REMARKS

ON

THE HEART-INDEX INTERVAL IN AORTIC
REGURGITATION.By PAUL M. CHAPMAN, M.D.LOND., F.R.C.P.,
Physician to the Herefordshire General Hospital.

IN July, 1898, I published an article in which I suggested that a measure of the amount of compensation in aortic regurgitation might be found in the measure of heart-radial delay, which I had noticed was modified in direct relation to the improvement in the general condition of the patient. Owing to an error in the tracings I then published, which I only discovered when the article had gone to press, I hastened to withdraw the matter at once from discussion.

It is useless to publish any convictions one may have on the subject of pulse delay without furnishing proof to others by means of some evidence which can admit of no manner of doubt. Such a proof is most difficult to obtain. From the most characteristic case a good tracing cannot always be got. To be convincing, the cardiographic record should clearly show the aortic systole, as well as the whole course of ventricular contraction. To obtain such a tracing is in itself somewhat of an art. If to this is added the difficulty of getting on the same drum a good and simultaneous pulse tracing, together with a chronographic record of the whole, the pulse tracing to be of such a kind as to be capable of giving evidence of the blood pressure at the time of the experiment; I must offer these difficulties as an excuse for waiting two years before returning to the subject with confidence.

Even now, though (1) I hold in my hand the proof of pulse delay and of its modification in aortic regurgitation; and though (2) I consider that I am entitled to believe that the explanation I have to give of the method of production of pulse delay, when met with in these cases, is true, and perhaps complete, I cannot offer any very striking proof of what I believe to be true, namely, that (3) the duration of ventricular systole is lengthened in fully compensated cases of aortic regurgitation.

But with regard to (1) and (2), there has been so much debate that it is with great pleasure I now publish the tracing which illustrates this paper.

At the outset, I have to thank Messrs. Carl Hentschel, of Fleet Street, for the delightful accuracy with which they have reproduced my tracing. Save for the different appearance of the texture of the paper, the proof I have received from them would be quite indistinguishable from my own tracing, of which it is an absolute facsimile. An examination of it will leave no doubt as to the fact that there was a slight increase in the heart-index interval in the subject from whom the original was taken.

PHYSIOLOGICAL CONSIDERATIONS.

But on theoretical grounds and according to physiological experiments, we should expect to find the heart-radial interval shortened, owing to the disappearance of the "*Anspannungszeit*," or so-called "presphygmie interval" of Keyt; by which we understand the time which is occupied by the ventricle of a normal heart in raising the intraventricular blood pressure from about 20 mm. Hg. at the onset of systole to about 150 or 200 mm. Hg. at the time of opening of the semilunar valves. For only when the intraventricular pressure exceeds that in the root of the aorta is it assumed possible that a wave should appear in the arterial system.

As I hope that the question of the possibility of heart-radial delay in aortic regurgitation may now be settled once for all by the demonstration that it actually does occur in some cases, it would be well to state again the present theory on which the doctrine of heart-radial delay in aortic regurgitation is based. Not omitting to say that the possibility of *anticipation* of pulse in some cases is credible and consonant with physiological reasoning, and also that Keyt and others too have furnished tracings in proof of the contention that the heart-radial interval is shortened in some cases of aortic regurgitation. The discussion is indeed rendered necessary by

the very absolute statements made by both sides. To take Keyt first. He says:

KEYT'S REASONS WHY THE INTERVAL "IS ALWAYS
SHORTENED."

When the aortic valves are permanently open the pressure in the ventricle and aorta are always in equilibrium at the beginning of systole (the two cavities being in one), whether the mean pressure be low or high. In consequence of this oneness or equilibrium of pressure, the heart's impulse and aortic pulse will be synchronous.....the testimony is ample and complete, and establishes the fact beyond question.

Other observers are in agreement with him. I myself (Goulstonian Lectures, 1894¹), having obtained some graphic support of his doctrine, expressed adhesion to it, and "thought it should be accepted." My views are now modified, since I hold the proof that in aortic regurgitation there may be pulse delay.

But the absolute statements on the other side also go too far. For instance, as long ago as 1832, Dr. Henderson stated that the interval between the heart's impulse and the arterial pulse was prolonged in aortic insufficiency. In 1859 Flint says "the fact of prolonged delay is not to be denied." Sir William Broadbent² says "the pulse in aortic regurgitation is always retarded or delayed." I am predisposed in such matters to trust the observations of the great clinicians. But I must now express myself as fully in agreement with another great clinician and careful observer—Walshe—who confines himself to what I hold to be a far more accurate statement than any of these: "The retardation may with care be detected in many, but unquestionably not in all, cases of that disease."

As for the explanation of the delay of pulse in these cases (where all explanation is needed, as the fact of delay is in apparent conflict with what might be expected from physiological reasoning when confined to the consideration of the absence of the *Anspannungszeit*), I think I have a further, and possibly a very useful, one to give, in addition to the other factors which undoubtedly obtain, and which are so well stated by Sir William Broadbent that I give them here in his words:

SIR W. BROADBENT'S REASONS WHY THE INTERVAL "IS
ALWAYS LENGTHENED."

This delay is due partly to the collapsed and empty state of the arteries between the beats, and partly to their large size and loss of tone. There is no longer a continuous column of blood between the heart and the periphery, and the tension in the arterial system has to be considerably augmented by the launching of a large volume of blood into the aorta before the vessels are rendered sufficiently tense for a pulse wave to be transmitted.

When we turn back to François-Franck and find that he states, as the conclusion from many experimental clinical researches by the graphic method³: "In large pure aortic insufficiency the retardation of the pulse on the beginning of the systole of the heart is very notably diminished," we feel that there must be some fact underlying all these conflicting statements which, could it be brought to light, might afford an explanation of these discrepancies. They are opposed views held by men of undoubted acumen and ability. An explanation of the discrepancies would not only be extremely interesting, but might throw light of a most useful kind upon the whole subject, and bear some relation to treatment.

PRESENT EXPERIMENTS.

The tracing reproduced here was obtained on a small recording cylinder. I have used Dr. Augustus Waller's "digital sphygmograph,"⁴ which I have found much more convenient than any other. It is altogether a most admirable invention. It must be pointed out that there is a slight delay between the radial pulse and the digital pulse (obtained from the back of the finger nail). The maximum delay amounts to 0.02". The pendulum chronograph is also Dr. Waller's. As to a cardiograph I always prefer to cover it myself, and to discard the internal spiral spring. It connects with a simple, rather shallow, tambour on a sliding rod, which I obtained from Petzold (Leipzig). The latter I lash firmly to the digital sphygmograph, and attach the combination to an upright, on which it can slide up and down as one piece without disturbing the relative position of the levers. The pendulum chronograph I attach to a separate upright rod. The uprights stand on either side of the revolving drum. The patient usually lies inclining to his left side with his left

A. P., aged 49; simple aortic regurgitation. T, pendulum chronograph beating half seconds; I, sphygmograph from back of index finger nail; H, cardiograph; a, b, synchronous movements of the two levers describing I and H; b—b, heart index interval. Pulse-rate, 75; duration of systole, 0.330"; duration of diastole, 0.446"; heart-index delay, 0.216"; blood pressure of I, 16 mm. Hg.

hand under his cheek. He puts his right hand forwards (extending the right arm slightly) to reach the digital sphygmograph, into which he inserts his index finger, the wrist is supported by appropriate means, so that all is at rest. The operator himself adjusts and steadies the cardiograph with his left hand while standing at the back of the patient. This leaves his right hand free to adjust, start, or break off the parts of the apparatus.⁵

The description given with the tracing still leaves room for some analysis. The patient felt perfectly well until October last. He said he had "had no illness in his life before." The first thing he noticed was that he could not lie down comfortably in bed, as it gave him precordial pain and dyspnoea. He found that these symptoms persisted during the day, and at this time he noticed that his urine was scanty and thick and his bowels confined. The tracing was taken on November 6th, when I found he had a long, purely diastolic *bruit*, and characteristic, but not exaggerated, pulsation in the arteries. He was undoubtedly suffering from pure aortic regurgitation, without complications, a heaving heart impulse could be felt slightly below and outside the nipple, and there was some increased dulness towards the left.

According to my previous experiments⁶ the duration of ventricular systole at a pulse rate of 75 may vary from 0.3033" (after Turkish bath) to a maximum of 0.3300" (after exertion). The average systole is about 0.3100" to 0.3200". The duration of systole in A.P. is therefore at the extreme maximum for a pulse rate of 75.

Just in front of the second vertical line, reading from the left, there is visible in the cardiac tracing a very slight angle, which shows the end of ventricular systole; this is quite clearly marked in the last systole (last on the right), and the two points exactly correspond.

Following on the slight angle first mentioned is a forward swelling on the falling line, caused by the reflux of blood from the aorta into the ventricle. The third systole (with still more deceptive ones obtained from the same patient) shows me how I have been many times led into the error of believing that the duration of ventricular systole was very greatly prolonged, instead of but slightly, in some cases of aortic insufficiency. In A.P. I might often have measured the duration of systole as 0.366", owing to the first point (betokening the genuine cessation of ventricular contraction) not appearing at all.

On January 12th, 1901, I obtained another tracing from A. P., in which the duration of V. systole is still clearly 0.33", though there is a slight yet interesting variation in the heart-index delay, to be mentioned later. In the latter tracing the aortic systole is still more distinctly marked than in the published tracing, and is clearly measurable (0.07").

NORMAL DELAY.

The normal heart-radial delay has been variously estimated. It may be taken to be 0.18". The delay from radial

to index pulse is, at the very most, 0.02". After innumerable experiments made on myself, and very many on others, I find the heart-index delay to be 0.19" to 0.20". Allowing for errors, inseparable from minute measurements involving hundredths of a second, one may take it that there is probably slight heart-index delay in A. P. (at the most $\frac{1}{10}$ th of a second), probably indistinguishable by touch.

A great advantage of Dr. Waller's digital sphygmograph is that the blood pressure at the time of the experiment may be measured, by taking the tracing (as would in any case be desirable) at the period of maximum excursion of the lever connected with the fingernail, and raising the lever to the mean height when connected with a mercurial manometer. On November 16th this stood at 16 mm. Hg.

On January 12th the patient was feeling altogether better, and said he felt none of his troublesome symptoms of November 6th. On January 12th the heart-index delay was 0.185", but the index blood pressure was 20 mm. Hg.

It is impossible not to connect these two facts. They go some little way to explain the discrepancies noted earlier in this paper. The rate of propagation of a pulse wave through an elastic tube is accelerated by increased and delayed by diminished tension, a fact well known to physicists. The variation in tension is enough to explain the accelerated propagation of wave in the tracing taken on January 12th.

But since there is abolition of the "*Anspannungszeit*" when there is free communication between the aorta and ventricle, how are we to explain the fact, which my tracing certainly shows, that in pure aortic regurgitation there may be no diminution in the heart-radial or heart-index interval? Dr. Waller⁷ gives the time occupied in the rise of intraventricular pressure from onset of systole to the opening of the aortic valves as between 0.06" and 0.09". Keyt estimates it as 0.08" for a pulse rate of 70. Assuming this interval to be absent in the case of A.P., owing to the aorta and ventricle forming one cavity, we ought, if no other proposition were involved, to find the heart-index interval shortened, not lengthened. To be 0.136" instead of 0.216" on November 6th, and to be 0.105" instead of 0.185" on January 12th.

EXPLANATION OF DISCREPANCIES.

Emptiness of arteries and diminished tension of their walls, will not sufficiently explain this discrepancy. Keyt's dictum "that the heart-radial interval is always shortened in aortic insufficiency" must obviously be cast aside. But neither is it right to say with Sir William Broadbent that "the pulse in aortic regurgitation is always retarded or delayed." It is true that it is sometimes retarded. But were it always so, which is clearly not the case, even then Sir William Broadbent's explanation of the fact as being due "to the collapsed and empty state of the arteries between the beats, and partly to their large size and loss of tone," would not cover the difficulty, even assuming that these conditions invariably prevail in aortic regurgitation, which is not my experience. That the

delay when present would be always noticeable by the sense of touch seems to me incredible.

To explain that it is ever noticeable by touch, we not only have to assume a low arterial blood pressure and enlargement and want of tone in the arteries, but to reconsider the fact that in free aortic insufficiency there is an "aortic" pressure in the ventricle at the beginning of systole. It is here, I think, that the additional and complete explanation is to be found.

In a normal heart the blood pressure in the ventricle at the beginning of systole is about 20 mm. Hg.; in less than one-tenth of a second the intraventricular pressure is raised to 150 or 200 mm. Hg., the effect is a sudden impulse or "slap" against the ventricular surface of the aortic valves, and the wave-created thereby is sudden and of great velocity—28 feet per second—(Von Frey).

A FACTOR NOT HITHERTO NOTICED.

In aortic regurgitation, when the intraventricular pressure at the onset of systole is already 150 mm. Hg. or upwards, the contraction is more of the nature of a steady push against a weight, and the resulting wave, not being created by impact, is a slowly propagated wave—that is, one of very much less velocity.

The elucidation of this point is the main object of this paper.

Yet another factor is to be found in the large mass of muscle in a hypertrophied heart. The large mass would, for physiological reasons, contract more slowly, owing to the propagation of nervous impulse through its substance being more slow. To this, in all probability, is due the slight increase in the duration of ventricular systole (a rare phenomenon), showing that the ventricular contraction is actually a slow contraction, the duration of ventricular contraction being strangely independent of extraneous conditions. It is less in enfeebled and fatty hearts, and the measure of duration of systole I look upon, not as having any bearing on extraneous conditions, but to be some indication of *plus* or *minus* structural change in the muscular substance of the ventricle.

In combination with relatively empty arteries, especially if enlarged and lax of wall, a long heart-radial delay might well be manifested, quite noticeable by the touch.

RECAPITULATION.

The chief contribution I have to make to the elucidation of this difficult point is that the pulse-wave produced in cases of aortic regurgitation is slow. This I take to be the main phenomenon in pulse delay. When delay is observed in these cases, increased arterial tension tends to obliterate the delay. Diminished arterial tension makes it still more manifest.

The second point, relating to the slow movements of a hypertrophied heart, is new in this connection also. If, however, we consider that as compensation is attained the arteries are better filled, giving to the patient a corresponding sense of comfort and well-being, and that then the pulse-wave is accelerated, we see that the attainment of a normal heart-radial delay is some measure of the correctness of the compensation, and that this may be a guide to us to avoid further interference.

In the course of work many indications are obtained which are suggestive, and useful enough to the observer, but which would be useless to others in the absence of convincing proof. Thinking that the evidence I give in this case leaves no room for doubt, I have discussed the whole question at length. I believe that the conclusions will be found to rest on a firm basis.

REFERENCES AND NOTES.

¹ BRITISH MEDICAL JOURNAL, vol. 1. ² *Heart Disease*, 1897. ³ Société de Biologie, March, 1878. ⁴ BRITISH MEDICAL JOURNAL, September 22nd, 1900. ⁵ The whole apparatus, including the small clock recorder, can be packed away in an immovable manner in a box measuring 6½ by 6½ by 17 inches; entire weight 18 lbs. The box can be carried on a bicycle "bi-carrier" without rattling or injury to its contents. ⁶ Landois and Stirling's *Physiology*, 1886, p. 97. ⁷ Notes of Observations on the Rate of Propagation of the Arterial Pulse Wave.

A ROENTGEN-RAY EXHIBITION.—A Roentgen-ray exhibition will be held at Hamburg on the occasion of the seventy-third Congress of German Scientists and medical men which is to be held in that city from September 22nd to 29th. The scientific part of the exhibition will be under the direction of Drs. Albers-Schönberg, Walter, and Hahn.

THE EFFECTS OF SEVERE MUSCULAR EXERTION, SUDDEN AND PROLONGED, IN YOUNG ADOLESCENTS.

By W. COLLIER, M.D., F.R.C.P.,
Honorary Physician to the Radcliffe Infirmary, Oxford.

In any attempt to ascertain the effect of severe muscular exertion, it is necessary, first of all, to study the physiological changes set up in the body by moderate exertion.

Curiously enough, although the English are more addicted to athletic exercises than any other civilised nation in the world, no English physiologist, as far as I know, has ever systematically investigated this subject from a physiological standpoint and recorded the results of his investigations. The only works on the subject I am acquainted with are *The Physiology of Bodily Exercise*, by Dr. Lagrange, written for the International Scientific Series, and *The Physiology of Sport*, by Dr. George Kolb, a distinguished German athlete.

As I wish to make my paper as practical as possible, I will start by discussing the effects of exercise on two of the most important organs of the body, the heart and lungs.

Breathlessness.

One of the objects of respiration is to get rid of CO₂, which is uninterruptedly manufactured in the body while life continues, and the accumulation of which in the body can cause death in a very short time. One of the results of muscular contraction is the production of CO₂, therefore the greater the number of muscles contracting and the more rapidly they contract, the more quickly does their gas accumulate in the blood. It would appear, then, that the most important factor in the production of breathlessness is the saturation of the blood with this gas. A sleeping man, when the majority of his muscles are in complete repose and the manufacture of CO₂ is at its lowest, breathes less deeply and less frequently than when awake. In hibernating animals the production of CO₂ is diminished to an extraordinary degree, and so is respiration. Lagrange states that CO₂ injected into the veins of a dog immediately quickens respiration, while a continuation of the injection brings on intense dyspnoea and all the usual symptoms of asphyxia.

We may conclude, then, that the respiratory need is in proportion to the quantity of CO₂ in the blood, and that in athletic exercises the quantity is rapidly increased by increased production. If now muscular work in a given time is so great as to produce a quantity of CO₂ greater than the lungs are able to eliminate the gas must accumulate in the system, and the respiratory distress will increase every moment, and will finally interrupt the work. Further, CO₂ has a weakening influence on muscular fibre, and causes its contraction to be less forcible and effective.

The amount of muscular effort necessary to produce breathlessness will vary with each individual and with the individual capacity of eliminating CO₂ by the lungs. The boy with large lungs, powerful heart, and an acquired knowledge of expanding his lungs to the best advantage will get rid of CO₂ much more rapidly in proportion to its manufacture than the boy with badly developed lungs, weak heart, and feeble chest expansion.

The Lungs.

The first effect of exercise is to increase the frequency and force of the heart's beat and quicken the blood current, more blood is sent to the lungs, and we get an active congestion. As a result of this active congestion the space occupied by the blood which swells up the pulmonary capillaries covering the air vesicles is no longer available for all the air in those vesicles. The lungs in consequence make an increased effort of expansion, and many of the air cells not usually in action at once expand with air to make good the deficiency. These changes occur especially about the apices of the lungs and along the free edges. As the muscular effort continues an increasing amount of CO₂ is thrown into the blood, stimulating the respiratory centre in the brain, inspiratory efforts become deeper and more frequent, and more air is drawn into the air cells, at once impeding the circulation through the pulmonary capillaries; a fight for place is going on in the lungs between the blood and the air; the heart beats more