probably a good deal more well-to-do. We get there the following figures:

Name of Parish.	Recorded Duration of Illness.			
	Under 1 Year.	1 to 1½ Years.	2 to 3½ Years.	4 Years and Over.
<i>Exposed to S.W., W., or N.W.</i>				
Ottertton (exposed to S.W.) ...	5	1	1	
Littleham (exposed to W., S.W., N.W.) ...	11	16	12	6
Withycombe Raleigh (exposed to W., S.W., partly N.W.) ...	8	11	12	2
Lympstone (exposed to W., S.W., partly N.W.) ...	5	6	5	3
Newton Poppleford (exposed to N.W.) ...	1	4	1	1
Totals ...	30	38	31	12
Percentages ...	27%	61%	39%	
<i>Sheltered from W., S.W., and N.W.</i>				
Budleigh Salterton ...	6	6	13	4
East Budleigh ...	5	2	7	4
Collaton Raleigh ...		1	3	1
Bicton ...			2	1
Totals ...	11	9	25	10
Percentages ...	20%	36%	64%	

Here, again, the exposed parishes have a higher proportion of comparatively short cases than the sheltered parishes and a lower proportion of comparatively long cases.

It will doubtless be objected to these tables that the recorded durations of cases of phthisis cannot be implicitly relied on. To this objection I would reply:

1. That it is very unlikely that the errors in three consecutive areas would all tell in just the same direction; and

2. That there is no reasonable ground for assuming such error as would invalidate these figures.

The much longer durations generally recorded about Budleigh Salterton than in Barnstaple and Okehampton rural districts may perhaps be jointly ascribed to more wealth, less wind, less rain, and more pervious soil. These, however, are matters which only concern us so far as to account for a difference which might otherwise be put down to varying accuracy of record.

Taking these tables together with those published in my former paper, I think a very strong case is established for the view that strong, prevalent, rain-bearing winds exert an influence upon the course, as they certainly do on the prevalence, of phthisis.

My best thanks are due to Dr. Young, of Okehampton, for kindly helping me to obtain the figures for his district.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, February 10th, 1912, i, p. 291. ² *The Influence of Strong, Prevalent, Rain-bearing Winds on the Prevalence of Phthisis*, 1910.

In his report for the year 1911 Dr. Waldo, Coroner for the City of London and for Southwark, states that the number of inquests on persons whose death was due to injury by vehicles reached a higher total than in any other year since he has been coroner for these areas. The number was 57, mechanically driven vehicles causing 39 of them, horse-drawn vehicles 18. The Herscher apparatus for preserving bodies by means of formalin has proved of signal service in identification, as well as for the other purposes for which it was installed at the City mortuary some two years ago. A similar apparatus is much needed in Southwark, since the number of drowned and unknown persons landed there is large. There were only four deaths during the administration of anaesthetics, and only one of these occurred at a certain hospital in which in an earlier year as many as eleven such deaths took place.

THE GENESIS OF THE DICROTIC PULSE WAVE.

By D. W. SAMWAYS, M.D., D.Sc., M.R.C.P.,
MENTONE.

IN Dr. James Mackenzie's *Diseases of the Heart*, edition 1910, he says:

There has been a good deal of discussion concerning the cause of the dicrotic wave. The semilunar valves are so delicately constructed that they readily respond when the pressure on one side rises above that on the other. As soon as the aortic pressure rises above the ventricular the valves close. At the moment this happens the valves are supported by the hard contracted ventricular walls. The withdrawal of the support by the sudden relaxation of these walls will tend to produce a negative pressure wave in the arterial system. But this negative wave is stopped by the sudden stretching of the aortic valves, which, on losing their firm support, have now themselves to bear the resistance of the arterial pressure. This sudden checking of the negative wave starts a second positive wave, which is propagated through the arterial system as the dicrotic wave.

I am very glad to see Dr. Mackenzie advocating the idea of the muscular mass of the ventricle contracting behind the aortic valves and acting as a support to them, because for twenty years I have maintained that the auricles, especially in mitral stenosis, may perform that service for the auriculo-ventricular valves.

I am, however, loth to admit that the aortic valves thus petted, are immediately afterwards so betrayed that they become suddenly stretched sufficiently to start a second positive, or dicrotic, wave. One may take for granted that any theory demanding so great and sudden a strain of these delicate structures is incorrect.

In 1894 I advanced another theory, in my thesis for the degree of M.D. at Cambridge, which I think better explains the mode of production of this wave. I maintained that the dicrotic wave is a ballistic or catapult wave, due to the longitudinal recoil of the first part of the aorta, after it has been longitudinally stretched by the contracting heart.

When the ventricle contracts with sufficient force the aortic valves open, and the blood in the aorta and in the ventricle forms one continuous column under pressure. This rigid column occupying the aorta and ventricle presses its foot on the inferior wall of the heart near the apex, and is probably a chief factor in preventing the apex moving upwards when the ventricle contracts. In fact, the arch of the aorta and the lower wall of the ventricle are mutually repelled by the pressure of the blood column lying between them and thrusting them apart.

With a ramrod of blood pushing it down, the apex cannot come up during contraction, and the base has to be drawn down towards it (as shown by the needle experiments of our early physiology days) during ventricular contraction, pulling with it the aorta, which consequently lengthens. The aorta is drawn like the finger of a glove over the blood at the base of the ventricle, the remainder of the ventricular blood being driven through the aortic orifice after it.

As the aortic pressure rises and the outflow from the ventricle decreases, the aortic valves are pushed towards one another by the blood behind them in the aorta, and finally closed. The pressure of the aortic column now falls on the valves, supported temporarily from behind, as Dr. Mackenzie suggests, by the "hard, contracted, ventricular walls."

Something else, however, now happens. The pressure in the aorta near the heart begins to fall, and the longitudinally stretched aortic walls soon begin to recoil to their normal length, just as the circularly stretched walls have begun to return to their normal diameter. And just as the circular recoil drives onward through the arteries the main pulse wave so the longitudinal recoil, which follows closely on it, shoots on a second wave—the dicrotic or ballistic wave. Any one who will examine the first part of the aorta will be amazed at the amount of its longitudinal elasticity (using the word in the vulgar sense), and if it be closed and pumped up it will be seen to lengthen considerably.

It might be objected that the longitudinal and circular recoil of the aorta would combine to generate the main pulse wave. That would only be true if the longitudinal recoil were as powerful, and so could commence simultaneously with the circular. This does not appear to be

the case. The advent of the longitudinal recoil is therefore slightly delayed, and the dicrotic wave, due to the oncome of a diminution in the longitudinal capacity of the first part of the aorta, is superposed on the main wave early in its descent.

The mode of production of this ballistic wave can easily be imitated with a model consisting of rubber tubes and pump representing the circulatory system. If, when the pump is squeezed, it be also pulled to stretch the tube into which its contents flow, and, after squeezing, allowed to yield to the stretched tube in its recoil, one wave is generated by the pump and a second smaller one by the tube in its longitudinal recoil. The whole, properly recorded, exactly resembles the main features of a normal pulse tracing, with the dicrotic wave small or large according to the rapidity of recoil allowed.

A confirmatory experiment I performed on a large dog. After anaesthetizing it, the chest wall and pericardium were opened. A tube introduced into a main artery of the leg allowed the pulse tracings to be recorded graphically. The aortic walls close to the heart were then seized and held by two pairs of forceps, and prevented as far as possible from making any movement in a longitudinal direction. The dicrotic wave, which before was conspicuous, immediately disappeared, and reappeared at once on the tracings when the aorta was set free to perform its longitudinal movements.

I have since had no doubt that the dicrotic wave is to the longitudinal recoil of the first part of the aorta what the main pulse wave is to its circular recoil.

A NOTE ON BLOOD-PRESSURE READINGS IN CASES OF AURICULAR FIBRILLATION.*

BY

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IN examining a radial pulse tracing taken from a case of auricular fibrillation one of the most noticeable characteristics is the inequality of the heights of the systolic primary waves—forcible beats and feeble beats occur in no definite order.

This suggests that the systolic arterial blood pressure is also a varying factor in these cases, and that to assign a single value for the blood-pressure readings in such patients is scarcely justifiable.

A simple method for ascertaining the systolic pressure reading for the various beats is as follows: The armlet band of a blood-pressure apparatus is adjusted on one arm—the right is the more convenient. A wrist splint of a Mackenzie ink polygraph is strapped on over the right radial artery, and another over the left radial, and the corresponding wrist tambours are connected by rubber tubing to the receiving tambours of the polygraph. In this way the two radial tracings are recorded on the one strip of paper. The apparatus is adjusted till the best radial impulse in each is obtained, and so that the swings of the writing pens are approximately equal. The paper is allowed to run through, and the pressure in the armlet band is increased by 5 to 10 mm. at a time, and that pressure noted at which the various beats fail to affect the recording apparatus. Sooner or later, as the armlet pressure is raised, the smallest beats of the pulse on the corresponding arm are lost. The reading of the manometer, at which this occurs, gives the minimal systolic pressure. When all the beats are obstructed, the maximal systolic pressure

has been obtained. It is found that with highly irregular hearts there may be a difference in the maximal and minimal readings of 60 mm. Hg. With less irregular hearts—that is, those in which the rate is slower—the difference is not so marked, and in those that are only slightly irregular—as, for example, where the ventricular rate has been markedly retarded by digitalis—the difference may not amount to more than 5 to 10 mm. Hg.

Janeway¹ has previously called attention to the difference in the systolic blood pressure readings in cases of arrhythmia.

The readings taken from 8 patients, all of whom were typical examples of cardiac irregularity due to auricular fibrillation, are given in the following table:

Case.	Blood Pressure Reading of Smallest Beats.	Maximum Blood Pressure Recorded.	Difference in mm. Hg.	Pulse Rate per Minute.
1	150	210	60	56
2	80	100	20	72
3	100	130	30	92
4	95	140	45	74
5	130	140	10	44*
6	115	140	25	84
7	120	140	20	72
8	160	200	40	76

* On digitalis.

This table clearly shows the wide range of blood pressure which an individual case of auricular fibrillation may possess. This variation is of importance, because in the cases in which it occurs single observations of blood pressure, taken in the ordinary manner, possess only a restricted value as an index; the error introduced in continued and comparative observations, where each

Fig. 1. A, B, and C.—Three strips of tracing (right and left radial) taken from the same patient.

A. Before brachial compression; all the beats come through.

B. The smallest beats × × disappear at a pressure between 90 and 100 mm. Hg.

C. The largest beats fail to record at a pressure of 140 mm. Hg.

observation is confined to a reading of the most forcible beats, is also very considerable.

REFERENCE.

¹ T. C. Janeway, *The Clinical Study of Blood Pressure*, p. 205.

* From the Cardiographic Department, University College Hospital Medical School.

A BEQUEST of £300 has been left to the East Sussex Hospital, Hastings, under the will of the late Mary Jane Day of Bexhill.