stone in it. Examination of the ducts with the forefinger revealed a hard mass in the region of the head of the pancreas. An attempt was made to explore it further with the hope of removing it, but at this point of the operation breathing became somewhat embarrassed and the pulse flagged. Added to this was the difficulty of performing an operation of this nature on an old man with thick abdominal walls. It was therefore thought advisable to make an anastomosis between the gall-bladder and the transverse colon. This was quickly done with the Murphy button, the smallest-sized one being used. The button was passed on the fourteenth day. Convalescence was complicated by a severe attack of catarrhal pneumonia affecting the right lung, and the patient nearly died from the effects of this and the shock of the operation. He finally recovered, however, without further accident.

He was last seen in November, 1899, two years after the operation. His health was perfect. He had never had a pain since the operation. Digestion was good and in every way he was in a normal condition.

The case illustrates the great value of the Murphy button in an emergency of the nature described. The time consumed in adjusting the button was very short, and the result good. It is a question whether the stone has since been passed or whether the artificial passage between the gall-bladder and the large intestine is still serving as a passage for the bile.

SOME POINTS SUGGESTED BY THE CLINICAL STUDY IN ONE HUNDRED AND EIGHTY-SIX CASES OF VALVULAR HEART DISEASE.¹

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I. METHOD OF RECORDING.

ANY one who tries to record statistically a considerable number of cases of any one disease soon meets the following difficulty: Almost any form of chart in which the symptoms are arranged up and down and the patient's numbers across the top (or *vice versa*) soon grows so large as to be unmanageable; the eye grows confused in following the columns, and mistakes easily occur. It is also difficult to get a satisfactory material such as will resist wear and tear.

In recording the cases of which my paper of to-night is a study I have used a book in which points regarding etiology, symptomatology and physical signs are written in a column at the left margin of the first sheet of the book. Then all the other sheets in the book are cut off along their free margin to such an extent that as each fresh page is turned over for the recording of additional cases, it comes in close juxtaposition to without covering the printed column of data in regard to which we are collecting statistics. In this way a series of cases, even up to several hundred, can be recorded in a single book without any of the inconvenience which soon meets us with any form of chart with which I am acquainted, as soon as we get beyond 30 or 40 cases. To record any given point in regard to etiology, symptoms or signs, we need with this book only so much time as is required to make a plus sign or a zero mark opposite the proper word in the printed column which is always

visible and close at hand, owing to the arrangement of the book. I usually stamp a diagram of the front and back of the chest into each record and draw out the areas of the murmurs heard, marking their seat of maximum intensity, as well as the area of the heart.

II. THE APEX BEAT.

(a) When we speak of localizing the apex beat of the heart in order to ascertain the size of the organ, what do we mean, and what ought we to mean? It is well known that it is not the apex of the heart which gives rise to the so-called "apex beat," or cardiac impulse. The impulse is caused by a portion of the right ventricle. In localizing the maximum impulse, therefore, we are not localizing the apex of the heart. What then *shall* we look for? It has been my practice to give the name of "apex beat" to that portion of the chest wall farthest to the left and farthest down at which any rise and fall synchronous with the heart beat can be felt. This is often some distance outside of the *maximum impulse*, and it might easily be objected that what we are localizing by the method here suggested may be simply the outer limit of a *wave* transmitted to the chest wall by the heart. In deciding this question, the evidence of percussion seems to me of great value, and it has been my uniform experience that the percussion dullness begins not at the point where the maximum cardiac impulse is to be seen and felt, but some distance farther out towards the left axilla and farther down towards the base of the chest, in a position corresponding to the lowest and outermost point at which *any* pulsation even very faint, can be felt.

(b) In defining the apex beat, one should be careful so to frame his definition as to include those cases (not at all infrequent in my experience) in which we have a *systolic retraction* at the apex instead of a *systolic impulse*; for I need not remind you that the presence of a *systolic retraction* at the apex is in no way diagnostic of pericardial adhesion, but is often due simply to the negative pressure produced within the chest by the powerful contractions of a hypertrophied heart.

III. THE PLACE OF MAXIMUM INTENSITY FOR AORTIC REGURGITANT MURMURS.

Modern text-books on diseases of the heart and on physical diagnosis are beginning to correct the old misstatements handed down through many of the older books regarding the point at which the aortic regurgitant murmurs are best heard; yet in the "Twentieth Century Practice of Medicine," Whitaker stated in 1895 (Vol. IV, p. 251), "The murmur is heard in greatest intensity in the second interspace to the right of the sternum."

Musser, in his "Medical Diagnosis," page 382, gives three positions, at any one of which the murmur of the aortic regurgitation may be at its maximum. One of these is in the second right interspace and two are in the middle line of the sternum, at different levels. On page 380, he says, "When a murmur is heard with greatest intensity in the second right interspace, it is usually generated at the aortic orifice." On page 429, he admits that "in some instances, the maximum of intensity is greatest at the fourth left costal cartilage, or even at the apex."

Tyson, in his "Physical Diagnosis," page 136, says, "It is sometimes loudest in the aortic area but often

¹ Read at a meeting of the Boston Society for Medical Improvement, November 20, 1899.

over the mid-sternum, and it is even well heard as low as the ensiform cartilage or at the apex itself."

Osler, in the last edition of his "Practice," page 713, says, "On auscultation, there is heard a murmur, during diastole, in the second right interspace, which is propagated with intensity towards the ensiform cartilage, or down the left margin of the sternum towards the apex."

These statements are a considerable improvement over those of the older text-books, in which the occurrence of aortic murmurs with maximum to the left of the sternum is hardly mentioned, but I do not think that even the statements just quoted give a true idea of the facts, for they seem to suggest that the right side of the sternum is the *common* place to hear the murmur best, and the left side the *rarer* place. In 44 cases which I have carefully examined this summer with reference to this point the murmur was best heard to the left of the sternum in 36; best heard to the right of the sternum in four cases; in the centre of the sternum in two cases; while in two more there were two areas of equal intensity, one at the right and one at the left of the sternum; while over the sternum itself, the murmur was very feeble. In this series, which I believe to be a typical one, the murmur was best heard to the right of the sternum in only four out of 44 cases, or less than one-tenth.

IV.

My fourth point I want to put simply in the form of a question: Upon what evidence should we base the diagnosis of aortic stenosis? Knowing, as we do, that a loud systolic murmur in the traditional aortic area may be produced by simple roughening of the aortic valves or the aorta itself, without any genuine stenosis, and that this murmur may be propagated into the great vessels, what positive signs have we for distinguishing aortic roughening from aortic stenosis? Is the presence of a tactile thrill, in addition to the signs just mentioned, sufficient to warrant in making the diagnosis of mitral stenosis, or do we need in addition the *pulsus tardus, rarus, parvus* of the classic descriptions?

V. RELATIONS BETWEEN THE VALVE LESIONS AND HYPERTROPHY.

The study of the 186 cases of this series has given me, I think, reason to deny that there is any one form of valve lesion which is constantly associated with the clinical evidence either of hypertrophy or of the lack of hypertrophy of either ventricle. The classical textbook statements, that in pure mitral stenosis we have no hypertrophy of the left ventricle; that in mitral regurgitation evidence of a predominant or exclusive enlargement of the right ventricle can be made out; that in aortic regurgitation the left ventricle must be enlarged, all these seem to me contrary to the facts of observation. I have seen several cases of well-marked aortic regurgitation without any evidence of hypertrophy of the left ventricle, and several of apparently pure mitral stenosis in which there was every reason to suppose that the left ventricle was enlarged, while in a majority of cases of mitral regurgitation such enlargement of the heart as could be demonstrated seemed to affect the left ventricle rather than the right. In connection with this, I would like to call attention to the worthlessness of epigastric pulsation as evidence of hypertrophy of the right ventricle.

Surely we have all seen it present when the heart was normal, both during life and at autopsy, and, on the other hand, seen convincing evidence of hypertrophy of the right ventricle without any epigastric pulsation.

VI. PRESYSTOLIC MURMURS IN CONNECTION WITH AORTIC REGURGITATION.

It is a well-known fact, first described by Austin Flint in 1862, that we may have every physical sign of mitral stenosis, and yet at autopsy find the mitral valve normal in cases where there is an associated regurgitation through the aortic valve. I mention this fact simply to call attention to two points: (a) First, that unless we always have it in mind and are closely on the lookout for it, we may altogether overlook the aortic lesion. In certain cases the diastolic murmur is very faint and the presystolic roll and thrill at the apex impress themselves upon one's mind to the exclusion of everything else. The importance of not making this mistake, that is, the importance of recognizing a faint diastolic murmur in the presence of a loud presystolic roll and thrill, is that the prognosis of a combined lesion of both the aortic and mitral valves gives a much worse prognosis than a lesion of the mitral valve alone. (b) Second, I want to ask if any one here present can tell me whether it is ever justifiable to make a diagnosis of mitral stenosis in a case where *we are sure there is aortic regurgitation*. Is there any way, in such a case, in which we can tell the Austin Flint murmur from the murmur due to organic changes in the mitral valve?

EPILEPSY.¹

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THE symptom complex of epilepsy falls within the experience of every physician, and the diagnosis, prognosis and treatment of this affection are most important from the practical standpoint. The class of cases here considered belongs to so-called idiopathic epilepsy, and not to the cases in which the convulsive attacks are symptomatic. For although in many cases of epilepsy pathological cerebral changes have been found, by no means is there associated with epilepsy any constant pathology. Further study of epilepsy may demonstrate a uniform etiology, but if due principally to a disordered function of conducting paths, the etiology would vary widely in individual cases, and might well be beyond discovery. Féré² says of epilepsy, that "the most varied causes produce the same clinical phenomena."

In a general way it seems unlikely that a special toxin or class of toxins is solely, or even in large measure, the one factor causing epileptic seizures. It is reasonable to grant that all of the human kind elaborate the same chemical substances in the economy and that the central nervous system of every individual is subjected to practically the same metabolic processes and products. But there is a wide variation in neural endowment. Is not the cause of epilepsy almost absolutely in the machinery of the brain and cord—a vice of the nerve tissues, a limited

¹ Read at a meeting of the Boston Society for Medical Improvement, November 20, 1899.

² Féré: *Twentieth Century Practice*, vol. x, p. 586.