After a while, the patient began to make steady though slow progress, and with the exception of occasional severe attacks of dyspnoea there was nothing of particular note.

On March 2nd it was noted that the expectoration was muco-purulent, much less in quantity, and absolutely odourless.

He was discharged from the hospital on May 18th, having been in the wards just over three months.

He returned on May 28th, when it was found that the wound was firmly healed; the expansion of the lungs was good; there was slight dulness on percussion over an area some 8 inches in diameter surrounding the cicatrix, due to thickening of the pleura. He had put on a great deal of flesh, and was looking in very fair health.

He subsequently went a short sea trip, and returned on June 25th, looking in robust condition.

PULSUS BISFERIENS.

By WALTER BROADBENT, M.D. CANTAB., M.R.C.P.,
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WHILE house-physician at Brompton Consumption Hospital three years ago a patient came under my care who presented the curious condition of having a well-marked "bisferiens" pulse on the left side, while on the right the pulse was only that of aortic regurgitation; and since that time I have come across two similar cases, so the condition is apparently not uncommon.

CASE I.—A man, aged 41, who had had rheumatic fever at the age of 16 and again at 20. His radial arteries were large and a little tortuous; the pulse was short and sudden on the right side, but not completely collapsing; on the left side it could be distinctly felt to be double, especially when a little pressure was applied.

Right Pulse. Case I.

Left Pulse

This state of his pulses was invariably present during his six weeks' stay in the hospital. Three months later he died suddenly. His heart weighed 2 lbs.; both ventricles were greatly dilated and hypertrophied, especially the left. The aortic valve was incompetent and the orifice narrowed, admitting only the tip of a finger; the valves were retracted and rigid. The mitral orifice was also narrowed, admitting one finger easily; its aortic cusp was much thickened and contracted, the other less affected. The aorta was quite normal up to the origin of the left subclavian artery, but just beyond this point was a shallow pocket, the size of a shilling, in the aortic wall, which was here thinned and atheromatous, and from this point a long line of atheroma extended down the left side of the thoracic aorta. Nothing abnormal was found in the carotids or subclavians.

CASE II.—A boy, aged 18, who had had attacks of rheumatic fever at 13 and 16. His radial arteries were larger than in the last case, and they emptied almost completely between the beats; his right pulse was short and sudden, but the left was markedly bisferiens. The left carotid could be felt to be bisferiens, but the right not; a tracing of the right, however, showed an approximation to the bisferiens character. On examining the posterior tibial artery I found the pulse was not bisferiens, and the tracing corresponded to that at the right wrist.

Posterior Tibial. Case II.

This boy died suddenly about a year later; his heart weighed 2 lbs. 1½ oz., both ventricles were greatly dilated and hypertrophied. The aortic valve was incompetent, and the edges of the cusps were thickened, causing slight stenosis; the right posterior cusp had on its aortic surface a calcified prop which kept the cusp stretched across the orifice. The aorta was slightly dilated generally; nothing abnormal was found in the large arteries; the mitral orifice admitted three fingers; the valves were

normal; the tricuspid was slightly stenosed, not admitting two fingers its valves were thickened.

Case II.

Right Radial

Right Carotid

Left Carotid

Left Radial

The Aortic Orifice. Case II.

CASE III.—A woman, aged 32, had attacks of rheumatic fever at the ages of 17 and 20. Her radial arteries are larger than normal, and pulsation is visible in them; her right pulse is sudden and moderately collapsing, while the left is strikingly bisferiens to the finger. The left carotid feels bisferiens but the right does not; the dorsalis pedis artery gives a tracing similar to that of the right pulse. Her apex beat is in the sixth

Right Pulse

Left Pulse

Dorsalis Pedis

space, 1½ in. below and 1 in. external to the nipple. It is powerfully thrusting. At the apex are heard a low rumbling presystolic murmur leading up to a short loud first sound, a systolic murmur, and a diastolic which runs into the presystolic. At the tricuspid area a loud first sound, blowing systolic murmur, a second sound, and a louder and longer diastolic murmur. At the pulmonary cartilage a harsh rough systolic murmur, loud second sound, and a diastolic murmur. At the aortic cartilage a long rough systolic murmur, faint second sound, and a diastolic murmur. In the neck a loud rough systolic murmur and a faint diastolic murmur, no second sound. The amount of hypertrophy of the heart taken with the artery at the wrist, which is only moderately large and does not collapse entirely between the beats, makes it probable that there is in this case also aortic stenosis as well as regurgitation. Previous to these cases I had seen four patients in whom one radial artery gave an anacrotic tracing, but I do not know whether both arteries gave it or whether it was the right or the left; they all had evidence of aortic regurgitation with stenosis, and in one, *post mortem*, the aortic

orifice was two-thirds occluded by rigid and thickened valves, and there was also mitral and tricuspid stenosis and adherent pericardium; the heart was very large. I give here also the tracings of another case of aortic regurgitation with stenosis, in which there is a striking difference between the two pulse tracings, the left approaching the bisferiens condition, but I was not able with the finger to feel a double impulse.

In the first case the presence of the weakened spot in the wall of the aorta, and the line of atheroma beyond it, taken with the absence of atheroma between the valves and this spot, proved that the main blood stream from the ventricle impinged on this spot close to the origin of the left subclavian, possibly directed by the shape of the contracted valves; and in the second case the rigid condition of the right posterior cusp suggested that the blood stream was similarly directed by the shape of the orifice; but in the third case there is the same condition of the pulses, and it is unlikely that there should be a peculiarity in the shape of the aortic orifice in three cases directing the blood to the same spot, so it may be concluded that it is only the natural direction of the main blood stream (quoted so often as the cause of the greater frequency of cerebral embolism on the left side), accentuated by the increase of the volume of blood driven from the enlarged left ventricle through the narrowed aortic orifice, and it suggests that the anacrotic tracing in the left radial and carotid may approximately represent the intraventricular blood pressure.

In taking cardiograms of the apex beat in the second and third cases, the notch attributed to the contraction of the papillary muscle does not come after the highest point as in the normal apex beat curve, but I find that this occurs also in other cases of aortic regurgitation, and is probably therefore due to the hypertrophied ventricular wall not attaining its full contraction as soon as usual, or not being so much affected by the pull of the papillary muscles as in the normal heart, the relation of the cardiographic trace to the bisferiens pulse is thus uncertain.

Roy and Adami in their investigations on the hearts of dogs¹ found that a rise of peripheral resistance makes the intraventricular curve anacrotic, as if the contraction were reinforced by a renewed effort, and they conclude therefore a corresponding pulse; but in cases of high tension the bisferiens or anacrotic tracing is not found at the wrist, except sometimes in senile degeneration of the arteries.² But Roy and Adami took their tracings from the aorta; I have consequently taken a tracing of the pulsation in an aneurysm of the arch of the aorta, as the nearest approach to one taken

from the aorta, and find the anacrotic character there, while the pulses do not present it.

Tracing of Pulsation in an Aneurysm of the Aorta.

There is high peripheral resistance in this case, the arteries at the wrists being large and full between the beats. In aortic stenosis and regurgitation increased resistance is given by the narrowing of the aortic orifice, and therefore in all such cases one would expect an anacrotic tracing for the intraventricular pressure. In the aneurysm that tracing appears, and therefore would probably have been found if a tracing of the aorta could have been taken in any of the cases related. It does not occur in either pulse in the case of aneurysm, nor in any arteries in the other cases except in the left carotid and radial. Its presence in them has evidently to do with the direction of the main blood stream on to their points of origin from the aorta; and one must suppose that the elasticity of the walls of the aorta and the eddies of the blood modify the other pulse tracings in some way, just as a normal pulse tracing is very different from the normal curve of intraventricular pressure. When the arterial system becomes degenerate as the result of very long-standing high tension, one would not be surprised to get the curve of intraventricular pressure conveyed in the rigid arteries to the two wrists, and this would explain the occurrence of the bisferiens pulse in senile cases where the arterial tension is still high; and in cases of aortic stenosis supervening on regurgitation, if the arteries had been so damaged as to have lost their elasticity, one would expect a bisferiens pulse in both wrists. I have not had the opportunity yet of investigating whether the anacrotic tracing is often obtained from the pulsation of aneurysms, nor whether it disappears with the lowering of the vascular tension, but if so the anacrotic tracing would be a useful indication of too high pressure in an aneurysm.

The first two cases also had the visible retraction, synchronous with the cardiac systole, of both backs in the region of the eleventh and twelfth ribs, on which sign I published a note in the *Lancet* in 1895, attributing it to adherent pericardium; in neither of these cases was that condition present, though in both cases there were signs of old pericarditis, and I have now seen the necropsies of 4 cases of this sign, in only one of which was the pericardium totally adherent. In all four the heart was much hypertrophied, and adhesion existed between the two layers of pleura in front of the heart. Dr. Kingston Fowler has since pointed out in a case of pyopneumothorax, where portions of ribs had been removed for drainage, that the diaphragm could be seen to be raised at every systole by the normal heart, so that the explanation seems to be that a hypertrophied heart, helped perhaps by adhesion of the pericardium to the chest wall, is able to lift the diaphragm to such an extent as to pull in the lower ribs on each side behind.

Since writing the above my attention has been called to a most interesting paper by Dr. Graham Steell on The Pulse in Aortic Stenosis,³ in which he describes 6 cases of anacrotic or bisferiens pulse, and concludes that though suggestive of aortic stenosis the condition is not diagnostic. I have differed from Dr. Steell in the use of the terms "anacrotic" and "bisferiens," using the term "bisferiens" when speaking of the pulse, since it conveys the idea of the double effort one feels with the finger in these cases, and the term "anacrotic" as descriptive of the tracing. He does not go into the causation of the pulse, but I think his cases support my theory. His first case had marked aortic stenosis, also incompetence, and as well mitral and tricuspid stenosis and adherent pericardium. The pulse was most commonly rapid (120), and was not then bisferiens, but when it was slowed down by digitalis it became bisferiens. In this case I think the explanation is that, at the rapid rate of heart beat, the left ventricle was not properly filled through the stenosed mitral orifice, that consequently there was not high pressure in it, and thus no anacrotic tracing; the aortic stenosis seems to have been too pronounced for the raising of the peripheral resistance by the digitalis to have had much effect on the intraventricular pressure. The second case had precisely the same condition

of heart *post mortem*, the pulse was 50, and the tracing apparently always anacrotic. The third case had marked aortic stenosis, no regurgitation, a much enlarged heart, and constantly showed the bisferiens condition of pulse.

The next case is most interesting; a man of 49, with fibrosed kidneys, marked mitral stenosis, the aortic valves thickened, allowing slight regurgitation, but with absolutely no stenosis; he was first admitted with a bisferiens pulse (64 to 84); the condition of the arteries is not mentioned, but, if they were rigid from the prolonged high tension of the contracted granular kidney, the anacrotic intraventricular pressure curve could reach the pulse. When next admitted the man had pericarditis and heart failure, with a very rapid pulse not giving an anacrotic trace; either the fever of pericarditis had lowered the vascular tension, or else the mitral stenosis prevented the filling of the ventricle at the rapid rate of heart beat, and the intraventricular pressure was lowered, and the pulse therefore not bisferiens.

In none of the above does Dr. Steell state whether the pulse in both wrists gave the same tracing, but his fifth case corresponds to my second in having an anacrotic tracing in the left wrist, but a tracing of aortic regurgitation in the right. There was a large heart—20 oz.—tricuspid regurgitation, mitral stenosis, and very marked aortic regurgitation, the orifice being a thickened ring easily admitting one finger, so that there was no real stenosis, though, as Dr. Steell remarks, the orifice was smaller than normal, while the ventricle was much larger. The patient had, besides rheumatic fever, suffered from scarlet fever, followed by dropsy, 14 years before; the condition of the kidneys *post mortem* is not mentioned, but if diseased the increased peripheral resistance so caused would help to account for the bisferiens pulse. In the last case in the paper both pulses gave anacrotic or bisferiens tracings; the fall between the two cusps in the right tracing is much greater than in the left, so that the bisferiens condition appears more marked in the right pulse than in the left. But the fall before the diastolic wave is also greater on the right side, and it appears to me that this means that the column of blood is not so well sustained on the right side as on the left, and that the main blood stream is again carrying a truer tracing of the intraventricular blood pressure to the left pulse. In this case there was very marked aortic regurgitation, no definite stenosis, but a very large left ventricle; also parenchymatous nephritis, which by raising the peripheral resistance must have led to the development of the bisferiens pulse. These last two cases also strengthen my conviction that the difference between the right and left tracings, which was present in my cases of bisferiens pulse, will be found to be the rule rather than the exception.

REFERENCES.

¹ *Practitioner*, 1890. ² *The Pulse*, p. 26. ³ *Lancet*, 1894.

THE VISION OF SCHOOL CHILDREN.

By J. ACWORTH MENZIES, M.D. EDIN.,
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IN the BRITISH MEDICAL JOURNAL of June 18th, 1898, Mr. Arnold Lawson has published an Abstract of a Report on the Vision of Children attending London Elementary Schools. The report was the result of an inquiry held at the request of the Education Department, and embraced seven typical elementary schools. In some American cities such an examination is carried out annually under the supervision of an officially appointed oculist. The importance of such an investigation to the cause of education and to parents, teachers, and the children themselves is so obvious that it scarcely requires to be dwelt upon. The headaches consequent on refractive errors, and their disappearance when the errors are corrected are well known, and a diagnosis of stupidity has frequently to be reversed after the use of suitable glasses. As to more remote benefits many a boy would be saved from entering on an unsuitable career if he knew at the outset that for him in the near future spectacles would be a necessity for close work. One frequently meets with cases of men discharged, say, from the railway service, because presbyopic changes, superadded to a congenital hypermetropia, prevent

them at a comparatively early age from passing the "dot test." Or it may be a mechanic who loses his employment shortly after completing his apprenticeship, because he cannot do his work efficiently without correcting glasses. In such a case a skilled workman must learn a new trade in which spectacles are no disadvantage, or become a labourer, because he did not discover in time that he was hypermetropic.

The importance of examining the refraction of all children's eyes becomes more apparent when the proportion of cases of defective vision is realised. Mr. Lawson, in 2,014 London children, found only 698, or 34.6 per cent., with normal vision in both eyes. My own examination of 398 Rochdale elementary school children gives 177 normal, or 44 per cent. In addition to these I found 126, or an additional 32 per cent., who would have passed Mr. Lawson's test as normal, that is to say, they read $\frac{1}{2}$ with each eye, but I found on testing with weak convex glasses that they had a small amount (1D or more) of manifest hypermetropia. Allport found out of 25,696 children in the Minneapolis public schools, 8,166, or 32 per cent. defective, but he does not seem to have tested for low degrees of hypermetropia, and probably the percentage of emmetropic children was much lower than 68. Another consideration which makes it desirable that the vision of each individual should be tested at as early an age as is practicable is that a child, or a grown-up person for that matter, may be quite content with defective vision through ignorance that the vision of others is any better than his own. This is the case especially in the minor degrees of astigmatism.

The importance of detecting defective vision early in life is then, it seems to me, sufficiently obvious. The difficulties in the way of doing so are few. The method followed by Mr. Lawson is simple, and easily carried out. If adopted by the managers of schools throughout the country it would mark a distinct advance in the performance of the duties of those who have the care of children.

Early in the present year I undertook the investigation of the eyesight of children in the Rochdale Parish Church School at the request of Archdeacon Wilson. I carried out the entire investigation personally, first testing each eye of each child by means of Snellen's types and trial lenses, and then examining with the ophthalmoscope and retinoscope those whose vision was subnormal.

For convenience I may follow Mr. Lawson's method of classification in tabulating my results, except that I shall group all the cases of astigmatism together. Further, among the cases which I have grouped as hypermetropic some would probably, on careful examination, prove to have a certain amount of astigmatism. The percentage figures are approximate.

TABLE I.—Normal Vision.

	Boys and Girls.	Per Cent.	Boys	Per Cent.	Girls.	Per Cent.
Normal vision in both eyes ...	177	44	96	48	81	41
Ditto but slight Hm...	126	32	45	22	81	41
Defective vision in one or both eyes...	95	24	59	30	36	18
Totals ...	398	—	200	—	198	—

TABLE II.—Showing the Various Errors of Refraction and the Relative Frequency of One Form of Error to Another (Cases 221).

	Boys and Girls.	Per Cent.	Boys.	Per Cent.	Girls.	Per Cent.
Hypermetropia ...	165	75	66	63	99	85
Astigmatism ...	40	18	29	28	11	9
Myopia ...	16	7	9	9	7	6