter beat which preceded it.

When the ventricular action is fairly regular, these factors are not so troublesome but when the response consists of mixed ratios they cause much confusion, and may lead to the simulation of a completely irregular pulse. The doubt which they cause as to the exact timing of any single beat leads to the most important rule of analysis, namely, that in picking out any stretch of the curve for comparison with another it must be seen that the upstrokes are comparable at the beginning and end of the stretch: that is that the interval preceding and that ending the period selected are of the same length, for only then are the upstrokes likely to be delayed equally, and to correspond to an integral number of auricular systoles. If this rule is not observed discrepancies in measurement may occur which may amount to so much as one auricular period, causing incorrect valuation of some beat in the stretch selected, or an erroneous conclusion that the pulse is completely irregular.

The effect of these factors on a given pulse in flutter is not easy to estimate until some progress is made with the analysis. The proportional share of each can only be guessed at, especially as they are probably not independent of one another, but are all dependent on processes occurring in the muscle or nerve-muscle of the heart, and appear as resultants, each more clearly in some conditions than in others. In a rapid pulse containing mixed ratios they will cause after each shorter series of auricular impulses (ending in a v. beat) a weaker pulse-beat, occurring relatively later than its

stronger neighbours. Thus the pulse-intervals tend to become equalised, but the beats to be unequal in strength, and this without the presence of pulsus alternans; when this is present, the stronger beats will be further lengthened at the expense of their weaker neighbours; if indeed this factor can be supposed to be added to the others, and to be so simply applicable to an irregular pulse.

An arterial pulse sign which in the absence of mechanical investigation may lead to suspicion of flutter is seen when a regular radial pulse changes rapidly on exercise to one of double the former rate, as 78 to 156: or if a regular pulse becomes very irregular after exercise (7 p. 197). A 4 - 1 ratio, say, may change to a 2 - 1, or a mixture of ratios may set in. Several instances are recorded (7). The explanation lies in the diminution of heart-block by exercise. It may be noted here that Lewis (7.p.210) considers cases of auricular flutter important because of the scientific observations they permit on auriculo-ventricular conduction; as the rate of the auricular impulses is not found constant under any other conditions, but varies under the experimental conditions when the auricles are not in flutter.

To consider the <u>venous pulse</u> in diagnosis:- the naked eye appearance of venous pulsation in the neck does

not appear to have been reported in most cases. Ritchie (11) refers to "moderate degree in right side of neck" (case 2.), "pulsation in jugular veins" at time of probable onset in case 3: Hume has also noted "rapid pulsation in jugulars" (12). But it is hardly to be expected that visible pulsation however useful as a sign of circulatory embarrassment, should be helpful in exact diagnosis.

Tracings are usually best obtained on the right side of the neck, owing to the shorter course of the innominate vein on that side (21.p.11). Unfortunately they are particularly apt to be unsatisfactory at the time when they are most required, i.e. when the patient first comes under observation with a rapid ventricular action, and is probably unable to make the journey necessary to obtain electrocardiographic examination.

At high rates the "a" waves are not only closely adjacent, but are complicated by the "c" and "v" which occur independently of auricular contraction, but depends directly or indirectly upon the ventricle. "C" and "v" themselves at high rates tend to become small and confluent, and auricular in flutter must often fall within the period of ventricular systole, so that the identification of "a", "c" and "v" becomes difficult or impossible. The auricle at the same time is probably considerably engorged, so that its systole is at the best feeble. Such confused and irregular

waves are shown, for instance in Lewis's cases (7. figs. 5, 6, 8, 9).

However, if the "a" waves be strong, they may predominate, as in tracings published by Hume (12, figs. 2, 3, etc.), by Turnbull (9), and Ritchie (11, fig. 7). In the first of these the auricular systoles were so strong as to produce very distinct waves in the apex tracing (12, fig. 6). In Lea's case (13), although small, they appear clear and regular, probably owing to a fortunate coincidence with "c" and "v" rather than to their strength. (Fig. 11 of Lewis's series (7) seems much less a succession of "a" waves than large "c" and "v" with a small "a" coinciding with them, in spite of his remarks on p. 204 relative to this tracing).

When the ventricular rate is lower, the "a" waves may often be more readily identified as they appear in the longer diastolic intervals. Thus they are clearly shown in fig. 1 of Ritchie's original case: "c" and "v" cause irregularity when they occur.

The <u>electrocardiogram</u> in general seems to lead to the most reliable appreciation of the respective auricular and ventricular rates. ~~In cases~~ Where the a. - v. ratio is 2 - 1, each second auricular complex may in some cases be practically coincident with part of the ventricular complex (7, case 15; almost so in case 6); or it may be mistaken for the normal "T" wave (7, case

16): in either case the rate is liable to be underestimated by one half. At other ratios certain "P" (auricular) waves may also be concealed, but the intervals between those remaining distinct and adjacent gives the key to the real rate.

This confusion is less than might "a priori" be expected, owing to the fact that the "P" waves in flutter together occupy the whole length of the curve; there is no interval between them corresponding to auricular diastole: they are not only large, but contiguous in all cases published except Lewis's case 7 (7, fig. 33) where the auricular rate was as low as 228. A notch on the downstroke of "P" may cause a rough resemblance to non-contiguity, (as in 11, fig. 1, and 7, figs. 30 and 31).

The "P" wave is thus long compared with the short and sharp "R" due to the ventricle, and is the less likely to be submerged. Its resemblance to "T" is closer. The "R" wave may be superposed on the crest of a "P" wave, and the latter retain its general outline, as is well seen in figs. 28, 29 and 30 of Lewis's series (7), and occurred in fact in Mackenzie's case (8), where the curve was originally supposed by Lewis to represent a singular type, owing to the possibility of a 2 - 1 ratio not being considered, auricular flutter not having been then at all generally differentiated from fibrillation and tachycardia.

This contiguity of "P" leads to the conclusion that

it occupies not only the period of auricular systole, but also diastole, at such high rates. Its length is in fact longer than normal, the normal length being 1/5 sec, diminishing with ordinary acceleration of the auricle; in flutter it is evident from the contiguity that with a rate of say 260 its length is $\frac{300}{260}$ fifths of a second. Lewis thinks that "P" depends on chemical processes which continue during diastole. (7, p. 205).

As has been remarked (p. 5.) the general form of "P" in different cases is remarkably constant, and unlike the normal. The wave in flutter usually consists of a more abrupt upstroke and more gradual downstroke. This type was present in 10 cases out of 13 examined by the electrocardiograph, collected in Lewis's series. In Ritchie's series (11) it occurred in one out of three so examined. No electrocardiograms from cases of flutter have since been published.

The electrocardiograph has shown that the response of the ventricle at such high auricular rates is frequently not to the immediately preceding auricular systole but to the one further removed: so that two auricular systoles may be practically completed before the ventricle responds to the former of them; heart-block supplies the explanation (7. p. 210).

Other points which have strengthened diagnosis in some cases are <u>sounds</u> attributed to the auricle and <u>movement</u> seen on radioscopic examination. These are

apparently only of very occasional value.

Auscultation showed sounds in Morison's case, at 208-228 per min., double the rate of the ventricles: the case was one of mitral stenosis and incompetence with tricuspid regurgitation; the sounds were heard in the neck over the jugulars.

Radioscopic examination showed in one case (7, case 6) rapid movement of the left auricular appendix: in another (4) rapid movement of the right auricle (seen repeatedly).

Lewis (20 pp. 89-90) gives a number of references to auscultatory and radioscopic evidence of auricular movement in general, so that the possibility of their being found useful in diagnosis cannot be altogether excluded.

Prognosis in Auricular Flutter.

A survey of the cases recorded shows that no general prognosis can be based upon the mere presence or absence of auricular flutter. In only two cases yet reported did death supervene, in one case (11, case 3) within about 11 days of the onset of flutter, in the other (11 case 6) the next day. It is of course likely that such cases will pass unobserved, as systematic examination for flutter in cases of rapidly progressing circulatory failure is hardly likely to be carried out.

Auricular flutter is compatible with a fair amount of health and activity. The case in which it has been observed longest is Jolly and Ritchie's original one (2); Ritchie reports (11) that the flutter has now lasted for seven years almost incessantly, and that the patient, a man of 63, enjoys fair health and is able to climb steep hills without discomfort. Complete heart-block, with ventricular rhythm from 30 to 60 (usually about 33) was present from the first, and goes far to explain the immunity of the patient from the circulatory embarrassment which probably otherwise would ensue.

Other cases observed over long periods were Hertz & Goodhart's, about a year, during 7 months of which the patient remained well and did housework, with auricles still beating at 216: and Rihl's second case, about 18 months. From the histories given in other cases there is every reason to believe that tachycardia if not

flutter was present continuously or intermittently over much longer periods.

The observed and estimated duration of the most fully recorded cases is given on pp. 29 + 30, along with other details.

Although in Jolly and Ritchie's case the presence of a high degree of heart-block was probably very advantageous to the patient, Lea's case (13) shows that the immediate prognosis is not necessarily bad even if the ventricular rate is high. This patient continued in good health for at least 3 months with ventricular rhythm at 130 (auricular 260). This obviously implies, as Lea suggests, a fairly sound myocardium.

The advantages to the circulation of a moderate ventricular rate are so well known that it is difficult to believe that prognosis is not better when the ventricular rate can be controlled, as is usually possible, by drugs of the digitalis series: general improvement is noted in several such cases (7, cases 1, 2, 3, 4, 8, 9 & 12), but in others did not follow (11, cases 2 & 3).

Immediate prognosis is naturally better when there is reason to believe from the history that auricular flutter has lasted some considerable time without bringing the patient into an extremely grave condition; many of the recorded cases are, as might perhaps be expected on the most general grounds, of this nature: also when the attacks have repeatedly occurred and passed off

spontaneously or under treatment. (8; 11, case 4; 13; 7, cases 6 & 16).

When it is superposed upon an old myocardial or valvular lesion, associated with ventricular enfeeblement, the prognosis is unfavourable: like auricular fibrillation it adds to the cardiac embarrassment (Ritchie, 11): but its effect like that of fibrillation, may usually be diminished by the use of drugs of the digitalis series which counteract its accelerating action on the ventricles.

Ritchie's cases 5 & 6 (11) suggest that like fibrillation it may constitute a stage in serious failure of the heart muscle.

The age or sex of the patient does not appear to have any bearing on immediate prognosis. In the tables on pp. 29-30 it is seen that there are four female cases to nineteen male: ages vary from 19 to 74 years.

Treatment of Auricular Flutter.

Passing over individual treatment and that of the general disorder from which the patient may be suffering, such as rest in bed, dieting, aperients, diuretics, stimulants, sedatives, etc., we have to enquire what treatment will directly affect the flutter of the auricles. This is confined to the group of drugs known as the digitalis series, digitalis, strophanthus, and squill, which it happens are particularly useful on more general grounds in such cases owing to their action in slowing the ventricles by diminishing or cutting off auriculo-ventricular conduction in cases especially of myocardial disease with rapid ventricular rate.

Their action on auricular flutter has in a number of cases been such as would hardly be expected, namely to cause fibrillation of the auricles. Out of nine cases in which the records show that digitalis was pushed, in doses approaching 1 dr. daily of the tincture, or 2 oz. of the infusion, fibrillation supervened in seven either during its administration or some days later. In a few of these cases the drug had to be withdrawn on account of vomiting, nervous symptoms or diarrhoea (strophanthus), and fibrillation was not obtained, or as noted, followed some days later. But in most of the cases in which fibrillation did not follow the use of the drug, there is no evidence that it was pushed.

Hay has referred to four cases under his observation, in one of which fibrillation followed the use of digitalis, in another the use of squill. The doses are not stated (12).

It occurred in one case (9) after administration of 33 Nativelle's Granules in eleven days; in another case 36 in twelve days failed to produce it (7, case 3).

In Ritchie's case 3 (11) it occurred the next day after the injection of .001 g. strophanthin intravenously; digitalis had been also given, about ½ dr. daily.

In a few cases the transition to fibrillation occurred apart from the use of digitalis; in Rihl's case 3 on several occasions, and in Lewis and Schlieter's case, where flutter was transient; fibrillation occurred and ended in flutter or in normal rhythm spontaneously. In Ritchie's case 5 it occurred during the administration of chloroform, and normal rhythm was resumed a few minutes later.

A return to the <u>normal rhythm</u> of both auricles and ventricles has been observed in a number of cases, in all cases except two (8. & 10) without passing again through a stage of flutter. This return to normal conditions has not been observed to take place directly from flutter (except in 10 and 8), but has been recorded in seven out of ten cases in which fibrillation had resulted from the use of digitalis (or squill). The time of return has varied from one day to something over eight days after the onset of fibrillation, but has not

been observed with exactness in a good many of the cases, and may in some have been later.

These observations indicate that the return to normal rhythm is the result more directly of the fibrillation than of the digitalis administration. Lewis concludes (7) that the return to N.R. shows that flutter owes its persistence rather to habit than a continuation of the exciting cause: the fibrillation breaks the habit, and the auricles, should they recover from the fibrillation, revert to their physiological rhythm.

When restored, the normal rhythm has been observed to persist, and to be still present weeks or months later; in one case as long as two years later (7, p.194).

The production of fibrillation in cases of flutter by digitalis is not yet explained. Ritchie (11) attributes it to depression of auricular conductivity, either by the effect on the vagus or on the muscle itself. It may be mentioned that a survey of the cases shows that in four cases slight slowing of the auricular rate occurred during the administration of the drug. In none of these did fibrillation supervene.* In one case slight quickening occurred, and in this case fibrillation set in. In most cases, whether fibrillation occurred or not, no change in the auricular rate was observed.

* In one case (Lewis's case 5), after fibrillation had been discharged; time 16 days after omission of digitalis.

GENERAL SUMMARY.

Case	Sex & Age	Prob.Path. Nature	Duration Observed.	Duration History.	Onset caused by	A. Rate	V. Rate	General Result
Jolly & Ritchie	M. 61	Syphilis	7 years	Not longer	Atropine	234 - 300	30 - 63	Fair health
Hertz & Goodhart	F. 39	Mitral disease from scarlet fever.	1 yr.	Few months	-	234	80	Fair health
Lewis, Case 1	F. 50	Rheum.myocard.	2½ wks.	3 mo.	Over-exertion	300 - 324	150-160	Improved
" Case 2	M. 65	---	3 mo.	Over 5 mo.	Excitement	280	140	Improved
" Case 3	M. 53	---	5 mo.	Over 3 yrs.	Over-exertion	264-324	132-167	Improved
" Case 4	M. 62	Influenzal ?	6 mo.	8 mo.	Influenza	270	135	Improved
" Case 5	M. 60	---	3 wks.	3½ mo.	-	300	150	Improved
" Case 6	M. 47	Gout	Once	? years	Exercise, emotion	330	165	--
" Case 7	M. elderly	---	Once	Several mo.	-	228	114	--
" Case 8	M. 52	---	Once	Some while	Exertion	260	130-90	--
Mackenzie	M. 47	---	11 wks.	? years	Sl. exertion	320	130-300	Improved
Turnbull	M. 74	---	4 mo.	-	-	280-300	140-150	Fair health

over

GENERAL SUMMARY Contd.

Case	Sex & Age	Prob. Path. Nature.	Duration (Observed)	Duration (history)	Onset caused by	A. Rate	V. Rate	General Result
Rihl, Case 1	M. 55	---	3 wks.	3 mo.	Exertion	246-300	143-150	-
" Case 2	F. 32	Mitral disease	18 mo.	? One year	-	206-222	103-111	-
" Case 3	M. 72	---	-	-	-	200-214	100-107	-
Lewis & Schleiter	M. 28	---	Several occasions	? 2 yrs.	-	280	140	
Ritchie, Case 2	M. 45	Syph.or rheum.	3 wks.	? 8 wks.	Exertion	280	140	Health indiff.
" Case 3	M. 37	Mitral; acute rheum	1 day	Not previously	Feverish attack	320	160	Death
" Case 4	M. 21	---	Once	? 1 yr.	Exertion	256-270	122-133	Health good.
" Case 5	M. 19	Chloroform pois.	Once	Not previously	Chloroform	227 ? 454	227	-
" Case 6	F. 41	Exoph. goitre	One day	Not previously	-	?	120	Death
Lea	M. 57	---	Few days	8 mo.	-	260	135	Good health
Hume	M. 63	---	8 wks.	Months	-	260	87	Fair health

No. P.M. Exams with reference to the flutter are yet reported.

20.

EFFECT OF DIGITALIS etc. in A.F.

Case	Drug	Effect V. rate	Effect A. rate.	Fibrillation.	N. Rhythm.
Rihl Case 1.	Digalen. 24 m. daily	Slowed 150-85 in 3 days.	Slowing 300-288 in 19 days.	Not obtained.	Not obtained
Rihl Case 2.	Digalen.	Slowed.	-	-	-
Rihl Case 3.	Digalen.	Slowed.	-	-	-
Hay Case 1.	Digitalis.	-	-	Obtained	Not Obtained
Hay Case 2.	Squill.	-	-	Obtained	Obtained.

Continued on p. 32.

Case	Drug	Effect V. rate	Effect A. rate	Fibrillation	N. Rhythm
Jolly and Ritchie	Digitalis	Appar. sl. quicker	Sl. slowing 268-251	Not obtained	Not obtained
Hertz & Goodhart	Digitalis	Slowed	None	Not obtained	Not obtained
Lewis, Case 1.	Tr. Digitalis 18 dr. in 32 days.	Slowed 2 - 1 to 4 - 1 in 30 days	Quickened 312 to 324 in 30 days	33rd day Digit.omitted	41st day Still present 3 mo. later.
Lewis, Case 2	Tr. Digitalis 8 dr. in 8 days	Slowed 5th day	None	9th day Digit.omitted	12th day Still present 1 mo. later.
Lewis, Case 3	Nativelle granules 36 in 12 days	Slowed 156 - 74 in 13 days.	Sl. slowing 300 - 280	Not obtained	Not obtained
do.	Tr. Stroph. 15 dr. in 13 days.	Slowed	None	Not obtained	Not obtained

22.

over

Case	Drug	Effect V. rate	Effect A. rate	Fibrillation	N. Rhythm
Lewis, Case 4.	Tr. Digitalis 4½ dr. in 6 days.	Slowed 138-94 in 7 days.	None	Not obtained	Not obtained
do.	Tr. Stroph. 15½ dr. in 15 days.	Slowed 136-90 in 10 days.	None	Not obtained	Not obtained
Lewis, Case 5	Infus. Digit. 27 oz. in 16 days.	Slowed 150-73 in 8 days.	None	17th day Digit omitted	Before 41st day Still present 6 wks. later.
do.	Infus Digit. 62 oz. in 31 days.	Slowed 145-67 in 18 days.	Slowing 276 - 248 in 31 days	16th day after omission of digit.	Before 44th day after omission Still present 7 wks. later
Mackenzie	Tr. digitalis 1 dr. daily	Slowed 140-55 in 5 days	- - -	Constant on 13th day	Constant after 5th week
Hume	Tr. Digitalis 6 dr. in 6 days.	Slowed 87-45 in 5 days. Digit omitted.	None	29th day	Not obtained

Over

Case	Drug	Effect V. rate	Effect A. rate	Fibrillation	N. Rhythm
Turnbull	Tr. Digit. 7 dr. in 7 days.	Slowed 150-88 in 7 days.	- - -	8th day Digit.omitted	Before 11th day
do.	Nativelle granules 33 in 11 days	Slowed 160-76 in 11 days. Digit.omitted.	- - -	14th day	Before 16th day
Ritchie, Case 2	Tr.Digitalis 29 dr. in 32 days.	Slowed 136 to 72 in 6 days.	None	31st day Digit.omitted	Not obtained
Ritchie, Case 3	Tr.Digit.½ dr. daily. Strophanthin .001 g.intraven.	Slowed 160 - 100	- - -	Next day after stroph.	2nd day
Lea	Tr.Digitalis 14 dr.in 28 days.	Slowed 2 - 1 to 3-1 in 4 days.	None	Not obtained.	Not obtained.

94.

EFFECT OF EXERCISE IN AURICULAR FLUTTER.

Case	Auricular rate	V. rate
Lewis 2.	Posture no effect	-
Lewis 3.	Exercise or rest no effect	V. slower when resting.
Lewis 4.	Few beats quicker standing. Exercise no effect.	Same as on A.
Lewis 5.	Posture and exercise no apprec. effect.	Same as on A.
Hertz & Goodhardt.	Exercise no effect.	Increased V. rate.
Jolly & Ritchie	Exercise no effect.	-
Hume.	Exercise no effect.	-
Mackenzie	-	Return of tachycardia.
Lea.	?	(2-1 rhythm independent of exercise or posture.)

EFFECT OF VAGUS PRESSURE IN A.F.

Case	R. or L.	If under Drugs.	Effect on A.	Effect on V. rate.
Lewis 3.	R. or L.	Digit. or stroph.	None.	Further slowing.
Lewis 4.	R. or L.	Stroph.	None.	Further slowing.
	R.	None.	?	Long pauses.
Jolly & Ritchie.	R. & L.	-	None	(H.B. already complete).
Rihl 1.	-	Digalen.	None.	Further slowing.
Rihl 2.	-	-	-	Slowing.
Rihl 3.	-	-	-	Slowing.
Ritchie 2.	R.	Digitalis.	None.	Further slowing.
Ritchie 3.	R.	Digit. & Stroph.	-	Slowing.
Ritchie 4.	R.	None.	Not arrested.	-

EFFECT OF ATROPINE IN A. FLUTTER.

Case.	Dose.	Whether under Digitalis.	Effect on A. rate.	Effect on V. rate.
Herz & Goodhardt.	1/50 gr. At. Sulph.hypod.	Doubtful.	None.	Quickened 80-150 in 12 min.
Jolly & Ritchie.	1/50 gr. At. Sulph.hypod.	Digitalis.	None.	Quickened 41-48 in 21 min.
Rihl (1).	.001 g.	~~Digitalis~~ Digalen.	None.	Quickened by restoring 2-1 rhythm.
Hume.	1/50 gr. hypod.	Digitalis.	None.	Quickened 60-106 in ½ hour.

Notes on a Case in which Auricular Flutter was probably present. ----------

Mrs K. 57 yrs., 2 children, now 25 & 29 years.

Complaint: Palpitation, and at night vomiting and inability to lie down.

History: Has never been very robust. Rheumatic fever at age of 10 years: this left her with weakness of the left side, which gradually passed off in the course of years. First pregnancy tried her severely. Attributes her ill-health partly to the "change of life." Menstruation regular at intervals of six weeks up to seven months ago; since then erratic, at intervals of a few months.

First attack like the present a year ago, last five weeks ago. These consisted of swelling of ankles, shortness of breath, "bronchitis," with frothy sputum; no haemoptysis. All have been relieved by treatment. (Digitalis.)

Present condition: 22/2/13.

A small, well-built, well-nourished woman; rather depressed. Lips pale and cyanosed. No jaundice. Respiration slightly wheezy and rapid; about 26; slight cough, but no sputum. Walks slowly

and with effort. Considerable amount of Oedema of ankles and legs. Can lie recumbent during day, but at night finds it necessary to sit up, chiefly on account of the vomiting; the vomit is copious, clear, frothy, colourless. Very short of breath on exertion, and during night, but gives no history of asthmatic attacks.

Circulatory System:

Chest well-formed. Considerable diffuse pulsation in praecordium and epigastrium. Jugulars show slight rapid irregular oscillation, but are not much distended. Apex beat in 5th space, 7in. from middle line. Upper limit of dulness 3rd space in left para-sternal line. Right border 1 in. to right of middle line.

Over the mitral area, a short, soft blowing systolic murmur of fairly high pitch, immediately following the first sound: not propagated to other areas. Over a small area below the nipple a low-pitched distant rumbling sound is heard during the longer diastolic pauses. The intervals between 1 & 2 are mostly equal; both are well defined, but weak in A.A. and P.A.

Radial Pulse about 155 (recumbent) soft, small, poorly sustained. Vessel

small, wall normal. Varies appreciably in force, and is frequently regular in rhythm, with occasional longer intervals (better seen in tracing 6, two days later.)

The lungs show a few rhonchi all over and some crepitation at the bases. No dulness.

The liver measures six inches vertically.

Tracing 5 shows the radial pulse. Alternation is conspicuous, and the analysis is difficult, but it is suggested that it corresponds to mostly 2 and 3 ratios, which would give an auricular rate of 333. Thus if there is flutter of the auricles, it is about the most rapid recorded, and is not far removed from fibrillation.

24/2/13. General condition unchanged.

	Pulse	Resp.	
Recumbent	150	28	
After exercise (6 times up)	157	30	1st Min.
	156	30	2nd Min.
	150	26	3rd Min.

Ordered Tr. Rhei Co & Spt. Ammon. Arom.

Tracing 6 shows the general regularity of the pulse, and the occasional longer (3 - 1) periods, with alternation. In 7 the venous pulse is seen to be small and irregular: it is impossible to identify the waves. In 8 an apex tracing is shown for comparison with 14, 17, 19, & 20.
Auricular rate 333.

26/2/13. Best night for two weeks. No sickness. Still orthopnoea. Oedema of legs increasing.

	Pulse	Resp.
Recumbent	152	27
After ex.(4 times)	155	29

Ordered Tr. Digitalis m. XXX three times a day. Other omitted.

Tracings 9 and 10 show a radial pulse of the same type as 5, 6 & 7. Tracing 9 happens to be the best and is the most readily analysed. The auricular rate calculated from them is 332. The venous pulse remains small and irregular.

28/2/13. Had bad night of palpitation and sickness on 26th. Yesterday appetite returned; feels stronger: no palpitation. Slept well in chair. Cyanosis still marked. Oedema less.

Radial pulse still small, but more forcible and better sustained, slower and more regular.

	Pulse	Resp.	
Recumbent	108	23	
After ex. (5 times)	116	26	
(pat. tired)	115	23	after ½ min.
	107	23	after 1 min.

Tracing 11 shows the venous and radial pulses. The latter appears analysable into periods of mostly 4 - 1 ratio, with occasional 5 - 1: the rate of the timing impulses would in that case be about 470 per min.: this taken with the rapid tremor in the venous pulse would indicate that fibrillation has established itself, although the picture is incomplete. The venous pulse shows some stretches of regular oscillation at a rate of slightly over ~~420~~ 540: this might be attributed to the rate of tremor in the veins almost coinciding with the natural vibration rate of the recording apparatus, but the latter

is found to be from 600 to 675. (see p.11 of case.)

3/2/13. Much subjective improvement.

Feels "like herself." No sickness. Sleeping well in chair. Still much oedema, some palpitation and cyanosis.

Radial pulse small, moderately firm, more irregular, about 92. Apex beat at same rate. Resp. 24.

Tracing 12 shows for the first time definite "c" waves in the venous pulse, with less definite "v".

8/3/13. No sickness or palpitation.

Oedema and cyanosis unchanged.

	Pulse	Resp.
	86	27
After ex. (3 times)	90	30

Tracing 13 shows definite "c" waves, and much irregular oscillation in the jugular pulse. There is still a certain amount of regularity in the radial pulse; four successive equal beats are bracketed.

12/3/13. Feels stronger. Oedema less. No palpitation. No diastolic murmur audible, and systolic very faint. Slight sickness,

Radial pulse 84, very irregular,

15/3/13. Much sickness last 24 hours: copious greenish vomit. No palpitation. Passing less urine. Oedema of sacral region: legs unchanged.

Radial pulse 52, very irregular, small, not easily compressed. Apex beat 86, strong and well-defined. Carotids beating forcibly. Resp. 24.

Digitalis omitted last night.

Tracings 14 and 15 show the very irregular radial pulse: and the lengths of the intervals which contain a dropped beat are almost identical.

"C" is very well marked in the jugular pulse: in the single intervals a wave appears at a variable distance before "c" (usually about ·75 sec.) Probably this is a "b" wave; in the short interval X it is apparently fused with the "c": its relation to the preceding "c" appears constant.

17/3/13. Good nights. No sickness or palpitation. Less cyanosis: Oedema still marked. Venous pulsation in neck very apparent. Passing usual quantity of urine.

Apex beat strong, 5 in. from middle line, same rate as radial pulse, 76. Resp. 25 No murmurs are audible.

Tracings 16 and 17 show the venous pulse with the radial, and with the apex beat. "c" and "v" are well defined, and the curve absolutely typical of auricular fibrillation.

18/3/13. Oedema less. No murmurs.

Apex and radial each 80.

19/3/13. No murmurs.

Apex and radial each 84.

Cont'd (f.g.)

20.3.13. Oedema rather less. No murmurs. Apex-beat and radial pulse 85. Resp. 24. Tracings 18 and 19 show again the marked effect of the strong carotid beat, which seems to produce a strong negative wave reaching its maximum about the middle of ventricular systole. The impression received during the taking of the tracing was that of movement of the tissues of the neck en masse at each beat. In 18 the "b" wave again appears. In the interval marked 1. it occurs a little under 1/5 sec. before "c"; at 2. about 1/10 sec. before; probably "c" is here delayed, so that the possibility of this being the "a" wave of a normal rhythm may be excluded.

21.3.13. Apex and radial pulse 90. No murmurs.

22.3.13. Oedema unchanged. Feels "very well". Apex-beat $5\frac{3}{4}$ in. from middle line in 6th space. Soft short systolic murmur at extreme apex. No diastolic murmur.

Radial pulse extremely irregular at times. (See tracing 20); rate 116 in chair, 104 recumbent.

Resumed Tr. Digitalis m. $\overline{XX}$ 4 hourly. Tracings 21 and 22 again show in places what might be taken for normal rhythm but there is no constant wave preceding "c" at a regular interval. *22 curiously resembles flutter.*

24.3.13.

24.3.13. Oedema diminishing. Cyanosis less than at any time since 22nd Feb. Apex 5 in. from middle line. Systolic murmur very faint. No diastolic audible.

Radial pulse 88. Resp. 24.

After 5 times up 84. Resp. 23.

Tracing 23 shows the very irregular radial pulse, and a venous curve which consists of marked "c" waves and much irregular undulation.

The case appears to have been one of flutter at about 333, which under the action of digitalis passed into fibrillation within two days. Neither the picture of flutter nor that of fibrillation is complete.

Slowing of the arterial pulse was definitely present on the second day (152-108). The venous pulse changed under the influence of the drug from one of a small irregular type to one showing a typical auriculo-ventricular curve.

After withdrawal of the drug the ventricular rate increased gradually from 76 to 106, but fell again on its resumption, with improvement in the patient's general condition. The auricular fibrillation probably continues, although at times an appearance of normal rhythm results from what seems to be a "b" wave in the jugular pulse.

Owing to the patient's serious condition it has unfortunately

unfortunately been impossible to obtain electro-cardiograms.

It was thought desirable to make some enquiry into the capacity of the clinical polygraph used (Mackenzie's), to record rapid variations in pressure or volume, and as to its tendency to vibrate in its own period. For the first, the jugular receiver was attached as usual, and held air-tight against the palm of the hand, while a succession of impulses was transmitted to the air enclosed by tapping the receiver. The result is seen in tracings 1 and 2, which show groups of 7 (in one case 8) waves recorded without confusion at a rate of over 400 per min. The tendency to vibration is damped at low amplitudes by the friction of the pen; in tracing 1, the paper being highly glazed, it is greater than in 2.

For the purpose of measuring the natural vibration time, the same arrangement was used; before taking tracing 3, the pen was practically emptied of ink by shaking; before taking tracing 4, it was loaded with as much ink as it would carry. The jugular receiver was again held air-tight and the air contained under slight pressure (as in 1 and 2); the pen was thrown into vibration by partial compression of part of the connecting rubber tubing, the pressure being sharply applied and removed.

removed.

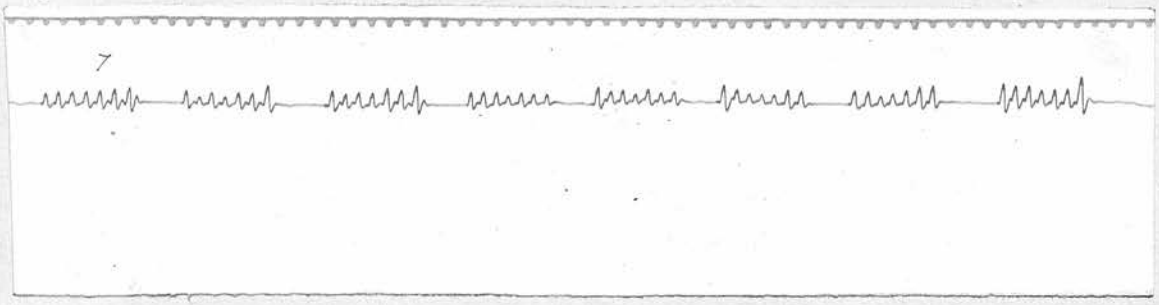
The rate in 3 is about 675 per sec.; in 4 about 640.*

In conclusion the writer wishes to express his indebtedness to Dr. James Mackenzie for the opportunity of working at the London Hospital; to Dr. Davenport Windle for the opportunity of seeing patients, and to both for the use of instruments; while he is indebted to both, and to Dr. W.T. Ritchie for suggestions.

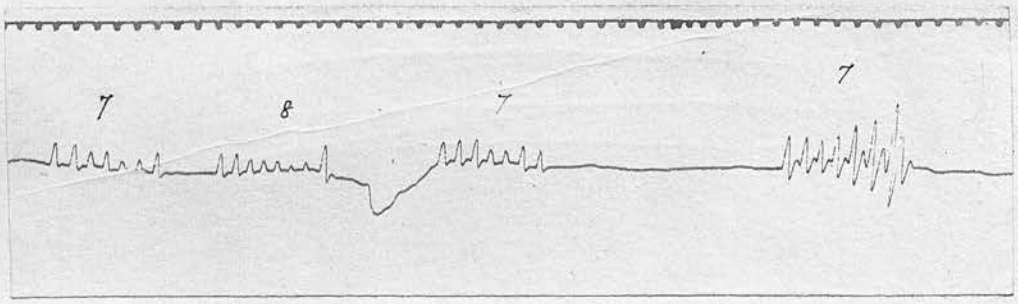
---

* Tracing taken on very rough paper with a fully loaded pen pressing heavily shows a rate of 600 per sec.

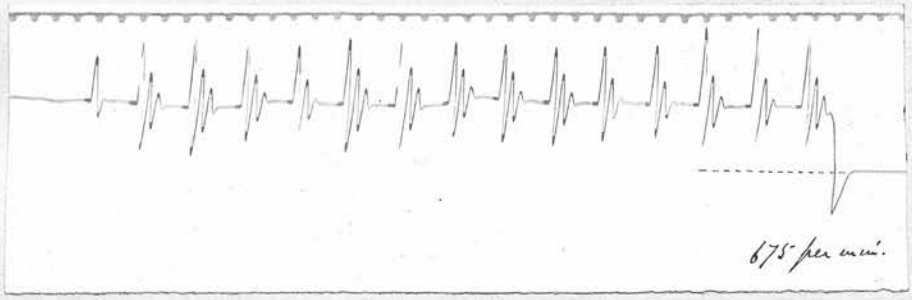
It was also found that the amount of pressure on the contained air was immaterial.



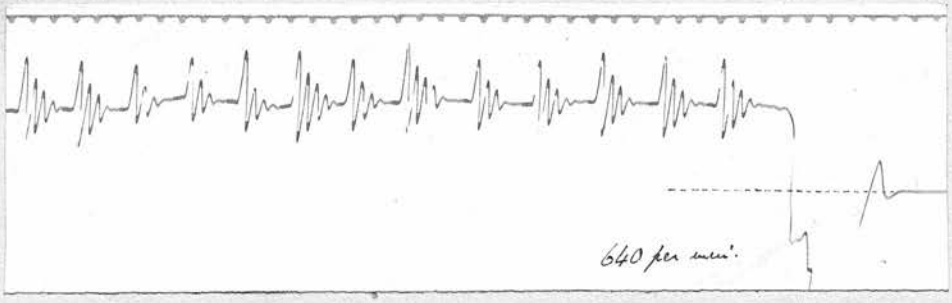
1.



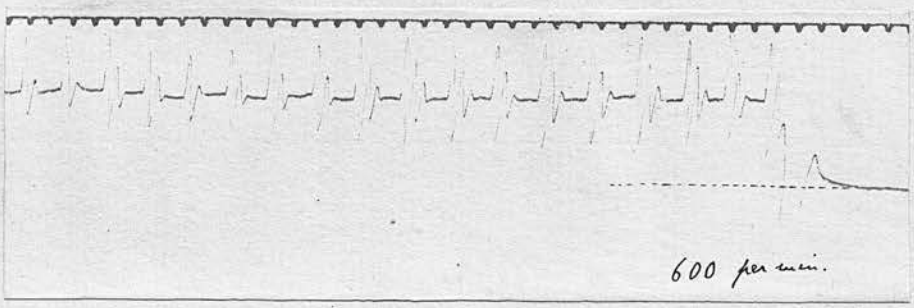
2.



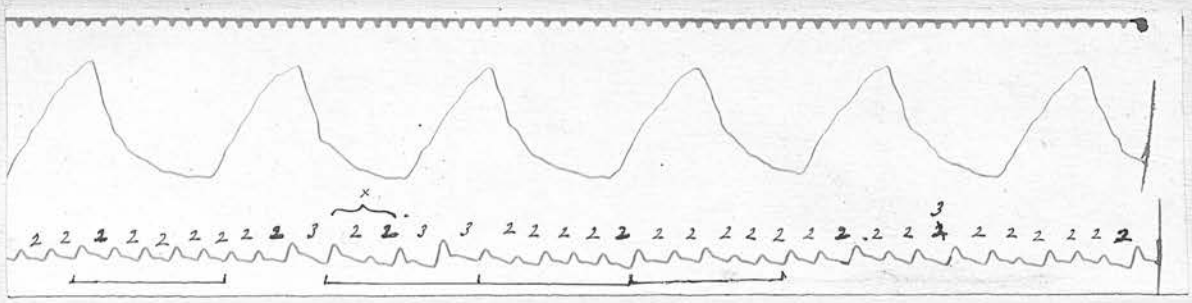
3.



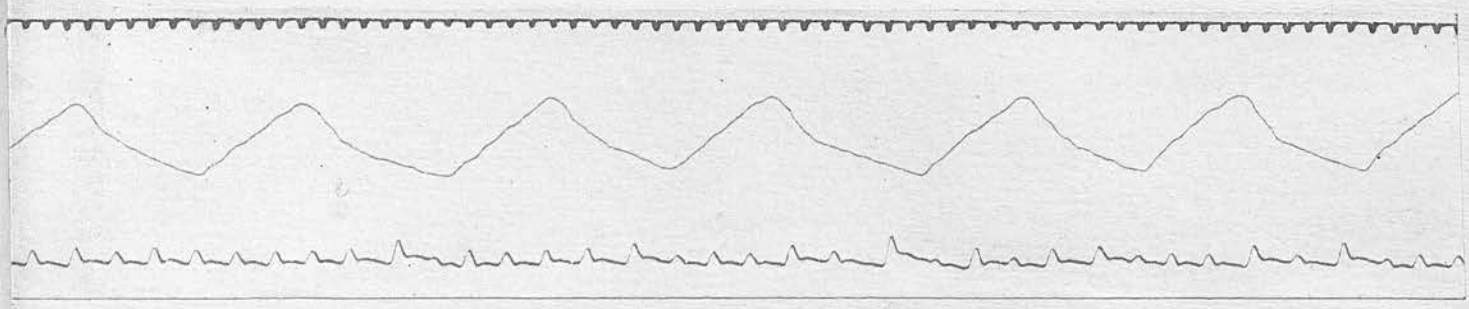
4.



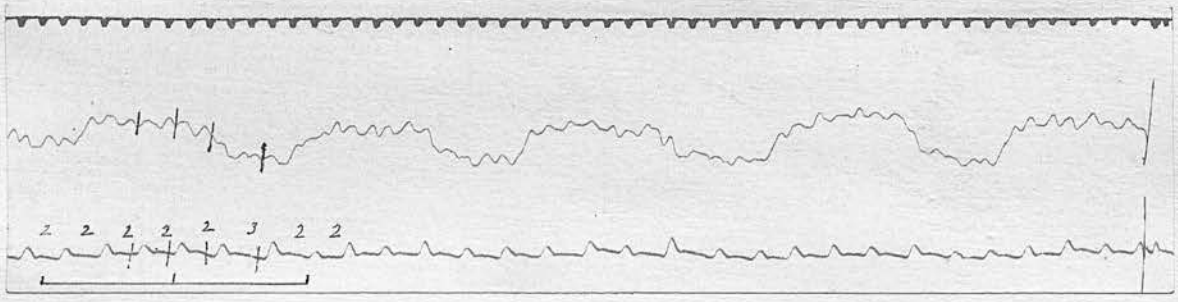
4a.



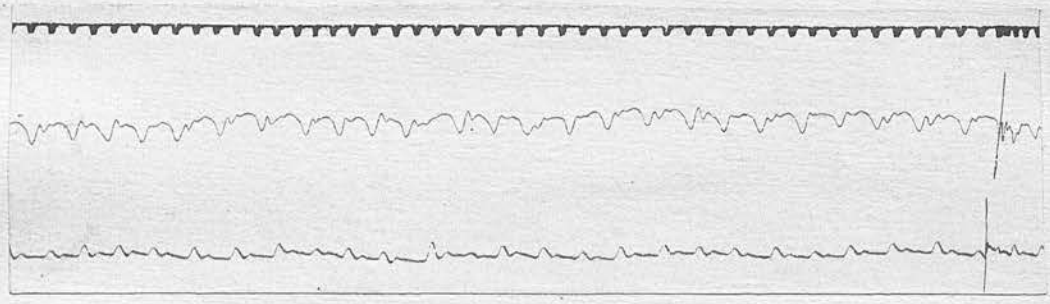
5.



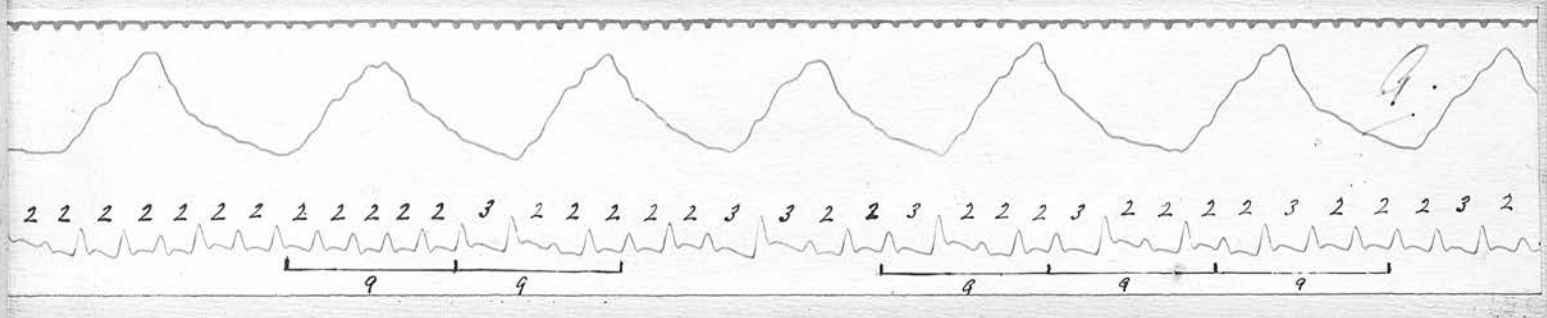
6.



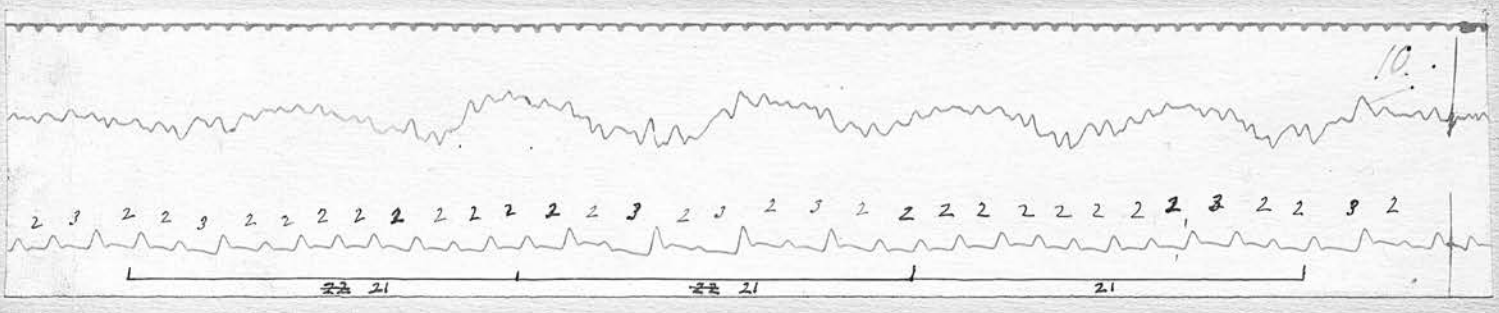
7.



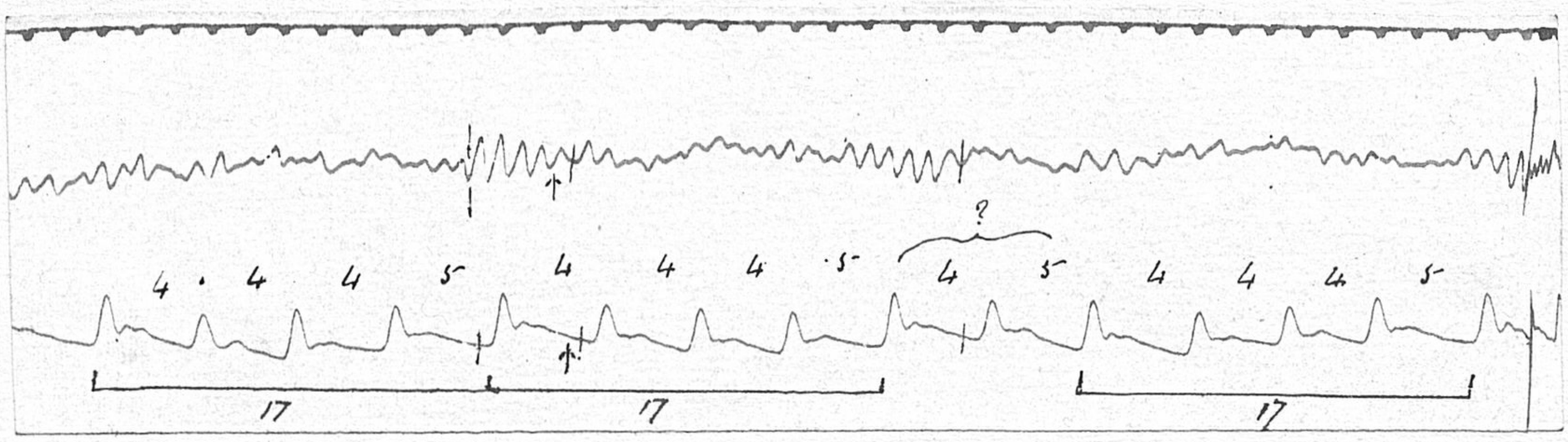
8.



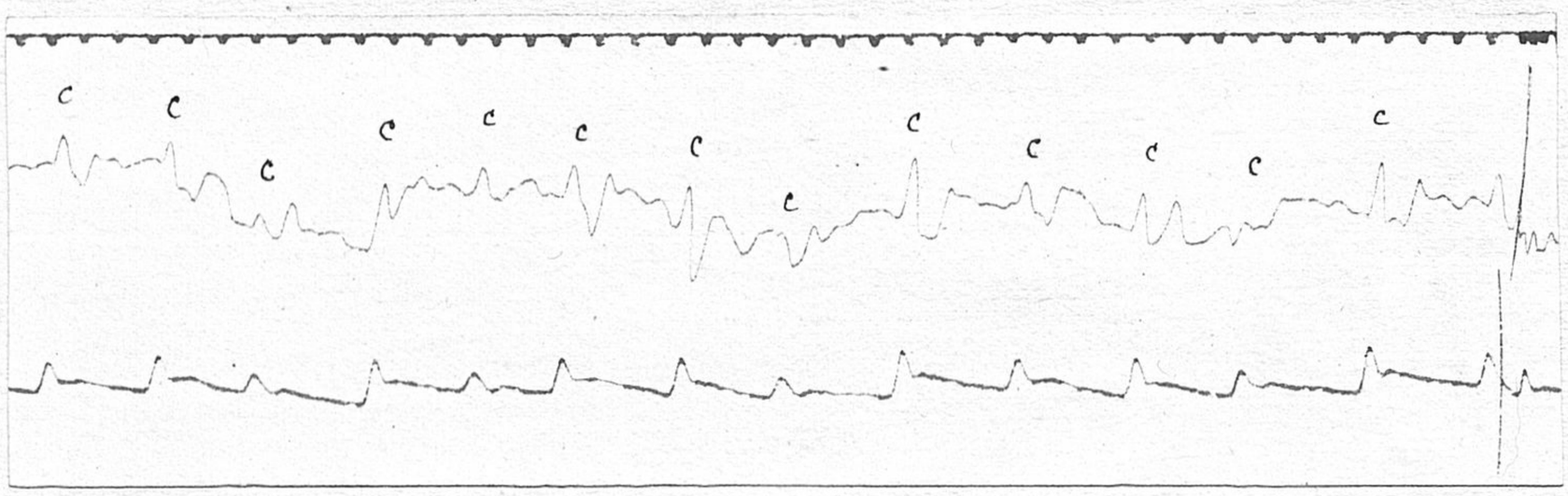
9.



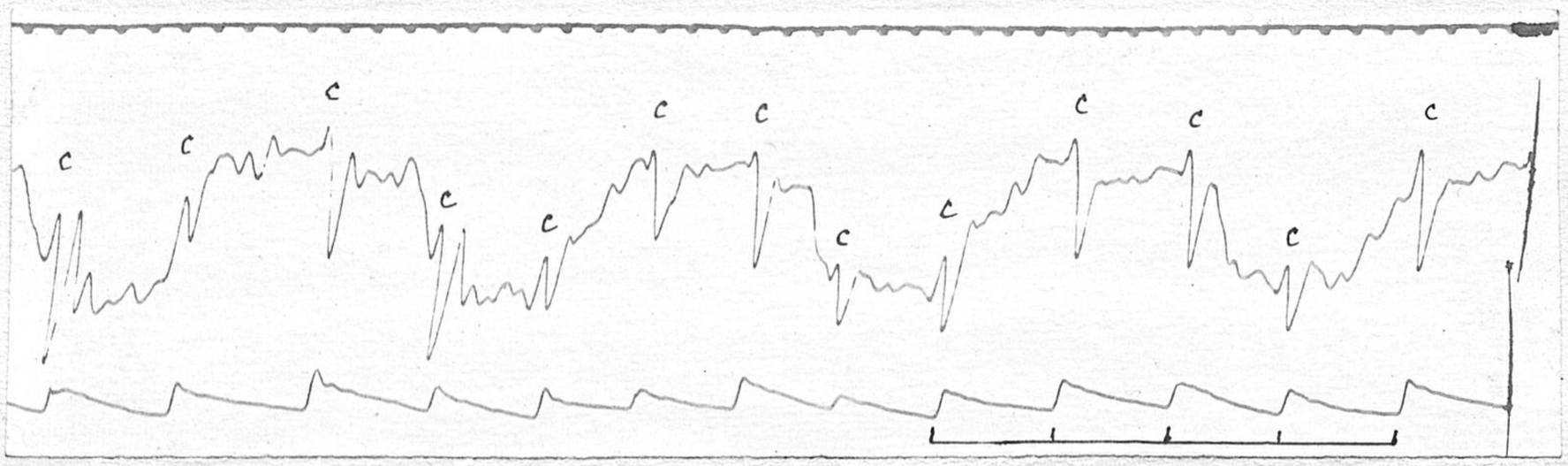
10.



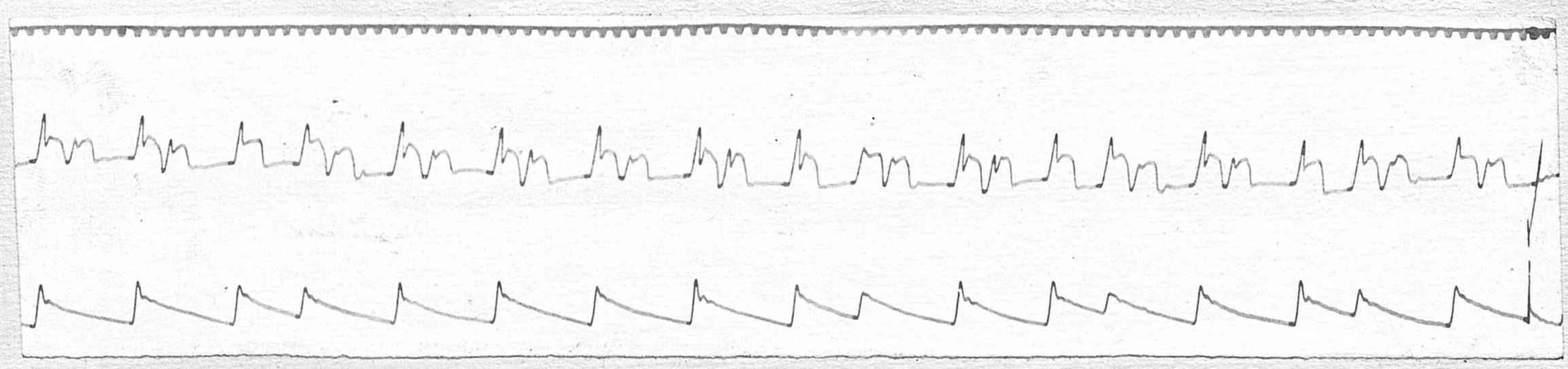
11.



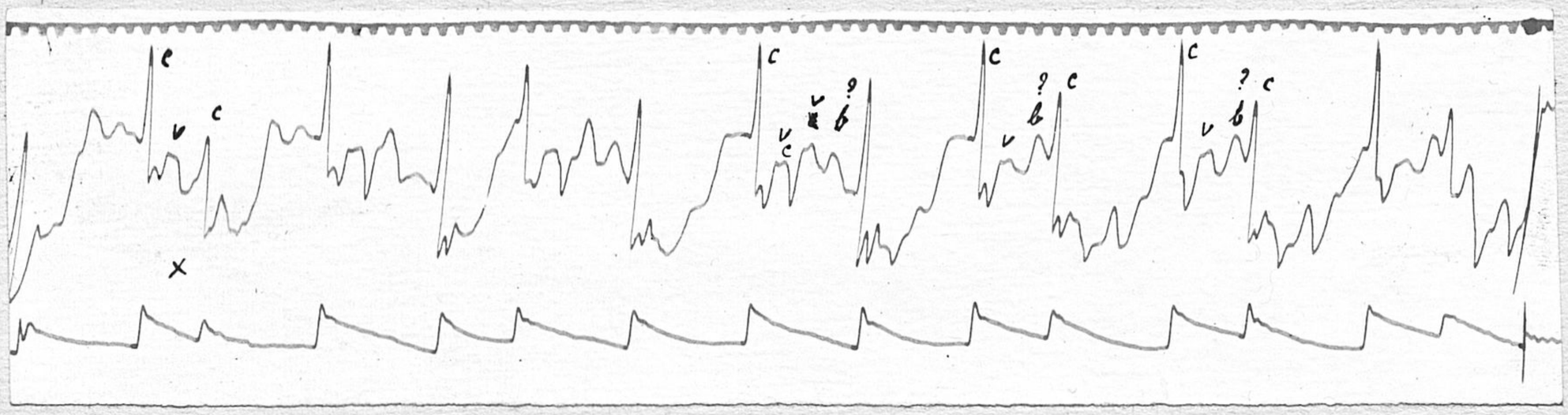
12.



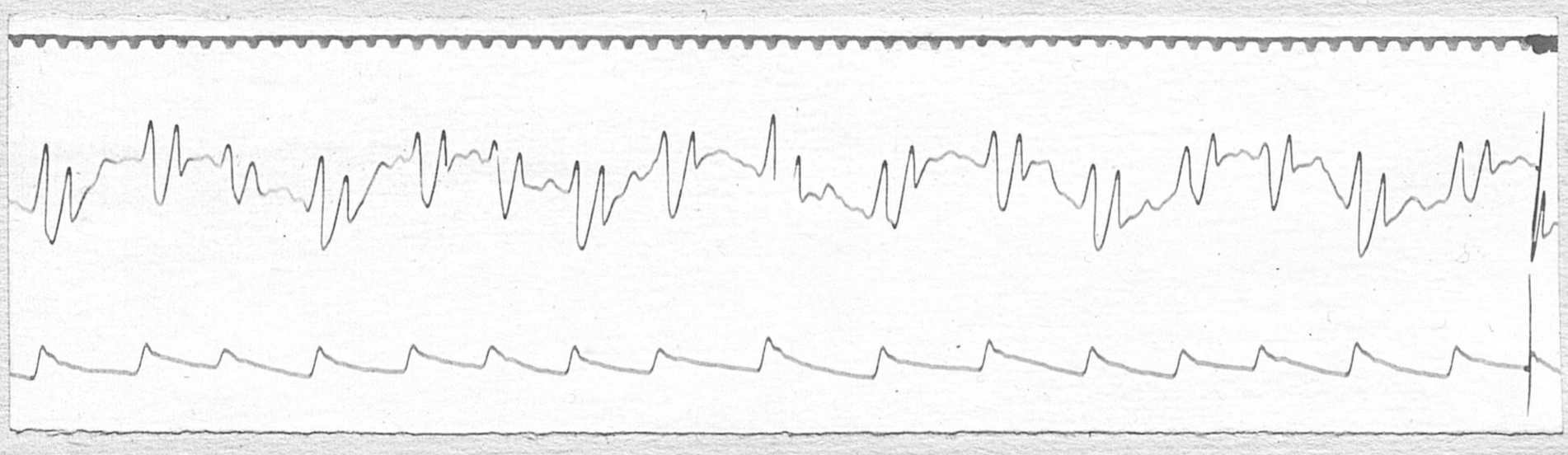
13.



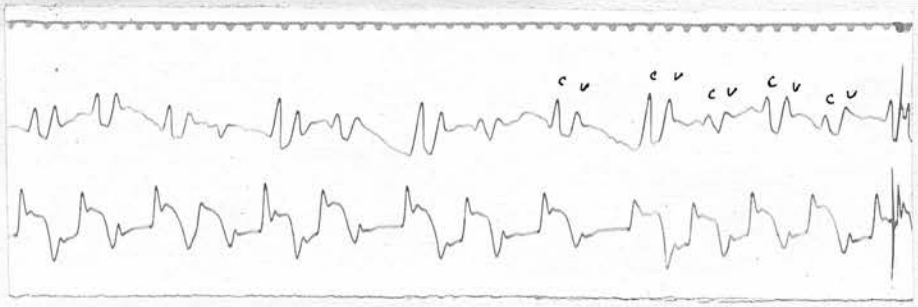
14.



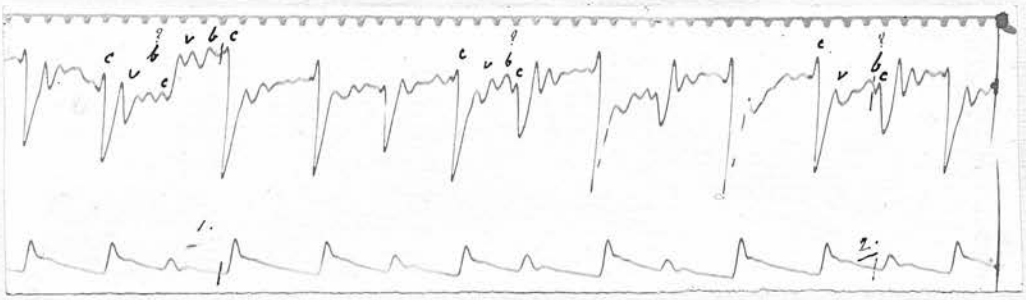
15.



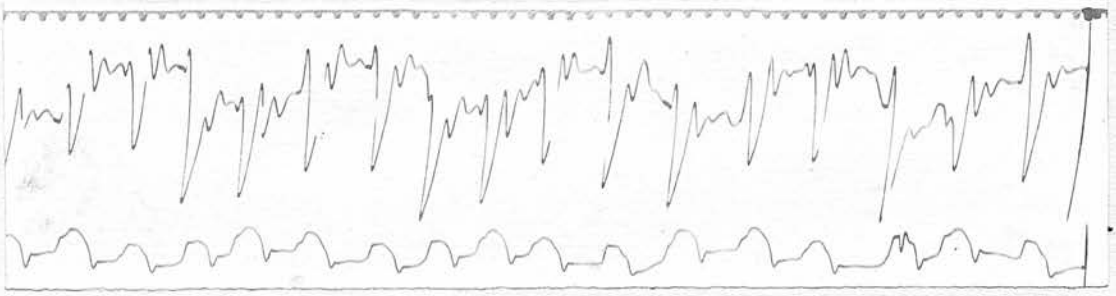
16.



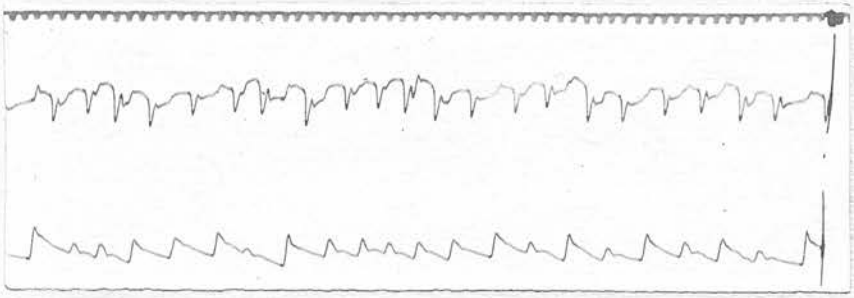
17.



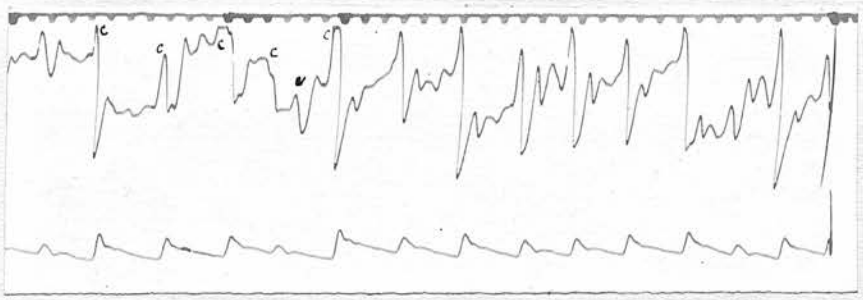
18.



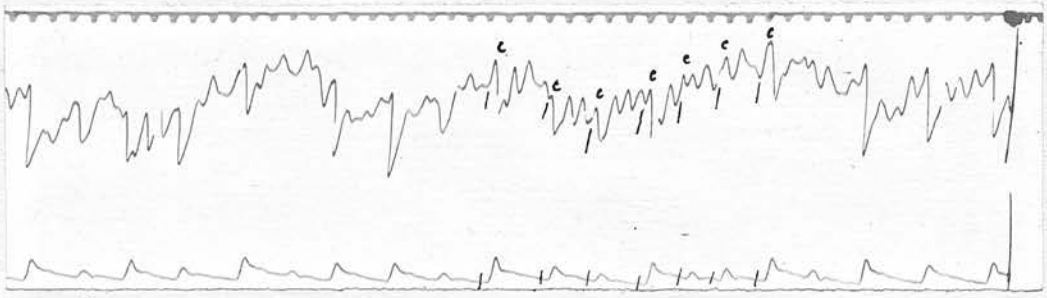
19.



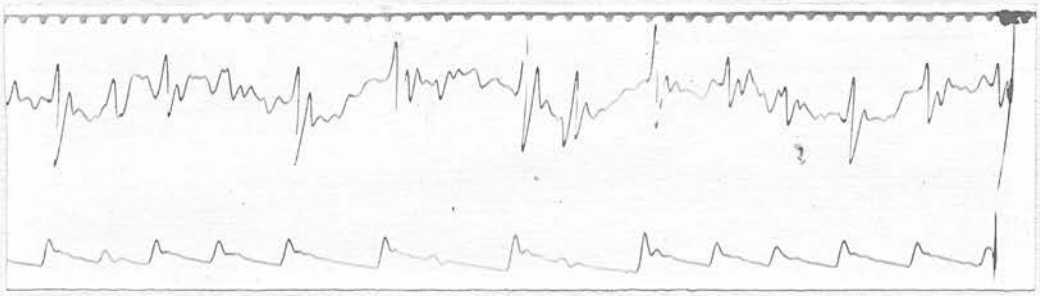
20.



21.



22.



23.