thoracic surface, as well as for the study of the intra-thoracic conditions—the latter, however, owing to the possible bony conduction of sounds originating within the chest, present a complicating element of difficulty and of doubt. It is noteworthy that the sign was first described in a case of tuberculosis. Although no general significance attaches to that circumstance, the sphere of its clinical utility seems likely to remain largely identified with a more thorough physical examination of the chest for the diagnosis of tubercular affections of the pulmonary apices and of the mediastinum, in addition to its everyday employment in the diagnosis of rheumatic affections of the scapular and sterno-clavicular articulations and of their ligaments and muscles.

The Practical Value of the Method.

The chief recommendations of the method are its simplicity when conducted with the binaural stethoscope and the amount of information which it is capable of furnishing. By opening up a new field of observation its systematic employment might lead to clinical results the importance of which we cannot foretell. I venture to suggest to clinical inquirers that they make a rule of combining the articular with the upper thoracic examination in all cases yielding any stethoscopic sounds, and of noting the result. The most convenient technique is to firmly steady with one hand the cup of the stethoscope over the front of the head of the humerus, which it fits well, or in dorsal examinations over its side or over the flat acromial surface of the spine of the scapula, whilst the other hand grasps the elbow and imparts to the humerus a full range of strictly passive movements. The sterno-clavicular examination presents no difficulty.

1. *For articular conditions*, as this is by far the finest test that can be applied, the examination should be *de rigueur*. A negative result from auscultation and from manipulations would be the quickest as well as the only conclusive evidence of absolute soundness. Should, however, any articular sounds be heard, let us continue the movement, and simply shift the chestpiece to the supra-clavicular and infraclavicular regions—a matter of a few seconds, and well worth our while if any unexpected sounds should happen to be heard.

2. *For pulmonary and pleural conditions* the indication is obvious. The earliest detection of apical tuberculosis is the question of the day. Our time and trouble must not count; the shadowy indications of radiology draw largely upon both, but they are worthless in the absence of the best technical and instrumental advantages, and this is also true of the stethoscope. Meanwhile, should we continue to neglect a whole line of evidence which lies close at hand and needs no technical refinements? In all doubtful cases we should proceed to submit our apical results, whether positive or negative, to the articular test. Perez has taught us that there are lesions (in my experience chiefly pleural, but sometimes pulmonary) which may give sound under the mechanical traction of movement, and not always under that of deep breathing only; and the no less important fact that some of the sounds audible over the apices may be merely conducted from the sterno-clavicular or shoulder joints. There is no choice for us but to take the trouble to apply the necessary differential tests. Without them there can be no strictly conclusive overhauling of the chest.

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A LABORATORY for the investigation of pellagra has been established by the Public Health and Marine Hospital Service in Savannah, Georgia. Dr. C. H. Lavinder is in charge of the work.

THE INFLUENCE OF STRONG, PREVALENT, RAIN-BEARING WINDS ON THE COURSE OF PHTHISIS.

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[Second Paper.]

In a first paper on this subject, recently published,¹ I have shown that strong, prevalent, rain-bearing winds probably exercised a considerable influence on the course of phthisis in the Exmoor portion of Barnstaple Rural District during the forty-five years 1860 to 1904.

I now propose to show, in two other areas in quite different parts of Devonshire, a similar influence of these winds—namely, in the rural district of Okehampton, in Mid-Devon, during the fifty years 1860 to 1909, and in the parishes round Budleigh Salterton, in St. Thomas district of South-East Devon, during the forty-five years 1866 to 1910. Following the plan of my former paper, I have only considered cases under 50, as I find here, just as I did there, that from 50 onwards cases tend to be more chronic. As cases under 10 in these two areas evidently tend to be disproportionately acute, I have also omitted them. So that the cases given died over 10 and under 50.

As I have carefully described elsewhere the rural district of Okehampton,² where a most marked contrast was shown to exist in phthisis prevalence between the parishes fully exposed both to west and south-west winds and the parishes sheltered from them both, it is only necessary to recall the facts that most of the parishes are on impervious soils with a very considerable rainfall, and that many of the sheltered, as well as most of the exposed parishes, are more or less open to north-west wind.

Taking the same two groups of parishes that afforded the striking contrast in regard to phthisis prevalence, we obtain the following table:

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 both W. and S.W. Winds.</i>				
Highampton	3	1	1	
Beaworthy	5			
Bratton Clovelly	2		1	1
Sourton	2			
Totals	12	1	2	1
Percentages	73 %		18 %	
<i>Sheltered from W. and S.W. Winds.</i>				
Inwardleigh				
Monkehampton				
Chagford	9	7	5	1
Gidleigh	1			
Throwleigh	5	3	1	
Drewsteignton	7	4	6	
Ashbury				
Totals	22	14	11	1
Percentages	45 %		25 %	

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

Turning next to the parishes about Budleigh Salterton. These are nowhere so severely wind-swept as the preceding, nor is their rainfall so great; the soils of all are comparatively pervious, and the average inhabitant is

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.

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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