ON THE ACCENTUATION OF THE SECOND
SOUND IN THE PULMONARY AREA. SKODA'S
SIGN.

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IN medicine, more than in almost any other branch of natural science, facts accumulate and theories disappear. Destructive criticism is a necessary force in scientific progress, and this must be my excuse for asking for a reconsideration of Skoda's theory of the accented second sound; even though the weight of authority has been on its side for fifty years. "When," he says, "the pulmonary artery is abnormally distended, which it always must be when the pulmonary circulation is obstructed, its second sound becomes very loud. . . . The pulmonary artery when thus distended presses with increased force upon the blood within it, and consequently the backward stroke of the column of blood against its semilunar valves becomes more forcible." This is anent aortic incompetence. Again, in discussing mitral insufficiency, he says "the second sound of the pulmonary artery is increased, and the accent falls distinctly upon it." In mitral stenosis, "the second sound of the pulmonary artery is increased."^a

This observation and explanation of Skoda's have been accepted, practically without reserve, by almost every subsequent authority in English medical literature, and, so far as I know, are equally uncontroverted on the Continent. Yet, I cannot help thinking that the statement of fact is not by

^a Skoda. A Treatise on Auscultation and Percussion. 4th edition. Translated by Markham. 1853.

any means a complete one; and I shall offer reasons why the explanation does not seem to me to be reasonable. So far as I can find Skoda's reasoning on the point is wholly *à priori*, and may, I think, fairly be stated thus:—*In many cases of disease involving the left side of the heart, the second sound is heard with marked clearness and even accentuation at that spot on the chest wall at which the pulmonary artery comes nearest to the surface. The sound is therefore in the pulmonary artery. Disease of the valves of the left heart induces a tendency to more or less delay in the circulation through the lungs: the pulmonary system is in fact over-full; there is therefore a condition of engorgement of the pulmonary artery, and consequent slamming, so to speak, of the pulmonary valves, indicated by an exaggerated noise or accent.*

With regard to the first conclusion in the foregoing statement, it will be granted, I think, that a vascular murmur heard on the surface is not necessarily produced by that vessel which happens to be most superficial at the point of observation. Without elaborating this point too far, I need only point out that the pulmonary artery is closer to the surface than the aorta in the mid-sternal line and even at the right border where one would make no difficulty about calling a systolic murmur an aortic one; and with regard to the diastolic basal murmur, that it is frequently best heard in the lower part of the so-called pulmonary area, in the third left interspace, and that no one would think of concluding from that fact alone that such a murmur indicated an incompetence of the pulmonary valves. The argument from position seems to me to receive its *coup de grace* from the fact that, although at the "pulmonary" area the pulmonary artery is superficial to the aorta, yet the valvular orifices are very nearly in the same plane, one of them actually overlapping the other, so that the mass of the pulmonary artery and conus arteriosus

forms an admirable medium for the conduction of the aortic sounds to the surface.*

With regard to engorgement of the pulmonary vessels as a cause of increased recoil in the pulmonary artery and slamming of the pulmonary valves, I think the argument *à priori* is equally weak. Other things being equal, the loudness of the diastolic note will be in proportion to the amplitude of the recurrent wave from the periphery in the pulmonary artery. But in mitral incompetence or stenosis the pulmonary artery tends to be still fuller than it is in health, and in health it is, as the physiologists teach us, somewhat distended or overfull. In disease of the mitral valve, therefore, the artery wall is stretched in the presystolic period nearer to its limit of elasticity, and therefore its range of post-systolic recoil is more limited. That is to say, that the amplitude of the recurrent wave will be less than normal, and the intensity of the second sound should be correspondingly diminished.

But theoretical considerations such as the preceding have no value compared with the results of actual observation. Let us see, then, whether there are any practical objections to Skoda's theory. In the first place I point to the fact that this accentuation is absent in many cases in which, according to the theory, it ought to be present: Thus it is by no means common in emphysema of the lungs; nor in phthisis, unless a consolidated piece of lung intervenes between the base of the heart and the thoracic wall; nor is it heard usually in aortic regurgitation (notwithstanding Skoda's statement to the contrary), even though there be

* It is misleading, it seems to me, to speak of the second right interspace as the aortic and the second left as the pulmonary area. This localisation had a value for the distinction of aortic from pulmonary *systolic* sounds; but when we come to diastolic murmurs it has no value at all; and I am inclined to think that even the ordinary second sound in the normal heart is better heard at the left border of the sternum than at the right.

signs of delay in the pulmonary circulation; nor even in aortic regurgitation when mitral incompetence has been superadded; nor indeed is it present in all cases of mitral disease anterior to failure of compensation. Again, the accentuation is present in a considerable number of cases in which there is not the slightest reason to suspect either heart disease or any tendency to delayed pulmonary circulation. Thus it is commonly present in children, in neurotic persons, in chlorosis, and at the beginning of fevers. Finally, I shall make brief mention of three cases in which the accentuation disappeared, and the second sound became very faint coincidently with the supervention of a diastolic aortic murmur, thus indicating, in my opinion, that the so-called pulmonary accentuation was really aortic.

CASE I.—R. O'C., aged thirty-two, Whitworth Hospital, 1896, mitral regurgitation and accented second sound: while under observation he developed a systolic aortic murmur, and a little later a short diastolic murmur became audible. At the same time the "pulmonary" accent, and indeed the second sound in that area, disappeared, and the only second sound of pure character which we could find was a faint one at lower right border of sternum. The necropsy confirmed the diagnosis.

CASE II.—E. D., aged eleven. Purpura with endo- and pericarditis (malignant.) Admitted to Children's Hospital, 10th September, 1896, under care of a colleague, who noted "loud mitral regurgitant murmur, with accentuated pulmonary second sound and hypertrophy of left ventricle." She came under my care about the middle of October. I examined her carefully on the 21st and 23rd, and found the mitral murmur, and also a double aortic murmur, but not a trace of any pulmonary accentuation, and in fact no pure second sound at all, notwithstanding that the increased bulk of the heart had pushed aside the lung. I had her examined again by my colleague, on whose accuracy in clinical observation I place the greatest value; and, though he accepts Skoda's views, he corroborated my observation as to the almost complete disappearance of a sound which a few weeks before had been intensified. The diagnosis as above was confirmed by necropsy.

CASE III.—Mrs. K., aged forty-two, was under my care in the Whitworth Hospital in 1897 with presystolic apex murmur and thrill, and “second sound accented in pulmonary area.” Re-admitted recently, and still under my care; she presents a short systolic as well as the presystolic apex murmur, but the accentuation in the pulmonary area has given place to a very faint second sound, with which is heard a soft murmur. I am driven to conclude that whether by reason of a sclerotic process ascending the anterior mitral valve into the aortic valves continuous with it, or of an independent valvulitis, she has now a slight aortic regurgitation murmur replacing the original aortic accent.

These cases indicate the direction in which I look for an explanation of the sound under discussion; though I am far from maintaining that the pulmonary artery takes no part in the production of the second sound—for in Cases I. and III. I am prepared to accept the faint second sound as mainly, if not entirely, pulmonary—I am slow to believe that the vigorous second sound of early mitral disease is mainly pulmonary. In my view it is produced at the aortic orifice as the result of increased amplitude of the recoil wave in the systemic arteries: the increase in the amplitude being caused either by diminished quantity of blood thrown into them by a vigorous ventricle, as in mitral disease, or by diminished vasomotor tonus, as in children and neurotic adults, for instance. An examination of the radial pulse in these cases will usually show that it is soft, rapidly dilating, and rapidly contracting; it might be called a resilient pulse, or a “puerile” pulse, from the absence in it, as in the child's pulse, of any hint of continued tension.

If this view of its causation be accepted, the accented second sound might help us, in addition, of course, to other and I confess much more valuable criteria, to recognise and, as it were, delimit three stages in an ordinary case of mitral disease. In an early stage there may be simply the murmur, without any accentuation of the second sound or

any other sign of lack of compensation. Later on the second sound may be accented—if transitorily, without much morbid significance; if constantly, indicating diminished systolic influx into the aorta from a vigorous ventricle or relaxed vasomotor tone. Finally, the intensity of the second sound diminishes from supranormal to subnormal in proportion as an impeded or engorged venous circulation offers more and more resistance to the arterial flow; in fact, as the condition of the general circulation begins to resemble that which had been established earlier in the pulmonary area. Given, then, an accented second sound, constantly present, I should assume it in almost all cases to be of aortic origin. The next question is, whether it is functional as in children, neurotics, and chlorotics, or due to increased facility of conduction, as by a condensed edge of lung or increased bulk of heart; or is it to be referred to an organic heart disease? Assuming evidence of the last condition present, I should fear that my patient had entered upon the stage of more or less precarious compensation.

It may fairly be asked, how much farther do we get by this view of Skoda's sound than by the older one? To which I answer, that the former theory has never enabled us to recognise mitral disease without the presence of a murmur, nor to predicate moderate compensation unless the signs of non-compensation were absent. It is a link in a vicious circle of reasoning, of which I could give very recent examples. It was deduced *à priori* from the impeded pulmonary circulation, and was offered as a proof that the pulmonary circulation was becoming impeded. Finally, as I have striven to show, it does not respond to facts, and fails to explain accentuation without heart disease.

The aortic theory, on the other hand, gives us a fairly clear conception of the conditions present; it is useful for other cases than those of heart disease, and it is not

inconsistent with facts. It brings some evidence to bear on three important points: the efficiency of the left ventricle, the quantity of blood thrown into the aorta at the systole, and the resiliency of the artery walls. Finally, it does not mislead us into the baseless and dangerous conclusion that the compensatory activity of the right heart can be measured by the intensity of the second sound in the so-called pulmonary area.