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FACTORS DETERMINING THE RELATIVE INTENSITY OF THE HEART SOUNDS IN DIFFERENT AUSCULTATION AREAS

AN EXPERIMENTAL STUDY *

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I. INTRODUCTION

It is commonly accepted as a fact that the absolute and relative intensities of the two heart sounds not only offer valuable information concerning the dynamic state of the heart muscle, but also serve as a criterion of the pressure conditions in the greater and lesser circuits. This conception, based on plausible physical assumptions and fortified by clinical experience, has, however, received only cursory attention from experimental investigators.

Lewis,[1] in his experimental studies of the recorded heart sounds, tested the effect of raising the arterial pressure by aortic compression, but was unable to corroborate one general idea, viz., that high arterial pressure, per se, is responsible for an accentuation of the aortic second sound. On the other hand, the experiments of Dean and myself[2] indicated that the intensity of the sounds directly recorded from the heart is determined by the dynamic conditions of the circulation. Thus, we found that the vibrations comprising both the first and second sounds increased, not only in amplitude, but also in number of vibrations, when the ventricles increased in vigor and the blood pressure was simultaneously elevated either by the injection of epinephrin, by aortic compression or by asphyxia. These experiments did not determine, of course, whether the increased ventricular activity or the simultaneous increase in arterial pressure was fundamentally responsible for the accentuated sounds; nor was the question of changes in the relative intensity of sounds over different cardiac areas investigated. With such questions, this investigation is concerned.

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* From the Physiology Laboratory of Western Reserve University, School of Medicine, Cleveland.

1. Lewis: Quart. J. Med. **4**:241, 1913.

2. Wiggers, C. J., and Dean, Jr., A. L.: Am. J. Physiol. **42**:476 (Feb.) 1917.

II. METHODS OF INVESTIGATION AND APPARATUS

It is possible, by experimental methods, to modify the circulation so that changes in blood pressure are directly due to or occur independent of cardiac activity; it is possible, also, to modify the circulation so that the pressures in the pulmonary and systemic circuits alter in reverse directions. When such changes are produced within short intervals of time, the changes in intensity of sounds over different cardiac areas can be determined by stethoscopic auscultation or recorded by instrumental means.

To accomplish the latter end, two or three receivers, one each over the apex region, the pulmonary area and the aortic area were snugly applied by an elastic band to the shaved thorax of a dog and held in close apposition by anointing the spot with petrolatum. The sound vibrations were then transmitted by rubber tubing to the sound recording capsules described by Dean and myself,[3] or, in some cases, where a third sound was also recorded, to a microphone connected through an induction coil with a string galvanometer. In all of these experiments, the technical procedures carefully explained in my previous communications with Dean[3] were followed.

III. CRITERIA FOR DETERMINING VARIATIONS IN INTENSITY OF RECORDED HEART SOUNDS

The intensity of tones appreciated by the human ear is directly proportional to the force imparted to the tympanum and is therefore dependent upon the amplitude of vibrations, not only, but upon their frequency as well, factors which may be expressed by the formula $Ia = (NA)^2$. It has, therefore, been proposed by Einthoven[4] that the relative intensity of the heart sounds may be computed according to this formula from photographic records and expressed numerically. This was at first attempted in connection with this investigation, but later given up as unfeasible on the following grounds:

1. The heart sounds, as shown most clearly in Einthoven's laboratory by Battaerd,[5] are composed of vibrations which are exceedingly irregular, both as regards their amplitude and period so that no average amplitude or period can be assigned to the sounds.

2. Owing to a loss of energy in transmission of sound waves to the chest wall, the vibrations originating within the heart reach the thoracic wall much reduced in their amplitude. Of these, a variable number have a vibration frequency which is subminimal, in the sense that they

3. Wiggers, C. J., and Dean, Jr., A. L.: Am. J. M. Sc. **153**:666 (May) 1917; also Arch. Int. Med. **22**:28, 1918.

4. Einthoven: Arch. f. d. gesam. Physiol. **120**:31, 1907.

5. Battaerd: Heart **6**:121, 1915.

do not affect the recording membrane at all. When the amplitude of all vibrations increases, the graphically recorded vibrations become greater in number and, consequently, waves with still different vibration periods are added to the sound complexes. If these are considered, the average frequency of the vibrations — composing particularly the first sound — changes in such a way that the suggested computation has no correct relative value in estimating the objective intensity of a sound.

A rougher, but nevertheless a better estimate of the intensity of the sounds and one which more nearly agrees with the auscultatory findings, can be made by comparing the general picture presented by the vibration groups composing each sound. Thus, if the intensity of the sound increases, the amplitude of the whole wave group increases and the total number of vibrations which enter into the sound component becomes greater. If, on the contrary, the intensity of a sound is reduced, not only is the general amplitude of the sound vibrations much smaller, but the number of vibrations entering into its complex is reduced. This, of course, necessitates that a control record of the sounds be taken previous to the time that the circulation is modified by experimental procedures. Inasmuch as the heart sounds of the dog vary greatly in their intensity with the phases of respiration, it is furthermore necessary to make all comparisons during the same respiratory phase. Since the respirations alter their rate and depth, however, during the experimental procedures employed, it is necessary to make all comparisons during the time of expiratory rest.

IV. EXPERIMENTAL PROCEDURE

Animals which were kept evenly anesthetized by morphin and chloretone were placed on their backs. In the earlier experiments records of the vibrations from the right and left second interspaces and from the apex region were simultaneously taken. Inasmuch as it was found, however, that the directional variation of the first and second sound corresponded in the apex region and over the right second interspace, the experiments were simplified by taking only records from the pulmonic and apex regions in the later experiments. In order to preclude possible instrumental irregularities, the additional precaution was taken in the earlier experiments of connecting each area, in turn, with every sound recording capsule used in registration.

As soon as possible after a control of the normal sounds had been recorded, nerves were stimulated or drugs injected, the resulting effect being controlled in a rough manner by a continuous record of the mean carotid pressure recorded on a revolving kymograph. At sufficient intervals, i. e., usually during the onset of an effect, during

its maximum action, during its decline and after normal conditions had been restored, the sound vibrations were again recorded on the photokymograph. The time that these photographic sound records was taken was also indicated underneath the blood pressure on the kymograph through an automatic electrical signal arrangement.

V. EXPERIMENTAL RESULTS AND DISCUSSION

The results now to be analyzed were found to agree in all the separate trials carried out on fourteen different dogs.

1. *The Influence of Heart Rate Changes on the Intensity of the Heart Sounds.* — Inasmuch as many experimental procedures are attended by a change in heart rate, and inasmuch as the rate of the heart varies considerably in auscultated subjects, it was first necessary

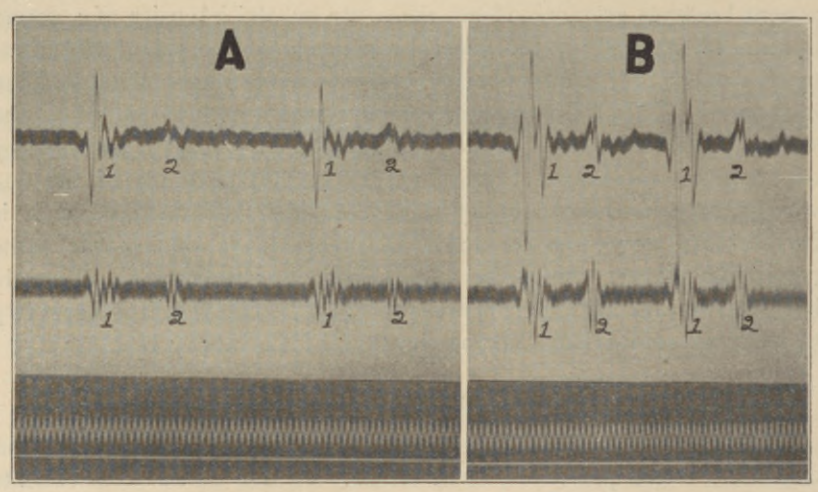

Fig. 1.—(Two-thirds actual size.) Two segments of records showing the change in amplitude of recorded sounds during cardiac slowing, A, during vagus slowing and B, after return to normal rate. Upper record, apex sounds; middle record, pulmonic sounds; lower record, time in 0.02 sec.—1, 2, first and second sound complexes (Experiment C, 210, V).

to study the effect that such primary changes in rhythm produce in the intensity of the heart sounds recorded from different thoracic areas.

In these experiments slowing was induced by mild stimulation of the peripheral end of the vagus nerve; acceleration, either by vagus section or by stimulation of the accelerator nerves.

(a) *The effect of cardiac slowing produced by moderate stimulation of the peripheral end of the vagus nerve.*—The typical effects of mildly stimulating the peripheral end of the vagus nerve are illustrated in the two segments shown in Figure 1. As indicated by the

amplitude and number of vibrations, the intensity both of the first and second sound decreases appreciably over both the pulmonary area and the apex when the heart rate slows.

(b) *The effects of cardiac acceleration produced by vagus section or accelerator stimulation.*—Cardiac acceleration appears to have a variable effect on the intensity of the heart sounds. The first sound is chiefly affected. At times no alteration is apparent, at times, again, an increased amplitude is observed or even a slight decrease may result. Even if allowances are made for the possible shifting of position of the receiving tambours, the results in any given experiment cannot readily be accounted for on experimental grounds.

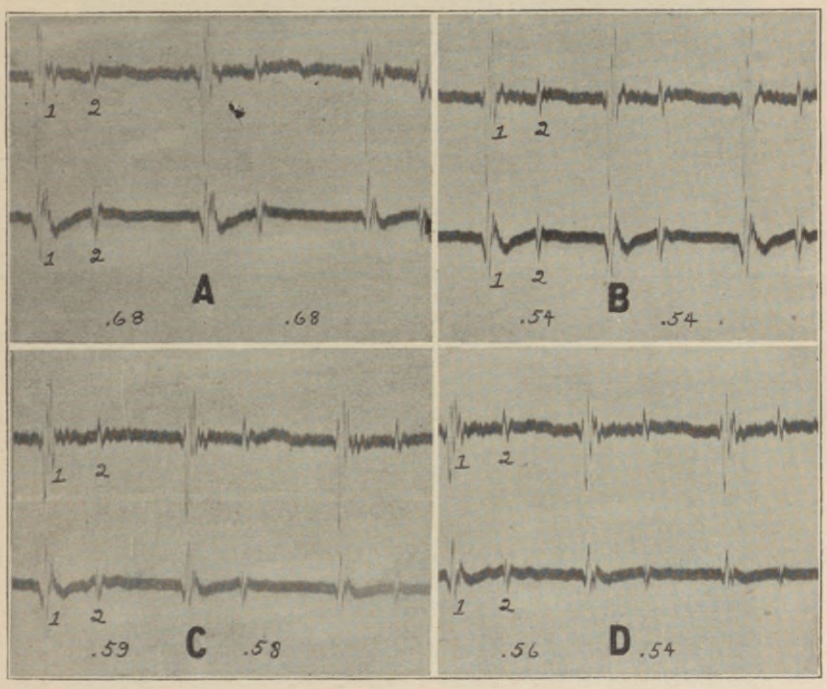

Fig. 2.—(One-half actual size.) Four segments of heart sound records showing effect of cardiac acceleration. Upper record, apex sounds; lower record, pulmonic sounds. Figures .68, .54, etc., marked on curve give duration of heart cycles. Detailed description in text. (Experiment C, 207, III).

In Figure 2, four segments from such an experiment are shown. The first segment, A, is a normal control. The segment, B, shows the sounds after acceleration was produced by section of both vagus nerves. Both sounds at the apex as well as over the pulmonic area are accentuated, as shown both by the larger amplitude and number of vibrations. At the time that the record, C, was taken, the heart had slowed to a slight extent. The first sound is reduced in amplitude in

both curves, the second sound, especially in the pulmonic tracing. Record D was taken during further acceleration of the heart induced by stimulation of the stellate ganglion. All sounds are somewhat reduced over both the apex and pulmonic area.

(c) *Discussion of Results.* — It is common knowledge that when the vagus nerve is stimulated and the ventricles are thus slowed, a fall of the mean systemic and pulmonary arterial pressure results, due to a decrease in the minute volume discharged by the heart. The experiment shows that this fall of pressure, even if moderate, is accompanied by a slight enfeeblement of the second sound; whereas, if the

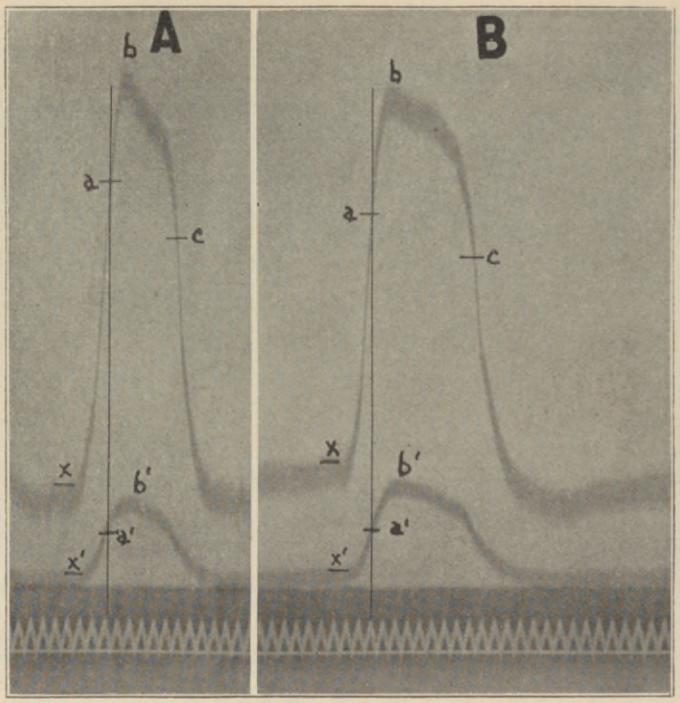

Fig. 3.—(One-half actual size.) Two segments of records showing, left (upper) and right (middle) intraventricular pressure curves. A, normal curves; B, during vagal slowing. Lower curve, tuning fork—50 v.d. Detailed analysis in text. (Experiment C, 216).

pressure drops to any extent, the sounds, both first and second, are diminished in all regions.

Myocardiographic tracings of the ventricles indicate that during vagus stimulation the individual beats are more vigorous and volume curves of both ventricles indicate that the aggregate systolic discharge of the two ventricles increases. We are thus confronted with the

fact that the stronger ventricular beats are accompanied by first sounds of diminished intensity, and we might infer therefrom that the intensity of the first sound is no index of ventricular efficiency.

This unexpected and apparently paradoxical relation led me to reinvestigate the effects of vagal slowing on the right and left intraventricular pressure curves. From such records, it appears that the dynamic effects of continued vagal stimulation are not alike in the right and left ventricles, a fact to which attention has heretofore apparently never been attracted. In Figure 3 are shown two segments of the right and left intraventricular pressures before and during vagus stimulation. The normal pressure curves in the two ventricles may first be considered (A). Owing to the fact that the a-v valves close early in systole, and that the semilunar valves remain closed for an additional short period, each ventricle during its early phase of systole is not able to decrease in volume nor to shorten its fibers; hence the energy of contraction is utilized to develop intraventricular tension. This mode of contraction is spoken of as isometric and consequently

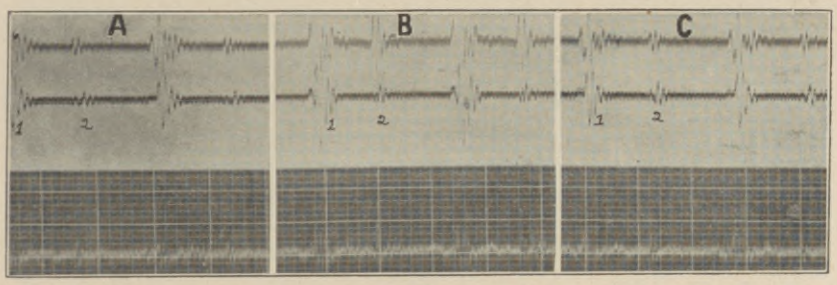

Fig. 4.—(Two-fifths actual size.) Three segments of sound records showing the amplitude of sounds. A, before; B, during central vagus stimulation; C, after return of arterial pressure to normal. Upper record, apex sounds; middle record, pulmonary sounds; lower record (taken by Einthoven's phonocardiographic method), aortic sounds. Abscissae = 0.04 sec. (Experiment C 143, IV).

the interval elapsing before blood is expelled is aptly and concisely referred to as the *isometric period* of systole. This interval extends to the point marked *a* in Figure 3. It is quite obvious upon reflection that the height to which the isometric pressure rises (point a) is determined by the height of the aortic pressure at this time, i. e., the higher the aortic pressure at the beginning of systole, the higher the isometric pressure becomes.

If we compare the right and left intraventricular pressure curves before and during vagus slowing, it is evident that, in the case of the right heart the height of isometric pressure, *a'*, is not greatly altered, while the maximum pressure developed during ejection, *b'*, is dis-

tinctly higher. In the left ventricle, on the other hand, such an effect is very transient, lasting for only one or two beats. After this, as shown in segment B of Figure 3, the height of the isometric pressure, *a*, decreases greatly owing to a falling arterial pressure. The maximum pressure reached during ejection, *b*, is also necessarily somewhat lower. Thus we have a reduction of pressure developed within the left ventricle at the same time that the systolic discharge is distinctly increased.

The reduction in the intensity of the first sounds makes it apparent that the loudness of the first sound is related, not so much to the efficiency of the ventricle as a pumping mechanism as it is to the intracardiac tension developed; and especially to the tension developed before the semilunar valves open. This accords entirely with the observations previously made by Dean and myself,[2] viz., that the vibrations of the first sound reach their maximum intensity before the ejection period has begun, i. e., during the isometric interval.

Cardiac acceleration produced either through double vagotomy or by stimulation of the accelerator nerve, should produce precisely

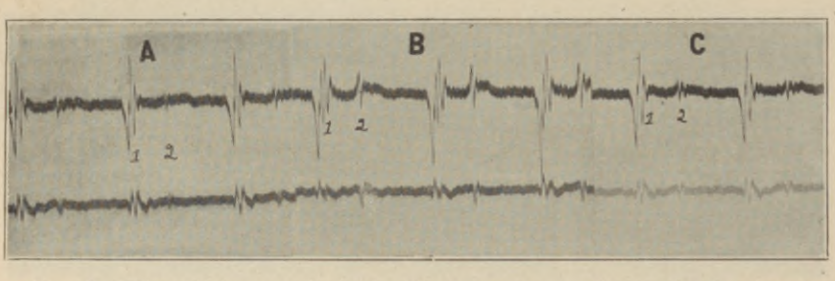

Fig. 5.—(Two-fifths actual size.) Three segments of records showing the amplitude of sounds. A, before; B, during aortic compression; C, after release. Upper curve, apex sounds; lower curve, pulmonic sounds. (C 207, XIII.)

the opposite effect. This result, as shown in the record B of Figure 2, was generally found to be the case. It also harmonizes with the general clinical observation that the first sound is louder in individuals with rapid heart rates. Experimentally, there are, however, distinct exceptions, as shown specifically in the segments C and D of this figure. The cause of this is easily explained. A careful comparison of heart sound records with the simultaneously recorded blood pressure tracings shows without exception that whenever acceleration produces a fair elevation of arterial pressure and an increase in the height of isometric pressure, then the first aortic and apex sounds are also increased. Whenever, through compensatory mechanisms, e. g., reflex dilation, no distinct elevation of arterial pressure occurs and the isometric pressure does not rise above normal, then the first sounds do not alter, or, as is shown in this record, they may even

decrease slightly. These observations establish the fact that changes in heart rate can affect the intensity of the heart sounds only through the attendant dynamic disturbances incurred thereby.

Inasmuch as changes in heart rate are themselves capable of modifying the intensity of the heart sounds, it is obviously necessary to eliminate them, or, at least to take them into consideration in order to evaluate the effect that other circulatory conditions have on the intensity of the sounds. Where heart rate changes occur secondarily through nervous reflexes it is quite possible to eliminate them through the severance of the vagus nerves or paralysis of their terminals by atropin. Certain procedures, however, modify the heart rate through a direct action which cannot be prevented. The foregoing results, viz., that changes in intensity accompanying changes of heart rate are not determined by changes in rate per se, but secondarily by pressure changes induced, are of fundamental help in such instances.

2. *The influence of changes in the total arterial resistance in the intensity of the heart sounds.*—If, as seems probable from previ-

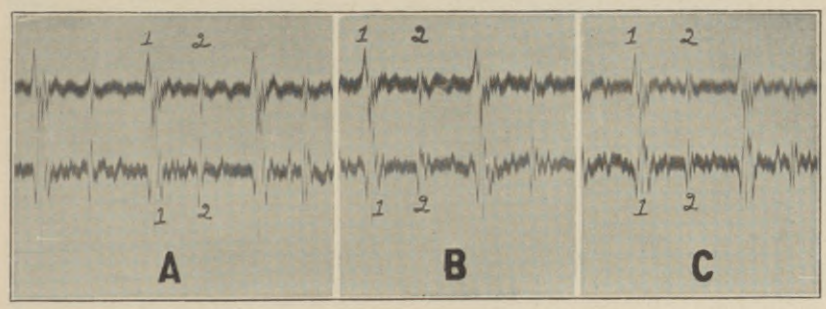

Fig. 6.—(One-half actual size.) Three segments of records showing the relative amplitude of sounds. A, before; B and C, during two stages of pressure decline following injection of sodium nitrite. Upper curve, apex sounds; lower curve, pulmonary sounds (Experiment C 208, X).

ous analysis, the intensity of the heart sounds is fundamentally due to changes in the arterial pressures, and the consequent changes in the height to which intraventricular pressure is elevated during the isometric period of systole, it should be possible to produce such changes without alteration in the heart rate by varying the total resistance against which blood must be pumped. This may be experimentally accomplished in various ways of which the following was utilized in this investigation, viz.: (a) By stimulation of the central vagus which causes a reflex general vasoconstriction. This increases the arterial pressure and, consequently, the height of the left ventricular pressure during the isometric period; (b) by compression of the aorta, which acts similarly, and (c) by the injection of nitrites, which causes a

reduction in arterial resistance, a consequent fall of arterial pressures and a lowering of left ventricular pressure during the isometric interval.

Such systemic changes are not without a secondary effect on the pulmonary and right ventricular pressures. When the total arterial resistance is raised within the arterial system, the left ventricle acts less efficiently as a pumping mechanism and a slight damming back of blood into the pulmonary system occurs. This acts to increase the total resistance in the pulmonary circuit, causes the pulmonary arterial pressure to rise somewhat and increase the pressure in the right ventricle at the end of the isometric period.

(a) *The influence of increasing arteriole resistance by stimulating the central end of the vagus nerve.*—In Figure 4 are shown records before and during the elevation of arterial pressure in this manner. During the elevation of arterial pressure (B) the number and amplitude of the vibrations comprising the first sound increase definitely in all the auscultation areas, indicating an accentuation. In the great

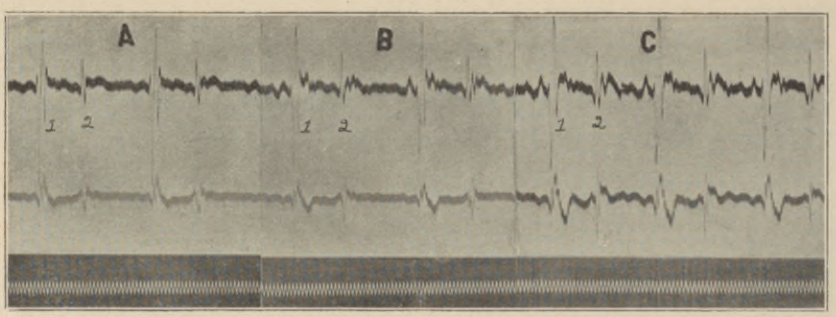

Fig. 7.—(Two-fifths actual size.) Three records of heart sounds showing amplitude of vibrations. A, before; B, during, and C, immediately following rapid saline infusion. The effect started in B continues in C. Upper curve, apex sounds; middle curve, pulmonic sounds; lower curve, tuning fork 0.02 sec. (Experiment C 207, X).

majority of cases, the second sound is also accentuated over the aortic and apex regions, as is well shown in Figure 4. The second pulmonic, though definitely accentuated, increases to a lesser degree.

The relative extent to which the two sounds are accentuated varies greatly in different regions. Over the pulmonary area the first sound is, as a rule, affected most, while over the aortic area, the second sound is predominantly affected. Five minutes after cessation of stimulation (C), the sounds return to normal although the second pulmonic often remains somewhat accentuated.

(b) *The effect of aortic compression on the intensity of heart sounds.*—From a priori considerations we would expect precisely the

same changes in the amplitude of the two sounds as follows central vagus stimulation. As shown in the typical records of Figure 5, this is precisely the case and contrary to the findings of Lewis.[1] The increase in the amplitude of the first as well as the second apical sound is very evident on comparing the second segment (B) with A and C, which are the normal controls before and after compression. While an accentuation of both sounds accompanies a mechanical elevation of arterial pressure, it is quite obvious that the second sound at the apex is accentuated relatively more than the first. In the pulmonic area, the changes in the sounds are not as marked. This is especially true of the first sound, the pulmonary second being somewhat intensified.

(c) *The influence of peripheral dilatation produced by sodium nitrite.* — When sodium nitrite is administered intravenously to an

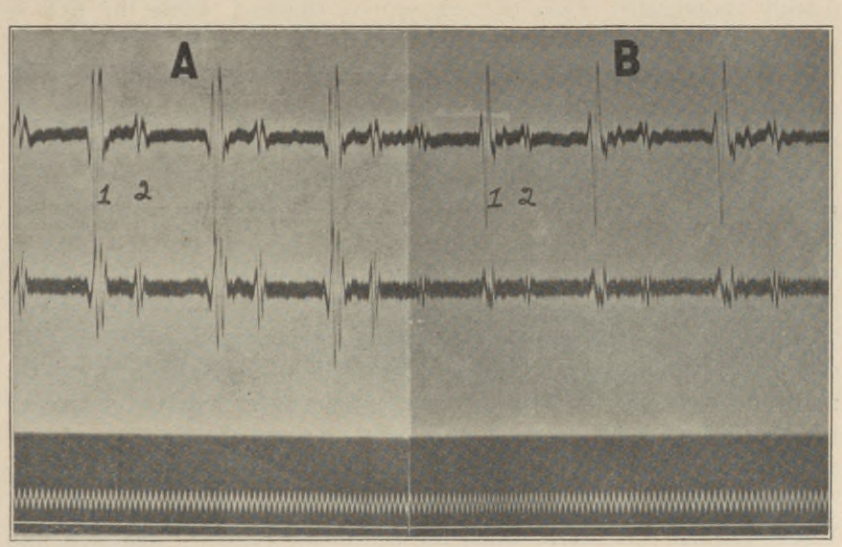

Fig. 8.—(Two-thirds actual size.) Two records of sounds. A, before; B, during compression of the inferior vena cava. Upper record, apex sounds; middle record, pulmonic sounds; lower record, tuning fork vibrations, 0.02 sec. (Experiment C 210, IX).

animal whose vagi nerves have been previously divided, the amplitude of the first and second apex sounds decreases, indicating a reduction in their intensity. A slight, but by no means marked, decrease is also observed in the pulmonary sounds. These changes are well shown in the three segments of Figure 6. Comparison of the relative changes in intensity of the two sounds shows that the enfeeblement of the first sound is greater than that of the second sound.

(d) *Discussion of results.*—These observations confirm the previous conclusion that the intensity of the first sound is related definitely

to the height of the arterial pressure within the pulmonary or systemic circuit and is determined by the height to which the pressure is elevated during the isometric period.

Owing to the fact that the systemic pressure variations are much more extensive in these experiments, they demonstrate much more clearly the relation between the intensity of the second sounds and the height of the arterial pressure. Whenever arterial pressure is raised the second sound augments, and whenever this produces a back effect on the pulmonary circuit, the pulmonary second sound also increases.

It is quite obvious on careful analysis, that the first and second sounds do not always intensify to an equal degree when arterial pressure is raised or lowered through peripheral causes. In the systemic circuit, at least, the rule seems to be that the second sound is predominantly accentuated when the pressure is elevated, while the first is predominantly enfeebled when the pressure is lowered. No explanation entirely satisfactory suggests itself. It is well to bear in mind in this connection that the tension developed during the isometric period, and, consequently, the intensity of the first sound, is determined by the pressure existing at the time that the semilunar valves open (at a, Figure 3). On the other hand, the closure of the semilunar valves is determined by the pressure existing at the very beginning of diastole, e. g., at c, Figure 3. It is quite apparent on reflection that the relative changes of pressure at these two periods may vary independently and to an unequal degree, depending on the particular set of dynamic conditions which operate. Additional work of a very fundamental nature must first be done, however, before a final and satisfactory answer may be given to the question as to why at times the first sound and at times the second sound seems to be affected predominantly.

3. *Influences affecting the systolic discharge of the ventricle.*— Under normal conditions, when the heart rate remains unaltered, the systolic discharge of each ventricle is determined by the volume of blood returning to it during diastole. This also determines the initial pressure (Figure 3, x) which exists within the ventricle at the time it begins to contract, and upon this, in a measure, depends the systolic tension developed within the ventricle itself. By increasing and decreasing the volume of blood returned to the right auricle, it is possible to at once influence the vigor of the right ventricular contraction, and, after a beat or two, also the vigor of the left ventricular contraction.

Such a modification of the venous return may be experimentally produced by the two opposite procedures of (a) rapidly infusing saline solutions into the jugular vein, and (b) clamping the inferior vena cava.

(a) *The influence of rapid saline infusion on the heart sounds.*—Typical sound records of the apex and pulmonary areas before, during and after a rapid saline infusion into the jugular vein are shown in Figure 7, A, B and C, respectively. A comparison of the first sounds in segments A and B indicates that the first sounds are not increased during the course of the infusion when the right heart becomes extremely distended; in fact, the pulmonic first sound may become decreased in amplitude. This is possibly accounted for by the cardiac slowing which is evident. While the mean arterial pressure elevates during infusion, the arterial pressure at the beginning of ventricular ejection is not changed. The reduction of the pulmonary second sound is further influenced by the predominant dilation of the right heart which occurs at the same time.

After injection had ceased, when the heart had recovered its normal rate and dilation was less marked, segment C was recorded. The first

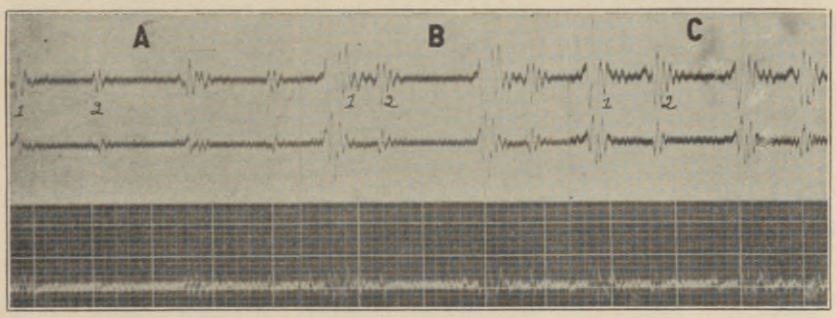

Fig. 9.—(Two-fifths actual size.) Three records showing the relative amplitude of sounds. A, before; B and C, during action of epinephrin. Upper record, apex sounds; middle record, pulmonic sounds, lower record, aortic sounds by Einthoven's phono-cardiograph. Time, abscissae = 0.04 sec. (Experiment C 143, VII).

sound now, true to our anticipation, increases at the apex, and even to a greater extent over the pulmonary area.

The second apical sound, as also the second aortic, increased appreciably in amplitude and number of vibrations as the pressure continued to rise during the infusion. Especially striking, however, is the amplification of the second pulmonic sound. It is noteworthy, also, that during and following such an infusion both pulmonic sounds are affected much more than the aortic or apical sounds.

(b) *The influence on the heart sounds of compressing the inferior vena cava.*—The changes of the heart sounds, before and during compression of the inferior vena cava, are illustrated in Figure 8. While the first and second sounds are both slightly reduced over the apex—

the marked reduction occurs in both pulmonic sounds. These and the preceding experiments where predominant changes in the right ventricle and lesser circulation are evidenced by the greatest variation in the pulmonic sounds are fundamentally important in that they give definite experimental proof (1) that the sounds heard over the left second interspace are really propagated from the pulmonic circuit and right heart, and (2) that valuable information regarding the dynamic conditions of the pulmonary circuits may be gained by comparing from time to time the relative intensities of the heart sounds over different areas.

4. *Influences simultaneously affecting the systolic discharge and total arterial resistance.*—A combination of the cardiac and peripheral factors before studied separately may be induced by intravenous injection of epinephrin into an animal whose vagi nerves have been cut or the terminals of which have been paralyzed by atropin.

(a) *Influence of epinephrin on the heart sounds.*—The changes in the heart following changes in the circulation after injection of epinephrin are very constant. Typical tracings are shown in Figure 9. It is quite evident that the amplitude of the first sound increases to a pronounced degree in all areas. The second sounds are also intensified, but not equally in all areas; the accentuation of the second aortic and of the second apex sound being greater than that of the second pulmonic.

(b) *Discussion.*—Owing to a stimulating effect on the ventricular mechanism, epinephrin causes a greater discharge from each ventricle. This, together with the cardiac acceleration (when the vagi are cut) causes an elevation of the pulmonary arterial pressure and consequently of the right ventricular pressure during the isometric and ejection phases not only, but also produces a higher pressure at the beginning of diastole, i. e., when the semilunar valves close. This accounts for the accentuation of both pulmonic sounds.

In the systemic circuit it causes, in addition, peripheral constriction, which, together with increasing cardiac discharge, causes an enormous elevation of systemic pressure at the end of systole. It may therefore be anticipated that the intensity of the second apex or aortic sound will be very much more augmented than that of the second pulmonic sound. This, as has before been pointed out, proves to be the case.

5. *Influences modifying pulmonary and arterial pressures in opposite directions.*—Finally, it is of great interest to study the relative intensities of sounds from different areas when the pressures in one circuit rise and in the other fall. This may be accomplished by the

use of pituitary extract which has been shown to effect a great rise of systemic pressure and a decline of pulmonary pressure (Wiggers[6]).

In interpreting the changes of the heart sounds during this condition, it is necessary, however, to analyze more precisely the dynamic changes produced in the greater and lesser circulation in more detail.

(a) *The influence of pituitary extract on the greater and lesser circuits.*—Studies of the mean arterial pressures in the pulmonary and systemic circuits,[6] as well as the pressure curves in the right ventricle,[7] have led the writer to insist that the primary effect of pituitary extract on the heart muscle is a depressant one. It causes a decreased systolic discharge which, together with a slower rate, brings about a reduction in pulmonary arterial pressure. The effect on the right ventricular pressure is shown in segments A and B of Figure 10. The pressure

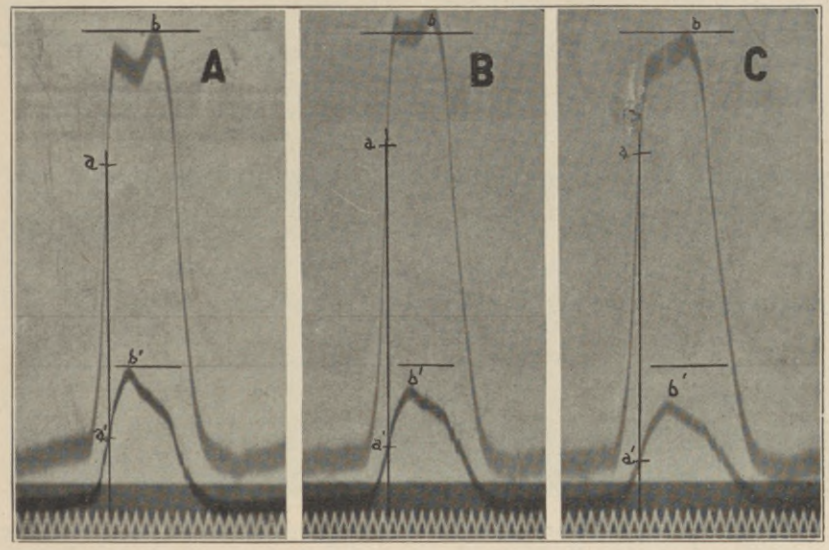

Fig. 10.—(One-half actual size.) Three segments of records showing left (upper curve) and right (middle curve) intraventricular pressure. A, before; B and C, during the stage of pituitrin action. Lower record = tuning fork vibration 0.02 sec. Detailed description in text. (Experiment C 206, XIII.)

during the end of the isometric period, as well as at the height of ejection, is evidently much lower. This is shown in the three segments of Figure 10 a′-b′). The effect on the left ventricle pressure is quite different. Owing to an intensive action on the musculature of the peripheral arterioles, the systemic pressure at once begins to rise. The left ventricle, thereby contracting against a progressively increasing

6. Wiggers, C. J.: Arch. Int. Med. **8**:30 (July) 1911.
7. Wiggers, C. J.: Am. J. Physiol. **33**:394 (Aug.) 1914.

tension, is forced to develop a considerably higher pressure during the isometric phase not only, but during the ejection period as well. This is shown on the three segments of Figure 10, at a and b, respectively.

The total effect of this extract therefore is to reduce the level to which the isometric and ejection pressures are raised in the right ventricle and to increase these levels in the left ventricle.

A study of the relative intensity of the heart sounds over different areas forms almost an experimentum crucis of our former conclusion, for if the intensity of the first sound depends primarily on the height of pressure during the isometric period of systole, and that of the second sound on the height of pressure at the beginning of diastole, then we should find that the intensity of the two pulmonary sounds, on

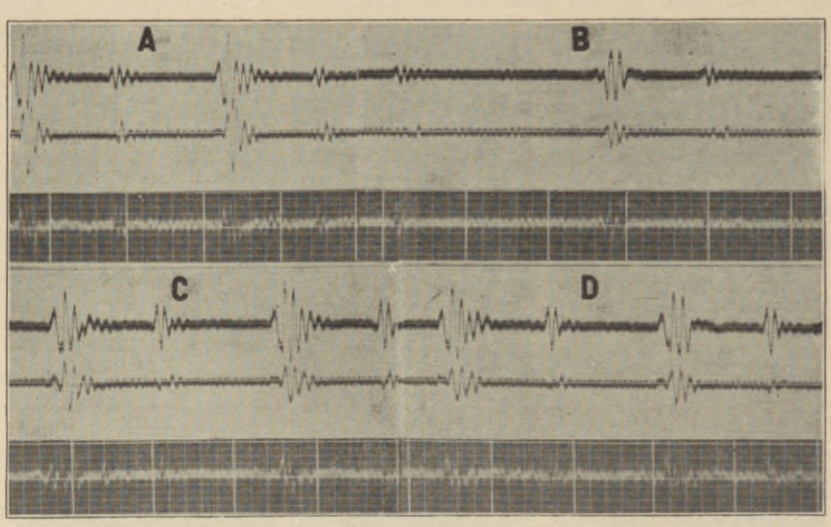

Fig. 11.—(Two-fifths actual size.) Four segments showing the amplitude of sounds. A, before; B, during slowing and first pressure increase following pituitary extract. C and D, subsequently—after marked elevation of pressure and return of cardiac rate to normal. Upper curve, apex sounds; middle curve, pulmonic sounds, lower curve, apex sounds. Time, abscissae, 0.04 sec. Detailed explanation in text. (Experiment C 143, V.)

the one hand, and the apex and aortic sounds on the other, would change in opposite directions during the action of pituitary extract.

(b) *The relative intensities of the heart sounds during the action of pituitary extract.*—Typical effects of pituitary extract on the two heart sounds are shown in Figure 11.

The changes in the first sounds may be considered first. During the rise of pressure (cf. segments A and B) the first sound decreases in all regions. This is, no doubt, due to a lowered isometric pressure

largely occasioned by the great slowing of the heart which complicates the action. The changes are similar to those produced by vagus slowing. As the arterial pressure rises to a maximum 1.2 while the isometric pressure elevates in the left ventricle and falls in the right (curves C and D), the first apical and aortic sounds increase in intensity while the second pulmonic sound decreases in intensity.

Comparison of the normal systemic and pulmonary sounds (segment A) with those occurring during the first cardiac slowing (B) shows that the second pulmonic sound is greatly reduced in intensity, and, in fact, may entirely disappear in some beats, while the second aortic sound is less affected. As the heart accelerates and the maximum pressure is reached within the systemic arteries (segments C and D) the second aortic and second apical sounds are markedly increased in intensity, agreeing with the previous dynamic analysis. The second pulmonic sound, on the other hand, continues to be of smaller amplitude as we should expect from the reduced pulmonary pressure.

VI. SUMMARY AND CONCLUSIONS

1. The heart sounds over the apex region, aortic and pulmonary areas were recorded simultaneously or in pairs by the direct sound recording capsules described by Dean and myself. Changes in the intensity of any heart sound over a particular area were determined by comparing the amplitude and number of vibrations entering into that sound complex before and after modified conditions of the circulation were experimentally produced.

2. The intensity of the first sound is not related to the volume of blood discharged by the ventricles, e. g., during slowing of the heart; when the systolic discharge is increased, the first sound is reduced in intensity.

3. The intensity of the first sound over all regions varies directly as the systolic tension developed within the ventricles — and there is good reason to believe, with the tension developed during the isometric period of systole.

4. The intensity of the second sound increases or decreases as the pressure (aortic or pulmonary) at the beginning of diastole.

5. When the pressures at the beginning and end of systole increase, particularly in one circuit and relatively little in the other, an accentuation occurs, predominantly in the sounds referred to the circuit in which the changes predominate.

6. In circulatory conditions where the pressures at the beginning and end of systole vary in opposite directions in the greater and lesser circuits, the intensity of the pulmonary and aortic sounds changes in opposite directions.

7. Direct experimental evidence supplements the anatomical basis for believing that sounds heard over the second left i. c. s. are transmitted from the right heart and lesser circuit, while those heard over the right second i. c. s. and apex have their origin in the left heart and greater circulation.

8. When reserve and caution are exercised, a change in the intensity of the first sound over any area is good evidence of a change in tension developed during systole of the ventricles, while a change in the intensity of the second sound over the aortic and pulmonary areas may safely be used as an index of a change of pressure at the beginning of diastole in the greater and lesser circuits, respectively.

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