

which we can obviate its effects. The continued use of mercury in small doses, I believe, may influence the elimination of this poison. As for the iodids, I am convinced that those of sodium and potassium are the preferable salts; they are better borne and have a less disturbing influence upon the heart. The mechanical treatment, I am convinced, is the treatment of the future for this disease. It should be given when the disease is forming.

DR. EDWARD F. WELLS of Chicago.—I wish to speak briefly upon a single point, that is, the reduction of blood-pressure. In interstitial nephritis there is an increase in the blood-pressure and a hypertrophy of the heart. This increase in blood-pressure causes a thickening of the arteries, the cardiac hypertrophy increases more and more, and if the patient takes some amount of active labor it is necessary that his nutrition should be kept up. The first thing to do is to stop any excessive amount of exercise he may be taking, and limit the amount of nitrogenous food, and I believe that a careful regime, such as the avoidance of draughts, regulation of his diet, the giving of large amounts of fluids to reduce the amount of poison in his system, will do vastly more good toward reducing the blood-pressure and relieving the patient and prolonging his life than any drugs. I do not wish to be understood as deprecating the use of drugs; I believe that the iodids are the drugs to be used. We should not lose sight of the careful regulation of the diet.

DR. JENKINS of Iowa.—I am very much pleased with Dr. Elliott's paper, and I wish to emphasize one portion of it regarding the use of nitroglycerin in cases of chronic interstitial nephritis. I have frequently had patients come to me with this disease for treatment, coming from other physicians, who had failed to try this remedy. Placing them on the use of strychnin, regulating their diet, and giving nitroglycerin relieved their symptoms and gave them many months of comfortable living. For this reason I am glad Dr. Elliott emphasized that portion of his paper. The disease has not been well treated until recently. Now it is, with careful bathing and dressing, dieting, regulation of bowels, with the administration of such vasomotor dilators as glonoin, one drop every two, three or four hours, and up to five drops, that benefit is to be derived.

DR. ARTHUR R. ELLIOTT, closing the discussion.—I wish to express my pleasure at the amount of discussion my paper called forth. I think cases of interstitial nephritis cannot be classified as all belonging to the cardiac type, any more than they can be said to belong to the purely nephritic type. The fibrosis is a general one affecting different portions of the body in different degrees. I would like to state that in the preparation of my paper I was influenced in setting forth measures of direct treatment against the disease in the later stages, i. e., when cardiac hypertrophy was present or failure threatened. I did not enter into any discussion of the early stages of the disease. If we could get hold of the disease in the earliest stages I think that hygienic, dietetic and other measures would give better results than could be obtained through the use of drugs. The cardiovascular changes of nephritis are Nature's protest against the results of imperfect metabolism in the blood-stream.

### SYSTOLIC MITRAL MURMURS.\*

THEIR TRANSMISSION, WITH SPECIAL REFERENCE TO THE NATURE OF THE SO-CALLED ANEMIC MURMURS.

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What is familiar to us is not necessarily well understood, and yet in medicine as much as in any branch of learning we are to accept this feeling of familiarity for knowledge. This thought must be my excuse, if one is necessary, for bringing to your attention a subject so familiar as the heart murmurs.

The question whether a systolic murmur is organic or functional is being constantly brought up for our decision, yet although it has been discussed many times there is still lacking that unanimity of opinion among the best authorities which would assure us that it has been definitely settled. The question is one of practical importance, and any addition to our knowledge, however small,

should be welcome in our efforts to find a solution.

A review of the literature shows that the discussion has turned chiefly on the points of maximum intensity of these murmurs. The second left intercostal space has been the battle-field of the champions. Whether the point of maximum intensity was just over the pulmonary artery or a little outside of it has been the burning question. So many theories have been advanced to explain the murmurs in this region that Balfour<sup>1</sup> has well called it the "region of romance" for cardiac murmurs. In all this discussion the area of transmission of these murmurs has received little attention, although it seems to the writer to be of considerable importance. In fact, the method of conduction of the sounds and murmurs of the heart to the surface of the chest, and the area over which they may be heard have only of late received much attention, and we have still much to learn of these important and fundamental matters.

Gibson<sup>2</sup> in his recent book says: "We are by no means thoroughly acquainted with the physical facts which regulate the extent to which the sounds are conducted." And again: "The direction of propagation and the extent of conduction constitute a subject of much difficulty in the study of murmurs."

When a murmur is heard at a given point on the chest, one fact at least is true, namely, that there exist at this point vibrations of the chest wall capable of transmission to the ear and of such a character and intensity that they produce the sensation of sound. How these vibrations originate, where they start, and how they reach the points at which they are heard, are questions less definitely determined—questions of probability rather than fact, which still come to some extent within the realm of opinion and theory. It is important to thus recognize when we cross the boundary separating fact from theory.

Clinically, we find that these vibrations reach the chest wall at certain areas; experimentally, we find that such vibrations may be caused by certain physical conditions; pathologically, we find these conditions existing at certain points in the heart and blood-vessels. In this way we can demonstrate the cause of certain murmurs. For example, we know that mitral regurgitation may cause vibrations which are heard as a systolic murmur near the apex of the heart. These vibrations are also heard in other areas, as will be considered later. It is worth while noting at this point, however, that the so-called anemic or functional murmurs frequently occur in trivial disturbances of health, are often transitory, and are rarely so associated with fatal disease as to give us the opportunity to definitely determine their cause by pathologic investigation. Our explanation of many of them is, therefore, based on inference, and carries a greater or less degree of probability rather than certainty.

It is generally accepted that mitral murmurs are heard best near the apex of the heart, and we commonly speak of this place as the "mitral area." The term is a good one, provided we do not assume that all murmurs heard in this area are generated at the mitral valve. It is not very uncommon to find a systolic murmur from the aortic valve transmitted to this point, and occasionally a presystolic murmur in this area finds its only explanation in aortic insufficiency. The vital point in all these cases is that the vibrations are carried here through the medium of the wall of the left ventricle and the blood within its cavity. This fact should be kept in mind when we speak of the "mitral area." In the same way it requires very little study of the distribution of murmurs to show that it is not safe to argue that because a murmur is heard at the aortic pulmonic, or tricuspid

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area, it necessarily originates at the valve with a corresponding name.

It is useful, in the ordinary examination of the heart, to keep these four valve "areas" in mind, but in a careful study of murmurs it is better to picture the anatomical relations of the normal heart and blood-vessels to the chest wall. If we will modify this picture to suit the given case, as shown by the evidence of inspection, palpation and percussion; if we further bear in mind the area and consistency of the lungs, the thickness of the chest wall, and the bony or muscular nature of the chest wall at a given place, then, and not till then, we are prepared to interpret intelligently the conditions which determine the distribution of cardiac murmurs. In this way we shall find an explanation of many things which at first seem confusing and obscure.

Continuing our study of the mitral regurgitant murmur, it is generally stated that it is transmitted to the left from the "mitral area" into the axilla and may be heard at the angle of the scapula. Gibson's<sup>3</sup> description is, in my opinion, more accurate. He says: "It is propagated from this region in every direction, but to a different extent it is conducted to a greater distance in the direction of the axilla and scapula." Again: "Very frequently the murmur is found to have a position of intensity almost as great as that over the apex-beat at a point between the left shoulder-blade and the vertebral column, and there can not be the shadow of a doubt that the conduction of the murmur to this point is due to the proximity of the left auricle."

This area in the back just described, which we may call the mitral area in the back, has received little consideration in most text-books. They seldom attempt to explain how the murmur reaches this point and apparently leave one to infer that it is transmitted around the chest-wall from the apex. This view is wrong, for it would not account for those cases referred to by Gibson in which the intensity at the mitral area in the back is "almost as great as at the apex," and greater than in the intervening space in the axilla. Still less will it account for those cases in which the murmur is transmitted as far as the axilla and is then lost, and yet reappears at the mitral area in the back. And, further, we sometimes have cases in which the murmur is actually louder in the back than at the apex. The explanation is undoubtedly found, as Gibson says, in the "proximity of the left auricle."

From our association of the mitral murmurs with the apex of the heart, which lies superficially, we are apt to forget how deep in the chest the murmur originates. The mitral valve is deeply situated, the left auricle more so. It is the blood passing through the mitral valve into the auricle which causes the murmur, and, except so far as the vibration of the valve curtains partake in the production of the murmur, it originates in the blood within the auricular cavity. It is a safe assumption then, that at the posterior surface of the auricle vibrations of considerable intensity exist. How do they reach the back?

Between the left auricle and the vertebral column lie the aorta, esophagus, vena azygos, and surrounding tissues of the posterior mediastinum. Although the distance to the spine is short, the vibrations do not traverse this path ordinarily, and we must conclude that these structures are not good conductors. Were the vibrations thus carried to the spine they would be heard with greatest intensity where the bony structures of the spine come nearest to the surface. This is not the case. The mitral area of the back, where the murmur is most constantly heard and with the greatest intensity, lies a short distance inside the inner border of the left scapula and a

short distance above its lower angle. This is the point where there is the thinnest layer of muscle in this part of the back. It lies just outside the deep longitudinal muscles along the spine, while it occupies a triangular interspace left by the trapezius, the latissimus dorsi, and the rhomboideus major muscles. Having ruled out the spine, the only medium of conduction from the left auricle to this thin part of the chest wall is the pulmonary tissue.

It is generally held that the pulmonary tissue offers an impediment to the transmission of vibrations from the heart, rather than a favorable avenue of conduction. This is certainly true in a relative sense, as compared with such avenues of conduction as the muscular walls of the heart or the column of blood within the heart or aorta, but it is true only in this relative sense. Furthermore, the extent to which it is true depends on the condition of the pulmonary tissue. If that is condensed or consolidated, it is recognized as a very good conductor of vibrations. On the other hand, if emphysema, the opposite condition to condensation—is present, we have a very poor conductor of vibrations, and it may be noted in passing that with an emphysematous condition of the lung a mitral murmur is inaudible or only very faintly heard in the back. Between these two extremes lies the condition of the normal pulmonary tissue, which, I think, from all the evidence, we must accept as a fairly good conductor of vibrations from the heart. This, at least, is an opinion into which I have been forced in trying to account for many of the peculiarities of the distribution of heart murmurs, and it seems the only rational way to account for the transmission of the mitral regurgitant murmur to the mitral area in the back.

Turning now to the front of the chest, let us consider how the conduction of vibrations by the normal lung tissue may affect the distribution of the murmur there. Does it not give us a more rational explanation of the characteristic distribution of the murmur in front? As Gibson says: "It is propagated in every direction, but to a greater extent in the direction of the axilla." Were it transmitted from the apex alone, and solely by the chest wall, we should expect it to be transmitted best and farthest in the direction in which there is the best chance for bone conduction, and this is to the right and not to the left of the apex. If, however, we accept the view that the vibrations may be carried a moderate distance by the lung tissue, we have an easy explanation of the transmission to the left of the apex, for the distance from the vibrating left border of the auricle and ventricle to the chest wall as it rounds back into the axilla is only a short one. It is not claimed that there is no lateral transmission of murmurs by the chest wall itself, for that certainly exists, but it is felt that too much stress has been laid on this factor, and that, taken alone, it does not fully explain the phenomena.

A consideration of the thickness of the chest wall in front will perhaps give us an explanation of the common view that the murmur is transmitted to the left from the *apex* instead of, as is the fact, from the whole left border of the heart. The pectoral muscles form a thick layer and offer more or less of an impediment to the transmission of vibrations to the surface over them. Outside the boundaries of the pectoral muscles the murmur is heard more distinctly because the chest wall is thinner, and this gives us an area which extends along the border of the pectoral muscle from the apex to the left and up into the axilla.

The removal of the obstruction of the pectoral muscles throws interesting light on the subject. It is easily accomplished. If the left arm is raised upward and is then laid across the face so that the forearm and elbow

lie across the eyes, the pectoral muscles are relaxed and may then be pushed inward toward the median line far enough to allow the bell of the stethoscope to be applied to the chest wall at or just outside the mammary line. We are now listening just outside the left border of the heart, with a chest wall as thin as it is farther down at the apex. This maneuver is best accomplished with the patient lying on the back, as better relaxation of the muscles is thus obtained. In females the breast may usually be carried out of the way in the same manner.

In this way we find not only that the murmur is audible all along the left border of the heart but that its intensity is greater at a given distance up from the apex than it is at the same distance to the left of the apex. Not infrequently the murmur is louder farther up the left border of the heart than at the apex itself. A very common place to hear the murmur loudest is in the third interspace. This is easily understood if we remember that we are now listening just opposite the mitral valve, the center of the left auricle, and the thickest part of the left ventricle—in other words, near the place where the vibrations should exist with greatest intensity. If we admit that the lung tissue is a fair conductor of vibrations, we readily explain why the murmur is heard so loud in the third interspace near the left border of the heart. Whether the murmur is heard loudest here or at the apex depends largely on the relative conducting power of the pulmonary tissue and the wall of the left ventricle in a given case.

We have followed the area of transmission of the mitral regurgitant murmur up the left border of the heart to the third interspace. In many cases it does not stop here, but we find the area extends still farther into the second interspace. Here we reach Naunyn's area and then the pulmonary area. I believe the medium of conduction here is the same as a little farther down, namely, from the upper part of the left auricle and the base of the left ventricle through the pulmonary tissue to the chest wall. It is quite possible that the left auricular appendix plays a part in the conduction of the murmur to this point, for it is the part of the left auricle which comes nearest to the surface of the chest. But by our explanation it is no longer necessary to assume that this appendix must touch the chest wall, or even that it must reach the anterior surface of the heart in order to have the murmur conducted to the second left interspace. The objection of Russell and his followers thus falls to the ground, while the conclusion of Naunyn and Balfour that this murmur may be of mitral origin stands, although their explanation that the left auricular appendix alone is the medium of conduction is, as Russell shows, "not proven."

Turning now from the conduction of the mitral systolic murmur beyond the area of the heart, we have still one area to consider over the heart itself. This may be called the "valvular area." It is about the junction of the third left costal cartilage and interspace with the sternum. Here we are directly over the anatomical position of the valves as they lie close together. It would seem almost superfluous to recall the fact that it is at or close to the valves beneath this area that all valvular murmurs originate, were it not that many very excellent text-books are careless about this matter, and say, for example, that the aortic systolic murmur originates at the aortic area. The chief medium of conduction from the valves to the front of the chest is the thick muscular walls at the base of the ventricles, in which the valves are imbedded. The path for conduction is direct, the distance is not great, and, as a result, any valvular murmur can as a rule be heard at this area and fre-

quently is louder and more distinct than at the more distant area corresponding to the valve at which it is generated.

We are not accustomed to pay so much attention to this "valvular area" because the fact that a murmur is heard here gives us little help in determining which valve is affected. Nevertheless, in studying the distribution of a given murmur this area should receive attention, for the murmur is sometimes heard here with marked intensity. This is true of mitral murmurs as well as others. Between this "valvular area" and the apex we find the mitral murmur transmitted with greater or less intensity according to the condition of the left ventricular wall beneath and the character of the lung and chest wall overlying the heart at a given point.

We are now prepared to consider the transmission of the mitral systolic murmur farther upward at the base of the heart and to the "aortic area" so-called. We have just seen that the vibrations are strong in the thick muscular base of the left ventricle. At this point the aortic begins and, remembering also the close connection of the aortic and mitral valves as well as the fact that during systole there is a continuous column of blood from the ventricle into the aorta, with the current flowing into the aorta, we should not be surprised to find the mitral systolic murmur carried into the aorta and audible at the aortic area. That it is thus carried to the aortic area may be frequently demonstrated, though I am not prepared to say how far it may be transmitted through the arteries beyond. The fact that mitral murmurs may be thus heard at the base and the mechanism by which this is brought about should be kept in mind when studying functional murmurs.

Mitral murmurs are frequently conducted over the chest far beyond the areas which we have considered, and the total area may include the whole chest. It is enough to say that the whole area of distribution depends on the intensity of the vibrations and the character of the conducting media. A further consideration of this topic is not necessary for the purposes of this paper.

We now come to the consideration of the so-called "anemic" murmurs. To say these murmurs are due to anemia to state a fact but to give no explanation of that fact. They are ordinarily included under the head of functional murmurs, indicating that they are not due to a pathologic condition of the valves or wall of the heart. This is not strictly true, for it is generally recognized that in some conditions of anemia the heart becomes dilated and the character of the murmur indicates valvular insufficiency as clearly as in organic disease. We shall consider only those murmurs which are heard in the second left intercostal space.

They are ordinarily ascribed to the pulmonary artery because they are heard in the "pulmonary" area. Since no lesions of the pulmonary artery are found, they are attributed to the condition of the blood itself. Is this interpretation correct? It certainly has not passed without challenge.

It is not my purpose to thoroughly review the discussion of this subject, but merely to mention a few explanations by well-known authorities to show that we need not feel bound to the view that the murmur originates in the pulmonary artery. Naunyn and Balfour contend that the murmur comes from the mitral valve. Russell asserts that it reaches the surface from the pulmonary artery. He does not, however, fall back on the state of the blood for an explanation, but thinks it might be caused by a dilated left auricle pressing upon and narrowing the lumen of the pulmonary artery, or that it might be a tricuspid regurgitant murmur transmitted

up the conus arteriosus. Gibson also attributes the murmur to tricuspid incompetence. Sansom thinks the murmur is due to fibrillary tremor of the muscle of the conus arteriosus below the pulmonary valves.

Let us turn to the testimony of the murmurs themselves. I hold with those writers who state that the most common site for anemic murmur is at the base of the heart, especially in the second left interspace. But although the point of maximum intensity is often here, a study of the area of distribution shows it to be comparatively rare to find the murmur limited to this area. By moving the pectoral muscle out of the way, as previously described, the murmur will very commonly be found to be transmitted around the left border of the heart from the second into the third and sometimes into the fourth interspace. In quite a large number of cases the murmur has as great or even a greater intensity in the third than in the second interspace. This at once recalls the conditions we found while studying the mitral murmur and it immediately raises a difficulty in accounting for our murmur, on the supposition that it originates in the pulmonary artery or at the tricuspid valve.

The similarity to a mitral murmur is increased when we find the murmur transmitted still farther to the apex of the heart. And when we find it also transmitted to the mitral area in the back, the conclusion becomes inevitable that we have to deal with a murmur of mitral insufficiency. That mitral regurgitation may occur in some conditions of anemia is recognized by all writers. The only new thing in this study is the demonstration of a continuous series of transition stages among the anemic murmurs from a location limited to the pulmonary area to that of a characteristic murmur of mitral regurgitation. May we not be dealing with the same murmur in all these stages? If not, where shall we draw the line between the anemic murmur due to mitral regurgitation and one due to some other cause?

Of all locations in which the murmur is heard, that at the mitral area in the back is the one showing most conclusively that it is a mitral murmur, and this is also the area which throws the greatest difficulties in the way of accounting for the murmurs at any other orifice. Using this area in the back as a test, I was surprised to find that the murmur was audible here in many cases in which it did not reach the apex. In fact, I have repeatedly heard the murmur in the back when in front it was only to be heard in the second left interspace and faintly in the third interspace. In some of these cases it was fully as loud in the back as in the front, in others it was only faint and distant, but still distinct. These observations I have frequently had verified by others so as to eliminate the possible element of enthusiasm and expectancy which leads one to hear what he wishes to hear, whether it is present or not. The results of the observations on a large number of cases carried on for several months may be summarized by saying that careful observation will show a systolic murmur at the mitral area in the back in a surprisingly large proportion of the anemic murmurs, even when they are not heard at the apex. This is not a constant accompaniment of the anemic murmur, neither is it of the organic mitral murmur, for reasons before stated. It should be added that in many of the cases, including practically all cases in which the murmur failed to reach the apex, there was no demonstrable dilatation of the heart. If we accept these data as correct, we are inevitably forced to the conclusion that many of the so-called "anemic" murmurs are of mitral origin, whether they reach the mitral area at the apex or not.

It may be urged that I have simply reported a series of cases which apparently grade into one another, and that it is a mere assumption on my part that they have a common origin. What proof is there that we are dealing with the same underlying cause through these various gradations?

This proof is to be found by watching the same case pass through the gradations from one stage to another. In a number of cases which have started with an "anemic" murmur heard in the second left intercostal space, extending around the left border of the heart to the apex, and heard also in the back, the area of distribution has diminished as progress has been made toward recovery. There has been left only a typical "anemic" murmur in the second left interspace, and in favorable cases this also has disappeared. The order of disappearance has not always been the same. Sometimes it has disappeared first in the back, less often first at the apex. In front we sometimes find a gradual recession of the murmur from the apex toward the base, more often we find the area splitting into an area at the base and one at the apex, and generally the area at the apex disappears before the one at the base.

It may still be urged that our murmur may be a composite one, that we have, to be sure, in some cases a mitral murmur but combined with it an anemic murmur at the pulmonary orifice, and that the two verge into one another. I should meet this objection first by citing those cases in which an apparently typical "anemic" murmur at the base is accompanied by a murmur in the back, but by no other evidence of mitral regurgitation. And then, as confirmatory evidence that murmurs in the second left interspace only may have the same origin as typical mitral murmurs, I will record some observations made upon long-distance runners.

On April 19 last I had the pleasure of assisting Dr. Harold Williams of Boston in an investigation of the effects of long-distance running carried out on the contestants in a Marathon race. This is a 25-mile road race, modeled after the course run by the ancient Greek who carried the news of victory from Marathon to Athens. The results of the observations were embodied in a paper read by Dr. Williams before the last meeting of the American Climatological Association.\* We need consider here only the effect upon the heart in causing murmurs.

The course of 25 miles was run by the winner in 2 hours, 54 minutes, 38 seconds—a little better than a mile in seven minutes. During the next hour twelve other contestants finished. These men were all healthy young adults. They were examined both before and after the race. The hearts of all were normal before the race, with the exception of a varying degree of physiologic hypertrophy due to exercise. No murmurs were heard before the race. The general effect upon the men may be described as one of extreme muscular exhaustion.

Here we had a number of human beings voluntarily submitting themselves to an experiment in which we could learn the result of pure muscular exhaustion of the cardiac muscle in healthy hearts. The exhaustion of the cardiac muscle was shown by a change from strong to weak action by lowered blood-pressure, as shown by sphygmographic tracings, by rapidity of action, and by a perceptible dilatation or distension of the heart.

Of the 13 men examined after the race, 11 had developed a cardiac murmur. The murmur was in all instances systolic. In every instance it was heard in the second left intercostal space. In two cases it was confined to this space. In three others it reached along the left border of the heart to the third or fourth interspace.

In the other six it was continued beyond this area to the apex, was transmitted a short distance to the left of the apex, and was audible at the mitral area in the back. In all cases the murmur was temporary and disappeared as soon as the heart had a little time to rest. It is also interesting to note that the two men who did not have murmurs were the winners, whose hearts were unusually strong, and better adapted to withstand the strain.

I think we are justified in assuming that the underlying cause of the murmur was the same in all these cases and was the weakness of the cardiac muscle from exhaustion. We may also assume that the mechanism of the production of the murmur was the same in all instances. Since, then, in the more characteristically marked cases it was a mitral regurgitant murmur we must assume that it was due to mitral regurgitation in all cases. Hence we have demonstrated by this experiment, first, that weakness of the cardiac muscle may cause mitral regurgitation, and second that a murmur thus due to cardiac weakness may exhibit all the gradations from what would ordinarily be accepted as a hemic murmur at the base to an undoubted mitral murmur at the apex.

It only remains now to show that weakness of the cardiac muscle exists in anemia, then our proof is complete that the anemic murmur is not due directly to the condition of the blood itself, but to a resultant mitral insufficiency. In the fatal cases of anemia the cardiac muscle is frequently found in a state of fatty degeneration, due to insufficient nourishment by the impoverished blood. Dilatation may or may not exist. It is inconceivable that the myocardium should remain perfectly normal through all the earlier stages of anemia and at a given point suddenly develop fatty degeneration. Various stages of impaired nutrition undoubtedly precede this. Impaired nutrition implies weakened muscular action. The mechanical results of a weakened myocardium will be the same whether the weakness is caused by poor nutrition or by tire from overexertion, as in the runners. In the runners we got mitral regurgitation, in the weak heart of anemia we should have the same condition.

It has been too often assumed that we cannot have mitral regurgitation unless we have actual dilatation of the cavity of the ventricle. Were this true, it would constitute a serious objection to our explanation of anemic murmurs, for such dilatation does not always exist. The studies of Ludwig and Hesse, however, together with the experiments of Roy and Adami, show that the complete closure of the mitral valve involves a much more complex mechanism than does the closure of the semilunar valves at the arterial orifices. The exact apposition of the mitral flaps is secured not alone by the force of the blood-pressure bringing the flaps toward each other, but by muscular contraction of the ventricular wall in narrowing the orifice and by the co-ordinated action of the papillary muscles in properly staying the flaps and preventing them from being forced too far. It is unnecessary to discuss here just what proportion of the proper adjustment is due to the muscle of the wall of the ventricle or to the papillary muscles. Probably a sufficient weakening of either force might allow regurgitation. In anemia both are probably affected, and the weakening of the muscle in anemia gives an adequate cause for mitral regurgitation, whether dilatation exists or not.

We may summarize our studies in this paper as follows:

Mitral systolic murmurs may not only be heard at the mitral area at the apex and extending toward the axilla,

but also at the mitral area in the back, along the left border of the heart, in the second left interspace, at the base, and at the "valvular" area.

Anemic murmurs are not confined alone to the pulmonary area and second left interspace. They extend by varying gradations around the left border of the heart, to the apex, and to the mitral area in the back, thus gradually assuming the characteristics of a true mitral regurgitant murmur. The transmission to the mitral area in the back may exist, whether the murmur is heard at the apex or not, and even in what appear in front to be typical anemic murmurs.

We find a dividing line between the so-called anemic murmurs and the mitral murmurs.

The presumption that these different gradations in the distribution of the murmur all have a common origin is strengthened by finding these different gradations successively in the different stages of the same case. It is practically proved by finding all these gradations in healthy hearts which have been subjected to the same severe exhausting strain, as in the runners mentioned.

Weakened muscular action of the heart exists in anemia as a result of poor nutrition. Weakened muscular action is an adequate cause for mitral insufficiency, whether dilatation exists or not.

The points which are new in this demonstration are the study of the area of distribution of these murmurs rather than the point of maximum intensity, the demonstration of the transition stages between the anemic murmurs and the mitral regurgitant murmurs, the demonstration of the frequency with which anemic murmurs are heard in the back, and the importance placed upon the transmission of murmurs by pulmonary tissue.

Other observers have noted that an anemic murmur is sometimes transmitted to the mitral area in the back, although they apparently have not appreciated the full significance of the fact. Barr<sup>5</sup> reports that in 115 cases of simple chlorosis he found the murmur in the back as well as at the base and apex in 22 cases. He does not specify whether dilatation existed or not, and he mentions no cases where the murmur was heard in the back and at the base, but was absent at the apex.

Sansom<sup>6</sup> says: "I have frequently noted a systolic murmur audible at the angle of the left scapula in uncomplicated anemia. In fact the murmur in anemia can answer to all the criteria of one due to regurgitation from organic causes. Such murmurs can be experimentally induced in animals by copious bleedings." He also quotes Macalister<sup>7</sup> "When an animal is bled till it is feeble, a murmur indicating regurgitation from the ventricle is heard with the heart sounds. You may inject proper saline solution to make up the normal quantity of circulating fluid, but still the regurgitation occurs. As the animal makes blood again, so that its muscles are again properly nourished, the murmur disappears."

Prince<sup>8</sup> and McCollom<sup>9</sup> have made interesting observations on the hearts of healthy men, examined for positions as firemen or policemen, where the murmurs were apparently due to excitement and were temporary. The murmurs were ascribed to mitral regurgitation due to imperfect action of the cardiac muscle. The murmur is similar to that found in the long-distance runners, already cited. The point in common in these cases of excited action, in the exhausted hearts of the runners, and in the anemic hearts is the failure of the muscle to do its part toward the closure of the mitral orifice. Prince found a systolic mitral murmur in 25 of 77 healthy men, and McCollom in 27 of 200 men. In 8 of the 25 cases noted by Prince, the murmur was heard also in the sec-



and left intercostal space "and sometimes can be traced upward to this point from the apex." In a number of cases in his table he notes that this area was not examined. "Sometimes the murmurs are heard in the back beneath the scapula—this was not always looked for." "They are sometimes heard equally loud at the junction of the fourth rib and sternum on the left side, i. e., over the mitral valve."

In reviewing the literature on this subject, it is not my purpose to give a complete bibliography—such a bibliography might well include all works which have been published on the heart. The following works have been of assistance in studying the subject:

DISCUSSION AS TO THE NATURE OF ANEMIC MURMURS AND REVIEWS OF THE VARIOUS THEORIES.

- Naunyn: Berlin Klin. Woch., 1868, s. 189.  
 Balfour: Clinical Lectures on Diseases of the Heart and Aorta, 3d ed. 1898. Lectures vi and viii. Lancet, London, 1877, vol. ii, p. 383. Edinburgh Med. Jour., vol. xxviii, pp. 183, 289.  
 Russell: Edinburgh Med. Jour., 1882, vol. xxviii, pp. 130, 403. British Med. Jour., 1883, vol. i, p. 1053. Investigations into Some Morbid Cardiac Conditions, Edinburgh, 1886, pp. 35, 53, 56, 68.  
 Bramwell: Diseases of the Heart and Thoracic Aorta, Edinburgh, 1884, pp. 187, 207. A Lecture on the Functional Cardiac Murmurs of Anemia. British Med. Jour., 1883, vol. i, p. 1213.  
 Sansom: The Diagnosis of Diseases of the Heart and Thoracic Aorta, London, 1892, chapters xxxv, xxxvi, xliii, xlv.  
 Gibson: Diseases of the Heart and Aorta, 1898. (Cardiac Weakness), pp. 627-634. (Mitral Regurgitant Murmurs), p. 546. (Conduction of Murmurs), pp. 166, 171, 172.  
 Ball: Some Remarks on the Acoustic Phenomena Produced by the Flow of Fluids in Tubes, and also upon the Site and Mechanism of Cardiac Functional Murmurs. N. Y. Med. Record, 1884, vol. xxv, pp. 383-398.  
 Garland: Theories Regarding the Mechanism of the Inorganic Cardiac Murmurs. Boston Med. and Surg. Jour., vol. cix, pp. 25-27.  
 Shattuck, F. C.: The Diagnosis of the So-called "Functional Murmurs." Boston Med. and Surg. Jour., 1883, vol. cix, pp. 28-30.

ANATOMICAL RELATIONS.

- Savory: Observations on the Structure and Connections of the Valves of the Human Heart. Lancet, 1852, vol. ii, p. 420.  
 Sibson: Reynold's System of Medicine.  
 Bramwell: Diseases of the Heart and Thoracic Aorta.  
 Keiller: Descriptive Anatomy of the Human Heart. Am. Jour. Med. Sci., 1898, vol. cxv, pp. 428-436.

SINGLE TOPICS.

- Tyndall: Lectures on Sound, 1867.  
 Ludwig and Hesse: Beiträge zur Mechanik der Herzbewegung, Archiv für Anat. und Phys., 1880, p. 320.  
 Macalister: Remarks on the Form and Mechanism of the Heart. British Med. Jour., 1882, vol. ii, p. 821.  
 Roy and Adams: Remarks on the Failure of the Heart from Overstrain. British Med. Jour., vol. ii, p. 1321.  
 Fenwick and Overend: Report on the Contraction of the Papillary Muscles in its Relation to the Production of Certain Abnormal Cardiac Sounds. British Med. Jour., May 23, 1891.  
 Heitler: Tricuspidalgieräusche: Localisation des Systolischen Mitralgeräusches. Wien. Klin. Woch., 1897, No. 7, pp. 161-165.  
 Ewart: Clinical Lecture on Heart Sounds and on Accuracy in Cardiac Auscultation. Lancet, London, 1893, vol. i, pp. 1241-1246. Note on the Auscultation of the Second Sounds of the Heart. Lancet, London, 1894, vol. ii, pp. 789-791.  
 Ringer and Phear: The Clinical Significance of Accentuated Second Sound. Lancet, London, 1894, vol. ii, p. 729.

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1. Clin. Lect. on Diseases of the Heart and Aorta, 1898, p. 217.
2. Diseases of the Heart and Aorta, 1898, p. 165.
3. Ibid, p. 546.
4. Also reported in Phila. Med. Jour., June 3, 1899.
5. Barr, Alfred G.: Clinical Observations on the Cardiac Bruits in Chloroses. Am. Jour. Med. Sci., 1891, vol. cii, p. 347.
6. Sansom, A. E.: Diagnosis of Diseases of the Heart and Thoracic Aorta, 1892, p. 341.
7. Macalister: Remarks on the Form and Mechanism of the Heart. British Med. Jour., 1882, vol. ii, p. 825.
8. Prince, Morton: N. Y. Med. Record, 1889, vol. xxxv, p. 421.
9. McCollom, J. H.: Boston Med. and Surg. Jour., 1889, vol. cxx, p. 103.

DISCUSSION.

DR. NORMAN BRIDGE of Los Angeles.—I do not know but Dr. Arnold, in that part of his paper which time did not permit him to read, may have referred to the point I have in mind. But as I recall it he took no account of the possibility of the air in the bronchi being capable of transmitting the heart murmurs to the back. I do not recall any one having referred to that. It may not be a matter of consequence, but it seems to me that, in auscultation of lung troubles and diseases and disorders of the pleura, we should and do take into account this element largely, and find it extremely useful from the standpoint of diagnosis, proving that sound, voice and air are transmitted through the air in the bronchi to the walls of the

chest; variations of these sounds so transmitted are matters of great moment in the diagnosis; and it seems to me that heart murmurs must be transmitted too in this way as truly as by means of lung tissues. That may account to some degree for the otherwise unaccountable loudness of the mitral murmurs at the back.

DR. HORACE B. ARNOLD of Boston.—Replying to Dr. Bridge I would state that I used the term "pulmonary tissue" in the general sense. I usually include transmission of vibrations through the air in the bronchi, i. e., its divisions after entering the lung, and include it as pulmonary tissue. I consider conduction in the lung as possible by conduction through the chest wall or spine.

MYOPIA.\*

OPERATIVE TREATMENT IN HIGH DEGREES THEREOF.

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CHICAGO.

The advance of civilization has brought many advantages and blessings to the human race but has also furnished extra care, extra requirements on mentality, and necessarily a decided increase in the use of the eyes, especially for near work. This constant and continued use of the eyes for near work is one of the most potent factors in the production of axial myopia. By axial myopia we mean a receding of the posterior portion of the eye, making the distance from the cornea to the macular greater than normal. This is brought about by the combined action of accommodation and convergence.

The condition is most frequently acquired by children compelled to do a great deal of studying, at close range, perhaps in very poor illumination, and also by persons engaged entirely in fine work, which they hold close to the eyes in order to get a perfect image on the retina. The accommodation is used to a greater degree than normal, and the action of the internal recti muscles is increased so far as to fix both eyes upon the near work, thereby bringing a strain directly on the sclera at the temporal side of the head of the optic nerve. Under this continued tension or stretching the sclera at this point soon assumes a condition of inflammation, and gradually we have a chronic sclerochoroiditis which is followed by an atrophy and thinning of the sclera and afterward by its gradual separation from the optic disc. In this way that portion of the eye between the optic disc and the macula glutea is weakened and is forced back by the normal tension of the eye, and we have established a change in refraction known as myopia. It is not necessary to state here that this is not the only cause of myopia. It is, however, the most frequent cause of myopia of high degrees.

Heredity plays a very important part in progressive myopia. It is true that the children of myopic parents are rarely, if ever, born near-sighted. On the other hand they have an undoubted predisposition to myopia, since they are prone to inherit the anatomical peculiarities of their parents, and if forced to continue studies or to enter occupations requiring near work they usually develop myopia.

Experiments have been made with children of normal eyes not predisposed to myopia and children with normal eyes predisposed to myopia, that is, whose parents one or both have been myopic, with the result that the greater percentage of the latter under close work in school have developed myopia.

\* Read before the Illinois State Medical Society, May, 1899.