

THE SIGNIFICANCE OF THE DIASTOLIC WAVES OF THE VENOUS PULSE IN AURICULAR FIBRILLATION.

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PLATES 5 TO 7.

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General Outline of Plan of Study and Results.

In a previous study of the photographic venous tracings taken from clinical cases of fibrillation,¹ it was shown that the types of waves occurring during systole could be satisfactorily harmonized with and explained by photographic tracings previously obtained from animal experiments. The diastolic waves, however, could be given several different interpretations. This phase of the subject was, therefore, submitted to further experimental investigation.

The plan of study resolved itself into several phases. In the first place, it was desirable to determine whether large diastolic waves in the jugular tracings and in the electrocardiograms of many cases are diagnostic of a state of coarse fibrillation or associated flutter, or whether they are also present during fine fibrillation. Anticipating somewhat the results presented later, it was found that although coarse fibrillation was accompanied by somewhat larger diastolic waves, vibrations of considerable amplitude could be present during a state of fine fibrillation. In the electrocardiograms the diastolic waves were sometimes larger during the state of fine fibrillation.

The second phase of the experiments concerned itself with the cause of these diastolic variations. It at once became important to establish whether they represented pressure variations from the right auricle. To determine this, the right intra-auricular pressure was

¹ Niles, W. L., and Wiggers, C. J., *J. Exp. Med.*, 1917, xxv, 1.

recorded by introducing into the auricle by way of the azygos veins and superior vena cava, a sensitive venous optical manometer (Wiggers²). Again anticipating, it was found that with very few exceptions the true diastolic waves³ were not accompanied by similar variations of intra-auricular pressure, even when extremely sensitive manometers were used.

By elimination it is impossible to ascribe the jugular waves to any other factor than a traction of some portion of the heart upon the veins leading to the lower cervical region. By attaching threads connected with delicate tambour systems to different portions of the veins, and by studying the accurately recorded myograms of the auricle, it was found that during fibrillation a distinct periodic traction is exerted upon the veins and that this corresponds in rhythm with the diastolic waves of the venous pulse.

What type of cardiac action is able to affect the extrathoracic veins so as to alter their lumen and pressure without acting upon the intra-auricular pressure? Previous experimentation had shown that position changes of the contracting and relaxing ventricles are capable of exerting a traction upon the auricle. A comparison of these traction oscillations of the auricular myogram with the traction curves of the large veins and with the venous waves, led to the conclusion that the position changes of the relaxing ventricle could produce the large oscillations, which accompany fine fibrillation. This, however, is not the only mechanism capable of producing diastolic waves during coarse fibrillation. It was shown by myographic curves and auricular electrograms that the irregularly spreading excitation waves strike different areas of the auricle in such a way that they undergo irregular mechanical contractions. These contractions are apparently also able to exert a traction on the veins sufficient to cause diastolic waves in the venous pulse. These coarse contractions bear no relation to the electrical waves and the latter are therefore not of differential value between coarse and fine fibrillation.

² Wiggers, C. J., *Arch. Int. Med.*, 1915, xv, 77.

³ Double rounded waves, which are systolic in time, may, as previously pointed out, be mistaken for diastolic, unless accurate evidence of the presence of a systole is recorded. Such waves are accompanied by intra-auricular pressure variations.

Having thus outlined the plan and object of the experiments and anticipated the results in a general way, it is desirable to present more in detail the precise procedures and results from which the conclusions are derived.

Experimental Procedures.

Dogs anesthetized with morphine and chloretone were used. The external jugular vein was dissected low down in the neck. Over the venous pulsation a small tambour (2 cm. in diameter) covered with light rubber was lightly pressed and the volume changes of the vein were communicated to Frank's segment capsules as in clinical cases. Electrocardiograms were recorded from the right fore leg and left hind leg (Lead II) by Dr. Williams' American model of Einthoven's string galvanometer.

The thorax was opened and records of the intra-auricular pressure were taken by the type of optical manometer described elsewhere (Wiggers²). This instrument was, in a number of experiments, made exceedingly sensitive with the hope of detecting the slightest variation of intra-auricular pressure. So used, these manometers unfortunately pick up vibrations of the animal and of the building which appear as small rhythmic oscillations, and must, of course, be discounted in the analysis of records.

To various points on the right auricle were stitched several miniature myocardiographs recently described (Wiggers⁴). These instruments recorded, through optical capsules, the approximation and recession of the points to which they were attached.

Auricular fibrillation was induced by applying a moderate tetanizing current by a pair of ordinary platinum electrodes, or by sending the current through pin electrodes inserted into widely separated points on the auricle. The latter mode of stimulation more frequently produced a fine fibrillary movement; the former, a coarser fibrillation. We also attempted to produce fine fibrillation by simultaneously stimulating the right vagus, as reported by Robinson.⁵

When an electrocardiogram was being taken, the electrical variations of the stimulating current naturally spread so as to affect the

⁴ Wiggers, *Am. J. Physiol.*, 1916, xl, 218.

⁵ Robinson, G. C., *J. Exp. Med.*, 1913, xvii, 429.

galvanometer leads and hence are indicated in these records. For this reason the fibrillating movements persisting immediately after cessation of stimulation were recorded whenever possible.

During fibrillation of the auricle the ventricular rhythm becomes irregular and the rate exceedingly rapid. This condition does not correspond to the majority of cases seen clinically after treatment where the beat is slow. A corresponding slowing was experimentally produced in a number of ways, the two most successful means of producing a prompt slowing being the stimulation of the left vagus and the use of pituitrin.

Analysis of Results.

Anyone who is familiar with the technical limitations of optical experiments realizes that it is not feasible to record more than three or four circulatory records at a single time. Hence various combinations of tracings were taken in a total of 18 experiments. In this way the different events were related and the results many times duplicated. In analyzing the results it is not possible to proceed in a manner as methodical as that used in the general outline of results, because each record brings out isolated points which make the basis of the facts. It is proposed, however, in the selected records appended to discuss each phase of the investigation as outlined.

Fig. 1 is a section of a record taken on June 16, 1916. The intra-auricular pressure, the venous pulse, and auricular myogram from two points on the auricle 16 mm. distant were recorded. They are indicated on the record.

The record starts while the auricle is in fine fibrillation caused by the continued passage of a moderate tetanizing current. This condition continued during the period marked A-B. Upon cessation of stimulation the auricle lapsed into a state of coarse fibrillation, extending through the interval marked B-C. This was followed by a condition of regular auricular and ventricular beats.

The myogram record is interesting, first as showing the mode of cessation of auricular fibrillation in the dog. It is at once apparent that fine fibrillation (A-B) causes exceedingly small variations in the myogram curve, which cannot be recorded at all by ponderable

levers. This means that the combined effects of the separate fibrillary movements spreading irregularly over the auricle do not by interference or summation act to produce a mechanical shortening of auricular tissue. When stimulation is discontinued the auricle of dogs frequently regains its normal rhythm rapidly. Before this occurs, however, the auricle lapses into a condition of coarse fibrillation. During this interval (B-C) the auricular myogram shows definite waves indicating that distinct mechanical shortening is taking place. Within the interval of 1 second six such waves occur, making their rate 360 per minute. During this period, moreover, two large beats (M, N) occur which resemble the myograms of coordinated contractions. An examination of the intra-auricular pressure curve, taken with a very sensitive manometer (1 mm. = 0.8 mm. saline), shows that after discounting the fine periodic vibrations (1, 2, 3) due to extraneous vibrations and the waves due to ventricular systole (sys), there are no waves during either fine or coarse fibrillation. The first auricular attempt at a definite contraction (M) is also accompanied by no marked rise of intra-auricular pressure. The second contraction (N), on the contrary, is accompanied by a definite wave (N'). Time comparisons show that the rise of pressure corresponds absolutely to the onset of the myogram contraction. The onset of real coordinated contractions (O, P) is indicated by their characteristic contour (Wiggers⁴). These contractions are preceded for the first time by the pressure rise, as is normally the case.

The second point of interest lies in the relation of the diastolic waves of the venous pulse to the auricular myogram and intra-auricular pressure variations. It will be seen that between the waves accompanying ventricular systole (sys) are definite diastolic waves (a) occurring at the rate of 6 to 8 per second. Although somewhat smaller, they are present during coarse as well as fine fibrillation. During the former condition they appear without any evidence of mechanical shortening in the auricular myogram. During the latter condition, as shown by the lines sketched, they follow a short interval after the smaller oscillations in the myogram.

A careful consideration of the records brings out the following points. (1) When the auricle passes out of a condition of fine fibrillation, it first passes through a stage of coarse fibrillation. During

this stage definite mechanical contractions gradually appear and these finally give way to normal coordinated contractions. (2) Diastolic waves of the venous pulse may occur during the state of fine as well as coarse fibrillation. In the latter condition they are, however, generally larger in amplitude, and correspond to an actual mechanical shortening of the auricle. (3) The diastolic waves of the venous pulse are not due to transmitted auricular pressure variations during fibrillation. Such variations occur only when the auricle gives coordinated contractions.

The records shown as Fig. 2 are from an experiment on March 8, 1916. The upper record shows the auricular myogram taken by a miniature myocardiograph and recorded by a segment capsule more sensitive than that used in the records of Fig. 1. The electrocardiogram (Lead II) is recorded below with time divisions of 0.05 of a second.

The records are typical as showing the relations between the electrical variations and accurately recorded mechanical changes in the auricle during fine and coarse fibrillation. The record starts with the auricle in fine fibrillation, a condition produced in this instance through simultaneous stimulation of the right vagus. When vagal stimulation was stopped the auricle gave several large contractions (1, 2, 3) simulating coordinated beats, after which it lapsed into a coarse fibrillation.

To facilitate reference to specific waves, the ventricular systoles as indicated by the R peaks in the electrocardiogram and the corresponding sound vibrations in the myogram are marked alphabetically. Following each R peak in the electrocardiogram a wave corresponding in time and contour to a T wave must be recognized.

During fine fibrillation the auricular myogram clearly shows a series of small mechanical variations irregular in period and amplitude. The electrocardiogram also shows small rhythmic variations. They are only occasionally related to the mechanical shortening. Thus between Systoles A and B only eleven distinct mechanical variations occur while in the electrocardiogram at least sixteen waves are present. Of the mechanical variations two (x') are definitely preceded by electrical waves(x); but two other distinct mechanical waves (w, y) are entirely without corresponding electrical waves.

Toward the end of the record where coarse fibrillation had taken place the auricular myogram shows a series of larger but still irregular mechanical contractions. If we eliminate the variations of the electrocardiogram which probably represent T waves, there remain very few waves that correspond to the mechanical contractions.

Separating the interval of coarse and fine fibrillation is an interval where several coarse beats occur (1, 2, 3) which somewhat resemble coordinated beats. A comparison of the electrical variations for these beats indicates that Wave 1 is possibly preceded by a corresponding electrical variation; Wave 2 is certainly preceded by no such wave, while it is impossible to determine whether the electrical variation accompanying Wave 3 represents a T wave or an auricular wave.

A study of the curves makes it evident (1) that while large electrical variations may be present during coarse fibrillation they are by no means a constant accompaniment or diagnostic of this condition, (2) that distinct oscillations in the electrocardiogram more frequently accompany fine fibrillation, and (3) that the lack of relation between mechanical and electrical variations during ventricular diastole are characteristic of auricular fibrillation.

Fig. 3, taken on June 25, 1915, shows in order, the external jugular pulse, the auricular myogram, and the intra-auricular pressure curve. The auricles were in a state of coarse fibrillation at the beginning of the record. Then the left vagus was stimulated, resulting in a state of finer fibrillation together with a slower ventricular rhythm.

The curves are of interest as showing first, the influence of mechanical auricular movements on the jugular and venous pressure during long diastoles. The case duplicates the clinical cases slowed by digitalis medication. The beginning of each ventricular systole is indicated by a series of large vibrations in each record (A). The end of systole can be determined approximately by the fall of intra-auricular pressure (B). During the first long diastolic period (1-2) the auricular myogram contains only very small variations, and except for an expected gradual rise of pressure the intra-auricular curve shows no oscillations. Each ventricular systole, however, exerts a definite traction on the auricle consisting of a primary vibration (a-b-c-d) and a slower secondary movement (d-e-f). It will be

noticed that waves corresponding to these tractions are communicated to the jugular vein, and are shown in the upper record. This indicates that ventricular systole, which in the normal heart exerts some traction upon the auricle, pulls even more vigorously upon the distended auricle and veins during fibrillation. Early in diastole a decremental series of oscillations occurs in the auricular myogram (g-h-i-j-k). As similar waves occur in long normal cycles, they can only be interpreted as due to position changes of the relaxing ventricle in both cases. It is evident that these waves are sometimes accompanied by waves in the venous pulse.

Experiments of this nature show that: (1) When diastole is long few wavelets in the myogram or in the jugular can be ascribed to the fibrillating auricle itself. (2) Both ventricular systole and diastole by active traction and recession are capable of producing a passive shortening and lengthening of the auricular fibers, and these may produce similar effects on the veins. This traction communicated to the veins is at least one cause of the large diastolic waves occurring during fibrillation.

Fig. 4 is another record taken from the same animal. It shows a state of coarse fibrillation modified by the action of pituitrin, which moderately slowed the ventricle.

The jugular tracing shows a series of rhythmical waves averaging 300 per minute. From such a record the erroneous impression could easily be derived that the auricles were contracting at this rate. A comparison with other records shows that the auricular mechanical contractions were entirely irregular and that the jugular waves were the result of a fortunate interference between the effects of auricular beats and ventricular traction. Upon the waves are superimposed smaller vibrations which upon careful study can be assigned only to heart sound vibrations. Such tracings show that large coarse rhythmical waves in the jugular must be guardedly accepted as evidence of regular auricular activity.

Fig. 5 shows four segments of a tracing taken on March 8, 1916. The upper curve is a record of the movements of the superior vena cava; the second curve is an auricular myogram; while the lower record is an electrogram obtained by leading off from the two auricular points to which the myogram was attached.

Segment A shows a normal cycle obtained during stimulation of the right vagus. The auricular myogram has the contour described by one of us (Wiggers⁴) as characteristic for a coordinated beat. Ventricular systole, the duration of which is indicated by the two sound vibrations (1S-2S) in the superior vena cava record, causes a jog (a) and several secondary waves (a-b-c-d) on the relaxation curve of the myogram. During early ventricular diastole another wave (d-e-f) is added. These tugs are transmitted to the wall of the superior vena cava as shown in the upper record. Slightly preceding the auricular myogram, the auricular electrogram shows a diphasic variation (1-3) evidently of auricular origin. This is followed by an R peak derived from ventricular activity. As no electrical variations accompany the secondary waves of the myogram (a-b-c-d-e-f) they cannot be assigned to an active contraction, but must be due to passive traction.

Segment B was taken during a state of auricular tachyrrhythmia (flutter) induced by previous tetanic stimulation of near points on the right auricle. Each ventricular systole is indicated by a small positive peak (R) in the electrocardiogram and by a series of sound wavelets (S) in the superior vena cava record. In contradistinction to the secondary wavelets following a normal vagus beat, for example, each mechanical variation in the myogram is accompanied first by a small positive, then by a large negative variation in the electrogram. The incidence of irregularly recurring ventricular systoles results in irregular traction upon the auricle and hence the auricular myogram waves during flutter appear of irregular amplitude.

Segment C shows the transition of an existing auricular flutter to a coarse fibrillation upon stimulating the right vagus. The myogram as well as the superior vena cava record still shows a series of small waves which could not have resulted from a ventricular traction, for (1) complete stoppage of the ventricles had occurred, and (2) the electrogram shows a rapid series of irregular variations.

Segment D shows a portion of the curve after vagus stimulation had ceased. The auricle lapsed into a state of coarser fibrillation and the ventricle resumed its beat. Owing to the large auricular waves present in the electrogram derived directly from the auricle, the ventricular systoles are difficult to differentiate in the electrogram,

but are clearly marked by sounds (S) in the superior vena cava record. While it is probable that some of the mechanical variations of the myogram are due to the traction of these ventricular systoles, the presence of the numerous electrical variations of undoubted auricular origin shows that many of the mechanical variations must have been due to the coarse fibrillation of the auricle itself.

A study of these records shows that: (1) Ventricular position changes may cause a series of passive mechanical changes in the auricle (unaccompanied by an action current) which may be the source of some diastolic variation in the venous system. (2) Since the waves during auricular flutter and coarse fibrillation are accompanied by electrical variations and since these waves may occur when ventricular activity is entirely in abeyance, it follows that not all the mechanical variations in the myogram and superior vena cava can be accounted for by ventricular traction. On the contrary, it must be assumed that during coarse fibrillation the impulses travel irregularly over the auricle and excite the various sections of auricular musculature to irregular active contractions, and that these contractions by drawing upon the veins cause large oscillations to appear in the venous pulse.

SUMMARY.

With the clinical recognition that different degrees of fibrillation occur and that these in turn are closely related to a coordinated type of auricular tachyrrhythmia (flutter); further, that one type may lapse into another or into a perfectly normal rhythm, the conviction has grown that finer and coarser types of auricular movement may be recognized by the amplitude of the diastolic waves of the electrocardiogram and venous pulse. The present investigation into the cause of these waves has shown that this is not possible. There is no theoretical or experimental reason for the assumption that any fixed relation exists between the amplitude of the electrical variations of the electrocardiogram, which are the resultant of variations accompanying the irregularly spreading excitation wave, and the degree of mass contraction following. The large recurrent waves of the venous pulse, which may with more reason be regarded as related to the size of the auricular mechanical contractions on theoretical

grounds, are also shown to be without differential value. The reasons for this may be briefly summarized.

In the first place, the presence of diastolic waves is contingent upon a slow heart rate and long ventricular diastoles. With rapid heart rate their occurrence is prevented by the closely placed systolic variations. It is therefore conceivable that both coarse and fine fibrillation as well as flutter will be without diastolic waves as long as the heart is rapid.

Even in the cases with long beats present their significance must remain doubtful. It is true that in the experiments the waves accompanying coarse fibrillation are as a rule somewhat larger than those occurring during fine fibrillation; waves of considerable size may, however, be present in fine fibrillation. Hence, as long as no calibrated method of recording is possible, it is difficult to draw any inference. It is possible from the same patient to record with the same apparatus diastolic waves of varying amplitude by merely changing the pressure of the receiving tambour.

The chief reason that the amplitude of these waves cannot be regarded as of differential value is found in their origin. Fine fibrillating movements of the auricle do not in themselves produce waves in the jugular. They produce neither pressure variations in the auricle, nor exert any traction upon the veins. The only factor capable of producing jugular waves during fine fibrillation seems to be the traction exerted by the position changes of the ventricle on the auricle and large veins. This may, in a measure, explain why the diastolic waves recorded from the apex and second left interspace of patients often closely correspond with those simultaneously recorded from the jugular. The term "fibrillary waves" commonly applied to the smaller of these variations is evidently poorly chosen when their etiology is considered. The coarser contractions of the auricle during coarse fibrillation also produce no pressure changes within the auricle. They are vigorous enough at times, however, to exert a traction upon the venous walls. Hence, the waves during coarse fibrillation may be regarded as partly of ventricular and partly of auricular origin, or, as is frequently the case, as due to an interference of the two tractions. It is owing to their dual origin that they are more numerous and distinct when recorded from the same animal without changing the position or pressure of the receiving apparatus.

EXPLANATION OF PLATES.

PLATE 5.

FIG. 1. Intra-auricular pressure, jugular pulse, and auricular myogram, showing transition from fine to coarse fibrillation and from this to coordinated contractions. Diastolic waves of venous pulse (a) occur during fine as well as coarse fibrillation. Shortening of the auricle causes downward movement in this and all other myogram curves. Time 0.2 second.

FIG. 2. Auricular myogram (downstroke in systole) and electrocardiogram, Lead II, showing that diastolic waves of the electrocardiogram do not correspond to mechanical changes of the auricle and that waves are more numerous during fine fibrillation.

PLATE 6.

FIG. 3. Jugular pulse, auricular myogram, and intra-auricular pressure showing the traction effect of the ventricle on the auricle and venous pulse (p. 27).

FIG. 4. The same as Fig. 3 (p. 28).

PLATE 7.

FIG. 5. Four segments of a record showing the movements of the superior vena cava (upper curve), a right auricular myogram (middle curve), and an electrogram of the right auricle (lower curve). Time 0.05 second.

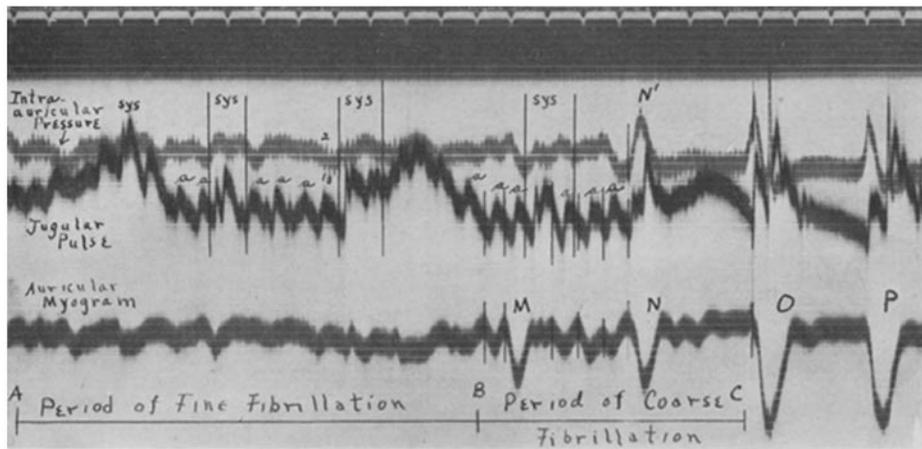


FIG. 1.

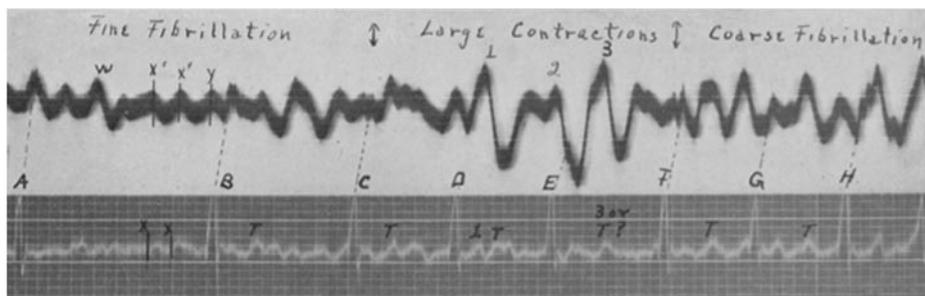


FIG. 2.

(Wiggers and Niles: Diastolic Waves of the Venous Pulse.)

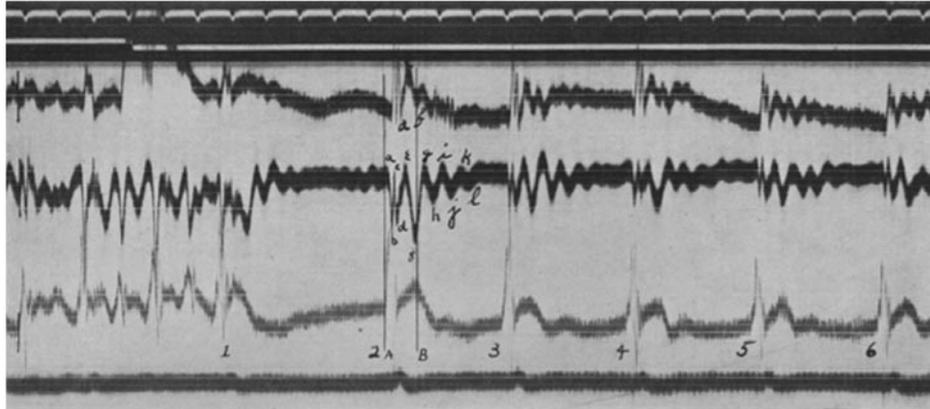


FIG. 3.

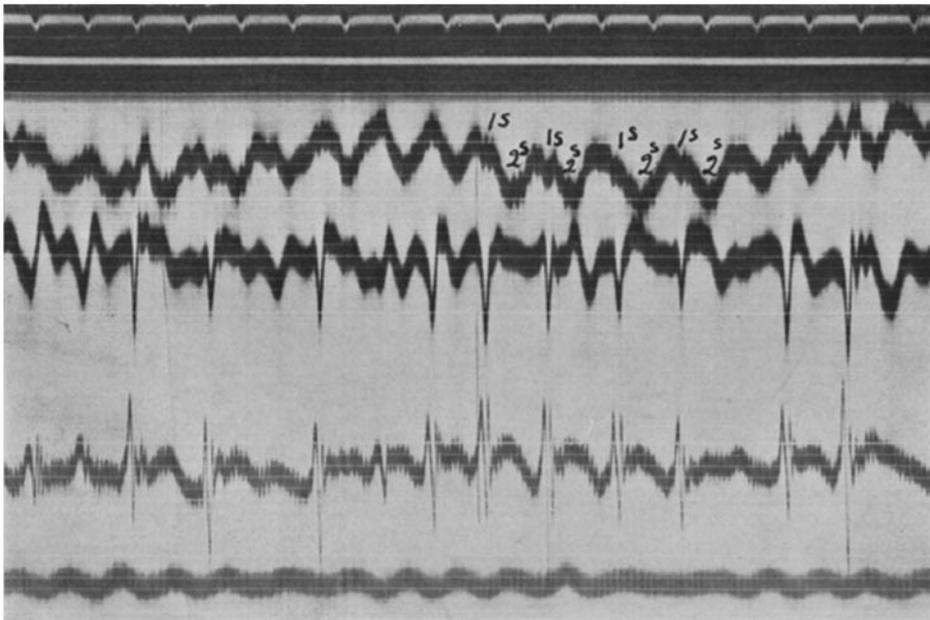


FIG. 4.

(Wiggers and Niles: Diastolic Waves of the Venous Pulse.)

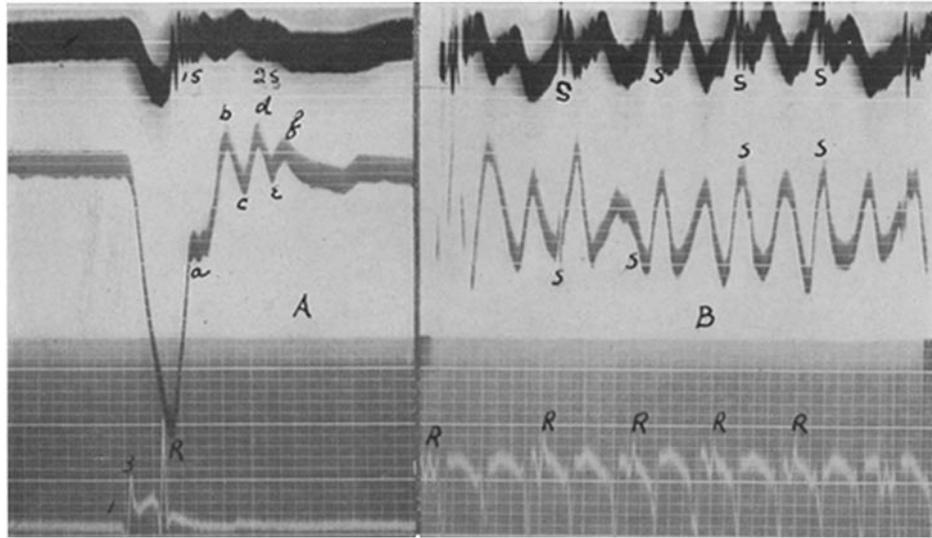


FIG. 5 a.

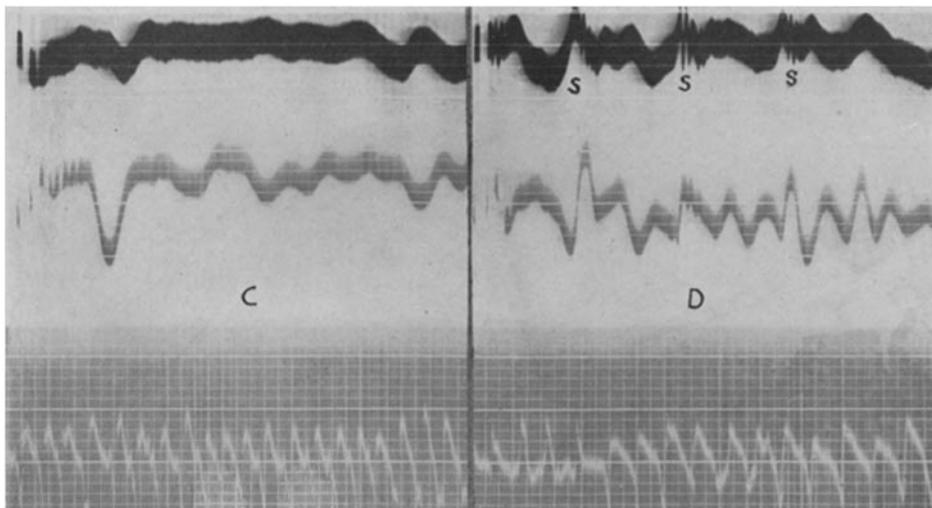


FIG. 5 b.

(Wiggers and Niles: Diastolic Waves of the Venous Pulse.)