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Significance and interpretation of heart murmurs

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THE SIGNIFICANCE AND INTERPRETATION OF HEART MURMURS

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PREFACE

Clinical investigation of the diseased heart has largely depended upon judicious interpretation of the variety of sounds brought to the physician's ears by auscultation. Diagnostic instruments such as the electrocardiograph and the phonocardiograph and the diagnostic methods such as the roentgenogram have been properly reserved for the more difficult problems where there is definite evidence of disease. This paper is not intended to infer that auscultation is the alpha and omega of cardiology. It is but one tool, albeit a very important tool, which the physician has at his disposal.

The busy physician of the present day knows that there are very few diseases of the heart that he cannot diagnose by history, inspection and auscultation. Is there a heart murmur; does it portend heart decompensation? These are the questions which he wants answered.

This paper represents an attempt to answer them. Wherever better understanding of the sound of the murmur under discussion would result, recourse to diagrams has been made. The charts included are felt necessary to the proper evaluation of the evidence presented.

CHAPTER I

HISTORICAL ASPECTS

There has been a great deal of discussion concerning the causes and significance of heart murmurs, particularly the systolic type. Much speculation has been expended on the physical and mechanical factors involved in their production and a considerable amount of literature has been devoted to the correlation of murmurs with anatomical and pathological diagnoses. Despite this much confusion remains in their clinical interpretation.

Historically interesting is the swing of the pendulum of medical opinion. Not so very long ago all murmurs were regarded as significant of heart disease and many healthy individuals were condemned to invalidism by being diagnosed a cardiac cripple. They were made to live their lives within the restrictions of heart disease and as a result developed constant fear of their condition, leading to the production of full-blown cases of cardiac neurosis. Other more hardy souls would live out their full span of years in defiance of their amazed physicians who had originally made a grave prognosis. Later during World War I the pendulum shifted gradually to the point where all murmurs were

regarded lightly. The advice of Sir Thomas Lewis to throw away the stethoscope and trust to early symptoms was frequently quoted.(57, 15, 43) Most physicians were now prepared to agree that the systolic murmurs were of no importance and that attention should be focussed on signs and symptoms.(47, 52, 87) This swing in opinion reacted upon the patients diametrically opposite to the previously held position. Injustice was now being given serious cases of heart disease, and many patients whose activity should have been restricted were allowed full freedom.

The true position, of course, lies somewhere between these two extremes. Many systolic murmurs and practically all diastolic murmurs indicate organic heart disease: on the other hand many murmurs are completely innocent.(44, 111)

It is, therefore, the purpose of this paper to evaluate the significance of the various heart murmurs and to attempt to establish criteria whereby proper interpretation of the sounds may be made. In order to accomplish this goal, references must be made to the works of the early cardiologists who discovered many of the fundamental principles upon which the discussion will be based.

The investigation of the heart really began by the introduction of the mediate method of auscultation by Laennec (55) early in the seventeenth century, even though it had been known previously that unusual sounds might be produced by the diseased heart. Allen Burns (16), a lecturer on Botany in Glasgow, described such sounds in a case where the mitral valve was indurated and the pericardium inflamed. Burns observed that examination of this patient during life revealed a jarring sensation on palpation and a hissing noise as of several currents meeting, the sound resembling that heard in a varicose aneurysm.

Laennec noted how "within the past few years some physicians have attempted to gain further knowledge about the heart by application of the ear to the heart region" and informs us that Boyle, a fellow student, was the first to examine the apex beat this way. Then he describes how in 1816 he had occasion to examine a very fat young woman with general symptoms of heart failure. Percussion was impossible and immediate auscultation inappropriate because of the age and sex of his patient, so he recalled a simple acoustic fact that the scratch of a pin at one end of a piece of wood was heard clearly by the closely applied ear at the other end. Because there was no wood available, he rolled a

quire of paper into a crude cylinder and applied it to her chest. To his surprise he heard the heart sounds clearly and distinctly. He repeated this procedure on other patients and gradually evolved better stethoscopes and stethoscopic technique. Through the use of these instruments he soon came to recognize that a "bellows sound" was often associated with the presence of organic heart disease. Laennec, however, also discovered that not all murmurs were accompanied by pathologic changes and was the first cardiologist to suggest that some murmurs were physiologic in nature. From that time to the present, murmurs have been a source of difficulty. Byron Bramwell's statement that physicians do not agree as to what conditions point to an endocarditis in the living, though written in 1884, might well be repeated today.(12) Throughout the nineteenth century medical men, playing for safety, tended to regard the presence of a murmur as definitive evidence of organic disease, although such heart authorities as Gairdner as early as 1861 pointed out how harmless many bruits are.(38) This attitude led to much unnecessary invalidism and brought no credit upon the profession. The number of young men with heart murmurs who suffered no ill effects from the rigorous exertion of military life in World War I caused medical opinion, as reflected by

MacKenzie, Lewis, Broadbent, and White, to change to the extent that systolic murmurs were regarded as inconsequential.(64, 61, 15, 111) Of this type of interpretation, it had been cynically remarked that "we wait to diagnose the murmur until it is no longer there to diagnose."(1)

CHAPTER II

GENERAL DISCUSSION OF MURMURS

Heart murmurs, as defined by Paul White, are abnormal heart sounds having generally a blowing quality which is due to more or less rhythmical vibrations of the cardiac or arterial tissues.(111) They are generally, but not always, produced at or near a valvular orifice by abnormalities in contour or structure. More amazing than the occasional presence of an adventitious sound is the fact that murmurs are not always present. Elliotson pointed this out in 1833 when he stated that "nature has given an exact proportion between the size of the openings and that of the cavities so that the blood passes easily and noiselessly."(29)

The cause of cardiac murmurs has had as many explanations as had their interpretation. In 1819 Laennec attributed their presence to spasmodic contractions of the heart or arteries.(55) Corrigan in 1828 was the first to offer an explanation based on experimental evidence.(25) He attached pieces of intestine and lengths of arteries to a tap and found that when the tube was constricted, a bruit and a thrill were produced beyond the narrowing, provided that the rate of flow was sufficient.

Another untiring and observing worker of the same period, Savart (96), discovered that turbulence as an excitant cause for a murmur was produced by rapid flow through a narrowing into a wider lumen, such as might be produced by a stenosed valve. He applied this principle to experimental animals by digital pressure on their arteries and produced murmurs. Corrigan, learning of this work, noted that bruits were produced in hearts with stenosed valves, even though the vessel or cavity beyond were normal and evolved the so-called "jet theory" of murmur production.

This hypothesis is better explained in Corrigan's own words:(25) "When an artery is pressed upon . . . the action of blood in the vessel immediately beyond the constricted part is no longer as before. A small stream is now rushing from a narrow orifice into a wider tube and continuing its way into the surrounding fluid . . . its particles tending to leave vacuums between them throw the sides of the tube into vibration which can be distinctly felt by the finger and which gives to the ear the peculiar sound, bruit de soufflet."

Chauveau (19), an ardent physiologist and an able physician, proved in 1858 that the murmur was produced just beyond the narrowing and that the sound was better conducted in the direction of the stream. In the

same article he stated without proof that roughness of the intima or endocardium would not cause a murmur and wrongly contradicted Corrigan for saying that murmurs may be caused by changes in the quality or quantity of the blood passing through the constriction. This flat contradiction of Corrigan's articles by Chauveau established two schools of thought on the production of heart murmurs which have persisted to the present day, although numerous investigators, particularly Reid in 1923 and Thayer in 1925, confirmed and added to Corrigan's ideas.

Reid (83), conducting his experiments with elaborate brass and rubber tubing models of diseased conditions within the heart, and Thayer (106), working on the exposed hearts of living dogs, came roughly to the same conclusions regarding the cause of heart murmurs. They found that murmurs were caused by eddies produced within the blood stream, thus confirming the "jet-theory" of Corrigan. Reid conceived that in tubes of uniform bore the movements of fluid were linear, the outer layer serving to wet the walls and remaining adherent, while the axial stream moves fastest and between these two extremes the blood stream slides forward like a series of concentric cylinders slipping over one another. Such movement is silent, but under certain conditions the flow becomes

turbulent and vibrations of the surrounding tissues give rise to the typical murmur.

Reid also discovered that there were several factors which contribute to the characteristics of the sound, the most important being the velocity and quality of the blood. The vibrations of the vessel walls to be audible were found to be present only when a critical velocity of the blood flow was reached. This critical velocity in turn was found to be influenced by the harmonics of the vibrating tissues and by the quality of the blood itself. Anemic blood, for instance, was found capable of causing murmurs at a lower critical velocity than was normal blood. Another factor in the production of murmurs was found to be a cul-de-sac or a lip which projected against a current. As Reid stated, this too is explained ultimately on the presence of a "jet." Other lesser factors were the size of the lumen, the harmonics of surrounding tissues and the character of the vessel wall at the point of sound production. Reid and Thayer both stressed that roughness of the intima or endocardium did not cause a murmur, putting to rest another of the fallacies of modern cardiology.

In view of this hypothesis, what then are the conditions which may exist in the heart to cause a murmur? Thayer (106) summarized them well when he said: "The

appearance of a murmur at some point in the circulatory apparatus means the audible vibrations of the vessel wall resulting from passage of blood from a narrower suddenly into a wider cavity, or through a constricted area in the course of a tube. Whatever the physical possibilities as to the origin of vibration in fluid currents, it is doubtful whether murmurs that are clinically appreciable ever arise in any other way."

More precisely, then, murmurs are produced by (a) structural disease of a valve causing obstruction or regurgitation, (b) dilatation of a valvular orifice, (c) dilatation of a vessel or cavity such as the aorta, (d) abnormal communications between heart chambers or vessels, (e) membranes or vegetations which vibrate in the blood stream, and (f) edema of cardiac structures. (83, 100, 31, 72)

Of these the commonest mechanism is structural disease of a valve. In regurgitation of a valve, the valve cusps are stiffened, shortened, roughened, or retracted, so that complete closure does not occur. Blood then is permitted to pass through a "constriction" into a wider cavity, fulfilling the conditions for formation of an eddying current and in this way causing a murmur. This is diagrammatically explained in the figure below.

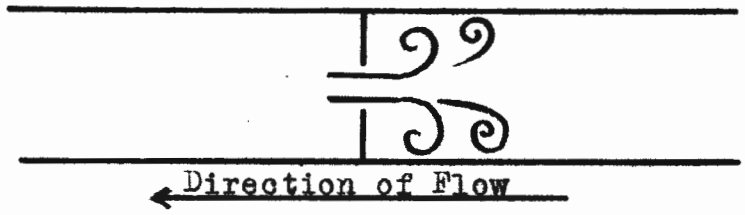

Fig. 1. Diagrammatic explanation of regurgitation.

In stenosis or valvular obstruction the valve is so deformed that complete opening of the orifice is impossible, and a jet is formed as is represented in the figure below. It must be clearly understood, however, that neither stenosis nor regurgitation can be "pure": logically, it is easily seen that a valve which cannot open must also by reason of its deformity be incapable of complete closure. Clinically valvular disease is seen accompanied by both stenosis and regurgitation, one phase predominating.(111, 61)

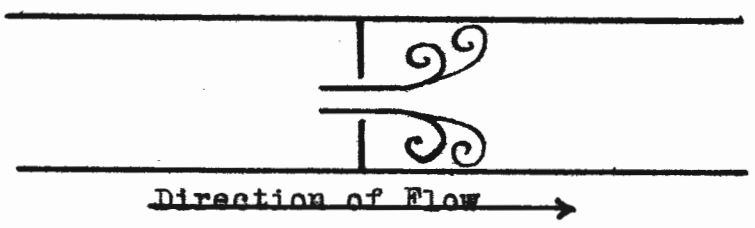

Fig. 2. Diagrammatic explanation of stenosis.

Endocarditis of the valve also produces erosions, vegetations, membranes, and penetrations which are capable of causing murmurs by the mechanism cited. These, however,

are rare, but should always color the significance and interpretation of sounds heard by the thoughtful cardiologist.

Widening of the valvular orifice frequently accompanies dilatation and hypertrophy of the heart cavities causing a relative insufficiency of the valves. Or if a chamber distal to an orifice dilates and the chamber proximal remains normal, the valve orifice will be relatively narrow and an obstruction result. This is relative stenosis. It was this mechanism which Billings (9) considered the definitive cause of organic heart murmurs in 1832. He erred, however, in believing dilatation of the chambers to be the only cause of murmurs.

Summarizing the mechanisms of murmur production, the final cause is seen to be a turbulence in the blood flow which sets surrounding tissues in vibration. Fundamentally the problem of evaluating the bruit depends upon determining whether the turbulence is produced by structural disease of the valve, dilatation of a chamber or great vessel, or decreased viscosity of the blood associated with increased velocity of the blood flow. This may be determined by a careful consideration of the character of the murmur.

The physician has several tools available to aid

him in the solution of this problem. Of these, the most commonly used, the most convenient and the most accurate is the stethoscope.(59) The phonocardiograph has often been useful but requires specialized and technical knowledge for evaluation of records. Moreover, inherent and intrinsic errors which are due to the limitations of the vibrating diaphragm demand interpolation: this casts grave doubt on the actual presence of whatever evidence may be found on the records.(54) Arenberg in 1941(3) stated that graphic registration and correlation of sounds by the phonocardiograph cannot replace the trained ear. This statement is particularly true in the short systolic murmurs of low intensity and even more so in cases of soft blowing diastolic murmurs of aortic insufficiency. The truth is that phonocardiographs cannot distinguish many murmurs from even the heart sounds. So the thoughtful use of the stethoscope, combined with the judicious choice of chestpiece is still the best instrument in the detection and interpretation of murmurs, for in an actual clinical test by Arenberg (3) the stethoscope was found to be superior to the machine.

Nevertheless, the phonocardiograph has given us much valuable evidence concerning the differentiation of murmurs from heart sounds and the proper choice of chestpiece for the stethoscope. Chiefly due to the efforts of

the indefatigable Orias and Braun-Menendez (76), criteria for normal heart sounds have been discovered which give the auscultator confidence when he attempts to distinguish the short blowing murmur from the closely attendant heart sound. Murmurs were found always to have a longer vibratory duration than the corresponding heart sound, giving proof to the statement of Norris and Landis (75) that the heart sound can be compared to a single stroke upon a drum; the murmur to the sound produced by blowing into a pipe. The sound bears the same relation to the murmur as does a pistol shot to the surge of the sea. The difference, then, consists in the suddenness of onset and of ending of the sound, as compared to the gradual beginning and uncertain termination of the murmur. As proven by the phonocardiograph, the essential difference lies in the fact that in the case of the heart sound, the first vibration is the greatest, whereas in the case of the murmur, we have at the outset a continuous series of equal vibratory excursions.

Moreover, thanks to the classical work of Rappaport and Sprague (80, 81), it has become generally known that few if any murmurs have a vibratory phase greater than 650 cycles per second and that most murmurs of significance occur within a range of 120 to 450 cycles per second. This had been announced by

Cabot and Dodge (18) in their experiments on frequency characteristics of heart murmurs. This knowledge has placed auscultation with a stethoscope upon a scientific basis, for proper design and choice of the chestpiece depends upon this data. For purposes of clarification it may be stated that there are no essential differences between the conventional bell and the diaphragm type according to the evidence presented by Rappaport and Sprague (80), for it was found that the bell stethoscope was converted into a diaphragm instrument upon application to the chest, the skin under the bell serving as a vibrating diaphragm. However, the larger the diameter of the open bell, the less efficient it was found to be at the lower frequencies, thus explaining the success of the diaphragm type in detecting the faint, high pitched murmurs such as are found in aortic regurgitation. Moreover, it was discovered that a rigid diaphragm acted somewhat as a filter by attenuation of the lower pitched heart and chest components.

Another way in which the phonocardiograph has been of service, in the evaluation of heart murmurs, has been as an aid to the exact timing of the murmur. Simultaneous records taken with an electrocardiograph have been of some aid in the determination of the presystolic murmurs particularly. But other than this and the

benefits listed above, the phonocardiograph has been of little clinical aid.

In estimating the importance of a cardiac murmur all of the foregoing must be kept in mind: it is the bridgework, so to speak, of the science of cardiology. However, there are several characteristics of the murmur itself that must be noted and recognized by the auscultator in order to evaluate its importance. They are, according to Reifenstein (87), and Norris and Landis (75), (a) the location, (b) the time, (c) the transmission, (d) the quality, pitch, and intensity, (e) the duration, (f) the effects of posture, respiration, and exercise. It is by these characteristics that the cardiologist recognizes the individual murmur of heart disease. Each is fundamental and the combined whole embraces the entire murmur. In its criteria for the study of heart murmur, the American Heart Association (23) lists intensity, pitch, quality, duration, and time as sufficient to describe the murmur. True these will suffice to describe the harmonics, but in order to evaluate the bruit, one must know its location, its transmission and the effects of posture, respiration, and exercise.

To localize a murmur is to find its point of maximum intensity. Murmurs heard loudest in the region of the apex are in the majority of cases produced in

the mitral region even though mitral valve lesions are occasionally best heard at a point midway between the position of the normal cardiac impulse and the ensiform cartilage. This serves to exemplify the rule of Norris and Landis (75) that murmurs are usually best heard at the points of the chest wall which are nearest to the orifices at which the bruit are produced. Nevertheless, murmurs are often audible over more than one valve area, or even over the entire precordium and it then becomes a difficult matter to decide as to whether one is dealing with single or multiple lesions. This situation occurs when the murmur occurs everywhere in the same portion of the heart cycle. The decision then is to be made on the following points, according to Adams: (2) The murmur produced by each of the valvular lesions has its own characteristic area of distribution and its intensity diminishes with increasing distance from that area. Any murmur whose distribution does not extend beyond one of these areas and whose intensity diminishes as the stethoscope is moved away from its assumed valvular area is probably due to involvement of that one valve; conversely, a murmur which decreases with intensity as the stethoscope is moved from one valve area, but increases as it approaches the second valve area may be assumed to be caused by multiple valve injuries.

The time of the murmur, or the phase of its occurrence in the heart cycle, is very important. Murmurs are classified primarily according to the phase of the cycle, being systolic, diastolic, or continuous. Systolic sounds ordinarily occur simultaneously with the first sound and extend into systole. Sounds which occur separately from the first heart sound are late systolic in timing. Sounds which occur shortly before the first sound are presystolic, or better, late diastolic in timing. Diastolic murmurs are termed early diastolic, mid-diastolic, or late diastolic, dependent upon when they start in the cardiac rest phase.

The commonest error in the evaluation of murmurs is failure to time the sound correctly.(60) It is this inability that Sir Thomas Lewis (61) lamented when he said, "Many physicians cannot and will not time murmurs correctly." As was pointed out by Wolferth and Margolies (116), ". . . the proper timing of a murmur narrows the diagnostic problem. To do so it is first necessary to distinguish between the first and the second heart sounds. If, as in certain cases of tachycardia this cannot be settled by timing the sounds with the apex beat or carotid pulse, auscultation below the apex and above the base may be helpful." In the very difficult cases, the method of Farfel (32) may be useful. This is the

use of a blood pressure cuff and a sphygmomanometer with the mercury column fixed midway between systolic and diastolic pressure. With this method the rise of the mercury column and the second heart sound are synchronous.

The transmission of the murmur is the third most important point upon which interpretation of the sound is based. Transmission is the ability of the murmur to be conducted towards the periphery just as the wind transmits sound. In the section dealing with production of murmurs it was stated that Corrigan (25), Chauveau (19), and Galabin (39) had discovered that murmurs are always transmitted in the direction of the blood flow. However true this may be, it is also known that murmurs are transmitted by the papillary heart muscles to the back. (109)

Transmission of a murmur has much diagnostic significance and always gives serious prognosis to heart disease. As was shown by McCrudden (67), transmission of murmurs increases severalfold the relative mortality from cardiac failure.

Of slight importance from a prognostic or diagnostic standpoint are the quality, pitch, and intensity of the bruit. Due to the nature of their production most murmurs have a "blowing" quality much like the sound of the breath forcibly exhaled through the lips. The

pitch may be compared to the sound of a fan or the sound of a whistle, dependent on whether it is low or high in the scale. The intensity, however, has considerable variation and is simply the degree of loudness.

The method of gauging murmur intensity in general use today was proposed by Freeman and Levine (35) in 1933. These investigators arbitrarily divided the murmurs into six gradations, the first being the faintest bruit auscultable, the sixth being the loudest and capable of being heard without a stethoscope. The other four gradations are shades of loudness within these extremes. These stages are described as very faint, faint, very moderate, moderate, loud, and very loud.

Although most authors are critical, White (111) attaches significance to the loudness of the murmur, having found a greater likelihood of organic trouble to accompany the louder murmur. This belief is also held by Freeman and Levine (35) who report that in an analysis of 1,000 cardiac patients, murmurs of greater than grade II intensity were considerably more prone to be organic, whilst murmurs of grade I and grade II intensity were largely non-organic in cause. Actually less than ten per cent of the systolic murmurs of grade I and II indicated organic heart disease according to their investigation. This led to the statement that "a proper

interpretation of the intensity of the murmur and of the possible causative factors . . . will aid greatly towards a more accurate diagnosis, prognosis, and treatment."

Intensity should not be a deciding factor in the development of a prognosis despite the authority represented by the names of White, Freeman, and Levine. It seems illogical that one should believe an extremely faint murmur is indicative of slight cardiac damage, when as a matter of fact it is commonly known that murmurs become less intense in acute cardiac decompensation. What may be true in their statements is the point that an increase or a decrease in intensity while under observation may have some significance.

Murmurs which are faintly audible at rest, or even inaudible, become more clearly heard through the increased blood flow resultant from exercise. However, as was indicated above, if the myocardium is weakened and failure imminent, exercise may accomplish just the opposite and lessen the intensity of the murmur.(2)

The effects of respiration upon a murmur are very slight. There is some interpretative value in distinguishing the cardiorespiratory murmur because the latter is apt to be better heard in some particular phase of respiration. The intensity of a murmur may also be

altered by fully expanded lungs in full inspiration. Therefore it is an auscultatory trick to listen to the heart in full expiration, in this way bringing the heart closer against the chest wall.

It is commonly known that posture exerts some effect upon the intensity of murmurs for almost all bruits are affected by the position of the patient. Those which are almost inaudible while the patient is sitting or standing might be clearly heard when he lies down; those not well heard in the recumbent position might be brought out by having the patient lie on his left side or face downward. This is especially true of the mid and late diastolic murmurs of mitral stenosis and the early diastolic murmur of aortic regurgitation.

Bartlett and Carter (7) would add one more standard to the criteria already listed. They state that alteration of the heart sounds is indicative of actual heart injury, and they proved by the aid of combined electrocardiograms and stethograms that murmurs associated with diminution, obliteration, or abnormal accentuation of heart sounds are usually organic in nature: heart sounds, they state, are seldom obliterated by a functional murmur. This has been noted clinically by many other observers but there are no statistical analyses of case histories which either confirm or negate.

Summarizing, then, the characteristics by which the significance of a murmur may be determined: we have considered its location, time, intensity, transmission, and duration to be of the greatest importance, and the effects of posture, exercise, respiration, and the quality of pitch to be of secondary value in its interpretation: also that alteration of the heart sounds may be of value in distinguishing the non-organic murmurs, from the highly significant organic bruits.

Before passing on to a discussion of the individual murmurs, it is necessary to provide the reader a terminology, and a general classification by which a comparison may be drawn between the murmur under discussion and those which have preceded it.

In 1943 the American Heart Association adopted a table and terms to be used in describing heart sounds and murmurs. (26) This chart is included to give the reader the descriptive words which may be used to describe the bruits, and which will be used throughout this paper. In the table the terms in capitals are recommended for routine use. Those in smaller type are regarded as less satisfactory synonyms for those terms in capitals immediately preceding them but may be used as alternates, or better as additions if it is thought that the recommended term is inadequate.

HEART SOUNDS				
INTENSITY	PITCH	QUALITY	DURATION	TIME
NORMAL		NORMAL	NORMAL	
FAINT Weak Distant Muffled		SHARP Snapping Valvular	SHORT	
LOUD Accentuated Increased		BOOMING Muscular	PROLONGED	
ABSENT Replaced		SPLIT REDUPLICATED RINGING		
		MURMURS		
FAINT Soft	HIGH	BLOWING	SHORT	SYSTOLIC
MODERATE	MEDIUM	HARSH Rough Coarse	MODERATE	EARLY SYS.
LOUD	LOW	MUSICAL RUMBLING CRESCENDO DECRESCENDO	LONG	LATE SYS. DIASTOLIC EARLY DIAST. MID-DIAST. PRESYSTOLIC Late Diast.

Chart 1. Table and terms to be used in description of heart sounds and murmurs.

In the diagrams which will be included in the hope of better interpreting the various sounds, Segall's (97) method will be used where possible, but somewhat modified. The figure which is included to illustrate the method is thought to be self-explanatory.

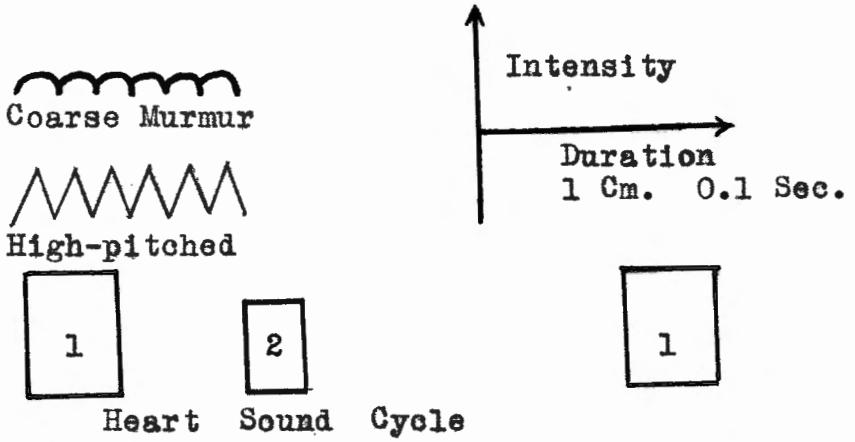

Fig. 3. Segall's graphic method of describing heart murmurs.

In the past any discussion of murmurs was arranged around a classification of "functional" and "organic." But this was misleading, unsatisfactory, and confusing. Some physicians when writing of a bruit would call it functional if it applied to a normal heart. Another set of authors would apply the term to a dilated heart. The term organic was saved to apply to actual structural deformity of a valve.

Yet serious organic disease of the heart muscle resulting in cardiac dilatation may in the absence of valve deformity cause a murmur. In infarction of the heart a systolic murmur is common due to mitral regurgitation, yet no valvular deformity exists.

It was with this in mind that White, Adams, and Craib in 1942 (108) suggested a classification by which the true nature of the various types of murmurs is more

closely indicated. The classification, being sound, was immediately adopted by the American Heart Association. It is included in order to suggest the inter-relationship of the types of murmurs.

I. Physiologic Murmurs

A. Intracardiac or Intravascular
B. Extracardiac

II. Pathologic

A. Structural Valvular Disease
B. Congenital Cardiovascular Defects
C. Dilatations of a Heart Chamber

In the discussion to follow the classification will be used as a broad outline on which to evaluate the murmurs. However, especial reference will be made to the murmurs which the American Heart Association has considered to be of particular interest.(23) These are the murmurs of Mitral Regurgitation, Aortic Stenosis, Dilatation of the Aorta, Ventricular Septal Defect, Mitral Stenosis, Aortic Regurgitation, and Patent Ductus Arteriosus.

CHAPTER III

THE SYSTOLIC MURMURS

The terms physiological, functional, accidental, and non-organic have all been used to describe the many harmless murmurs that may be heard over the precordium. All these are subject to criticism, but the best term is probably "non-organic" for it is well understood as the opposite of "organic."

That systolic murmurs may occur in the absence of organic heart disease has been known since the time of Laennec.(55) They may appear in a great variety of physical disorders and also in subjects who are in normal health, especially in the young. Levine (58) says that they are usually faint (grade I) while slight murmurs (grade II) are less frequent, and moderately intense ones (grade III) are relatively rare. This explains why they are circumscribed and poorly conducted. They are mostly soft and blowing, of short duration, accompanying rather than replacing the first heart sound, very variable, and readily influenced by posture, respiration, and the cardiac rate.

There are no very comprehensive estimations of the incidence of functional murmurs; reports seem to be colored entirely by the experience of the writers. A

high proportion may be found in normal individuals after strenuous exercise.(103, 113) Fahr (30) reports that 3.2 per cent of all army recruits examined showed functional murmurs. Siemsen (98) claims that 9 per cent of the general population possess non-organic murmurs, and that 46 per cent will exhibit systolic bruit after exercise. Thayer (106), on the other hand, analyzed the records of his clinical experience and states that 56 per cent of children under ten years of age, and 19 per cent of the general population under twenty years of age will show systolic bruits.

The Cardio-Respiratory Murmur

The cardio-respiratory murmur is probably the commonest of all the non-organic murmurs.(69) It is really a rhythmic exaggeration of the breath sounds and is often classified as a form of cog-wheel respiration. Two varieties of the cardio-respiratory bruit have been described: One is heard over substance lesions of the lung, such as consolidation, atelectasis, or cavity, usually at or near the anterior border of the upper lobes. In the earliest cases described (88) adhesions between the lung margin and the pericardium were found, but these are not essential to production of the murmur. (69) The other and more common type of the bruit occurs

in completely healthy people.(63)

Site: The murmur is audible about the apex and along the left border of the heart, more rarely over the right border. Its maximum intensity is not necessarily at the exact apex, but may be somewhat farther to the left. It may be well heard in the axilla and even over the back.

Time: The murmur does not replace the first sound but begins rather late in the cardiac cycle and always ends before the beginning of the second sound.

Character: It is a soft sharp "whiff," beginning and ending abruptly. It has also been described as blowing, but never as harsh or rough. The pitch is characteristically high: the intensity varies considerably, although it is ordinarily grade I or grade II. Hoover (50) recounts a case of a cardio-respiratory murmur being audible at a distance of three feet. The most characteristic feature of the bruit is the variation throughout the respiratory cycle. It is always loudest during inspiration. The posture, too, has a profound effect although there is no definite clinical rule.(46) Local pressure with the stethoscope may vary the intensity of the murmur.(105) Exercise, contrary to the experience with other murmurs, usually makes it more evident. The heart sounds are not affected.(110)

In the vast majority of cases, the murmur is of no importance; if lung disease is present, other signs will draw attention to it.

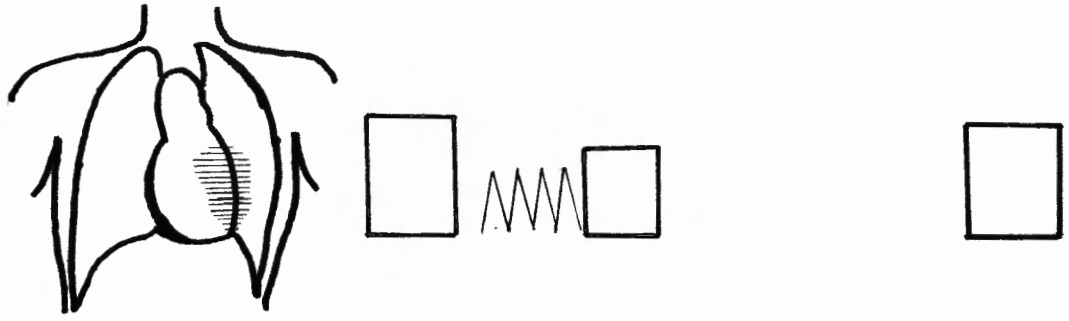

Fig. 4. Diagrammatic representation of the cardio-respiratory murmur.

The Eustace-Smith Murmur

A non-organic murmur frequently encountered in children is the Eustace-Smith murmur. Not much is known of it and not much is written about it in the available literature. Gedgoud believes that it is due to the spurt given the venous blood in its return through the jugular veins when the current encounters the jugular bulb.(41) Norris and Landis believe that it is a murmur caused by pressure of a persistent thymus on the innominate vein.(75) Whatever the cause, it is certain that the bruit has no pathological significance and can be safely discounted by the physician. There are no available statistics, but it is estimated that fifteen per cent of the children under three coming to the

University of Nebraska Dispensary exhibit the sign.(41)

Site: The murmur is most commonly heard at the jugular notch and at the aortic valve area. There is no transmission.

Time: There is no replacement of the cardiac sounds. The murmur starts definitely late in systole.

Character: It has a humming or a "whirring" sound which seems close to the ear. The pitch is high, the intensity, low. It is loudest when sitting or standing and often disappears completely in the recumbent position. There is no alteration or obliteration of the heart sounds, even though the murmur may extend over into diastole. The sound may disappear entirely when pressure is placed on both jugulars, to be heard with greater intensity when the pressure is relieved.

The murmur has no pathologic significance and the parent may be notified that the murmur will eventually disappear of its own accord.

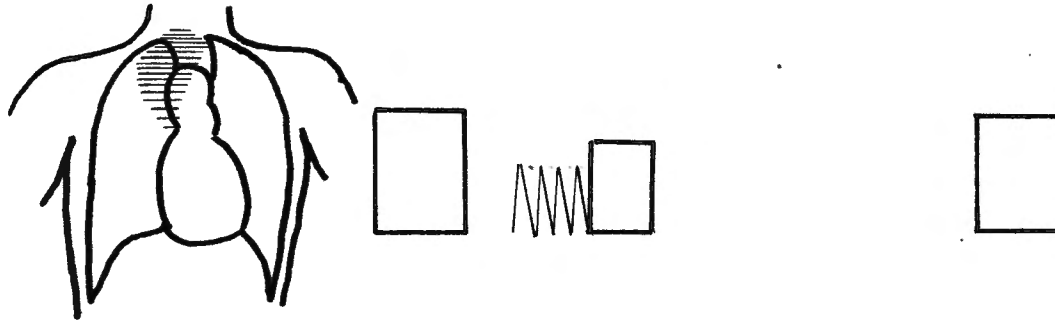

Fig. 5. Diagrammatic representation of the Eustace-Smith murmur.

Hemic Murmurs

White and Craib (108), in their classification of bruits, included the hemic murmurs with the organic type because they felt that the sound was due to relative dilatation of the valve orifices or heart cavities following tissue anoxia. This conception was proven invalid (except in extreme cases) earlier in this paper when the work of Thayer and Reid (105, 83) was cited. Thayer (105) produced murmurs in the exposed hearts of living dogs by substituting normal saline for blood, and noted that there was no immediate hypertrophy. The abnormal sounds were explained as being due to decreased viscosity of blood which more readily allowed turbulent motion at the valvular orifices.

The innocent character of the murmurs produced by anemia is shown by the fact that they disappear completely as the blood improves.

Site: All valve areas or any one singly may be the location of a hemic murmur. The commonest sites, however, are the apex and the mitral valve area. Ordinarily these bruits are not propagated but are sharply demarcated. Garrod (40) stated that they are infrequently loud enough to be transmitted to the back.

Time: All hemic murmurs are systolic in time; usually they are late systolic, but they always end

before the commencement of the second sound.

Character: The sounds are extremely variable and have no individual characteristics. Thayer (105) presents some evidence which seems to indicate that the intensity is proportional to the blood loss. The pitch, on the other hand, may vary from low to high, the quality from blowing to harsh.

The murmur should be diagnosed only when there are definite evidences of anemia. Organic causes should be ruled out. The murmur should disappear as the anemia improves.

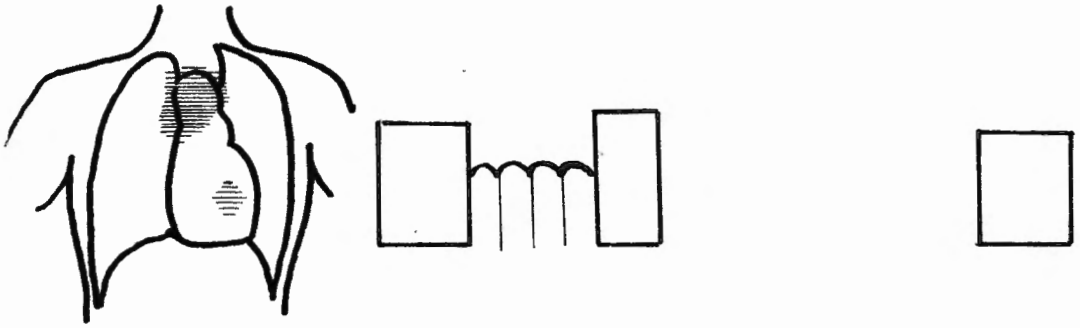

Fig. 6. Diagrammatic representation of the hemic murmur.

Non-Organic Pulmonic Murmurs

The pulmonary valve area has been named from Balfour's (6) classical remark, "the region of cardiac romance." Murmurs are very commonly found in this region, though very few are organic. They are hemic, cardio-respiratory, pressure, and traction in cause. Rudolf (94) stated that a murmur may be found in this

area in sixty per cent of all children under fourteen years of age. They are especially common in flat chested adolescents and thin chested women. A murmur peculiar to this area is produced simply by firm pressure over the region with the stethoscope.(74) Thayer (105) substantiated this and proved that the murmur was caused by constriction of the closely underlying pulmonary artery by the transmitted pressure.

Many explanations have been put forth to explain the existence of certain other murmurs in this area. Most writers postulate some alteration in the relationship of the conus and pulmonary artery to the surrounding tissues.(15) This substantiates the post mortem experience and observations of Foxwell (37) who was struck by the dilatation of the conus. Rudolf (94) suggested from this that in dilatation of the conus the pulmonary artery becomes lengthened and kinked, shorter and broader.

There is one other bruit found in the pulmonic region which is not due to organic heart disease. This is found in conditions of increased pulmonary pressure, especially when due to obstruction of the blood flow in the lungs. Needless to say, the condition is rare. Differentiation cannot be made on the qualities of the murmur itself but on the associated signs and symptoms of the disease process.

Site: Since the murmur has its origin in the conus or in the artery, its greatest intensity will be in the pulmonic region.

Time: Physiologic pulmonic murmurs accompany, rather than replace the first heart sound. They may begin in late systole. All, however, terminate before the commencement of the second heart sound. There is no propagation.

Character: The quality is variable and may closely similate the characteristics already described for hemic murmurs. It is most commonly of low pitch and intensity. Accentuation of the second heart sound should be interpreted as due to increased pulmonic pressure. Should this be present an organic cause should be sought for the murmur.

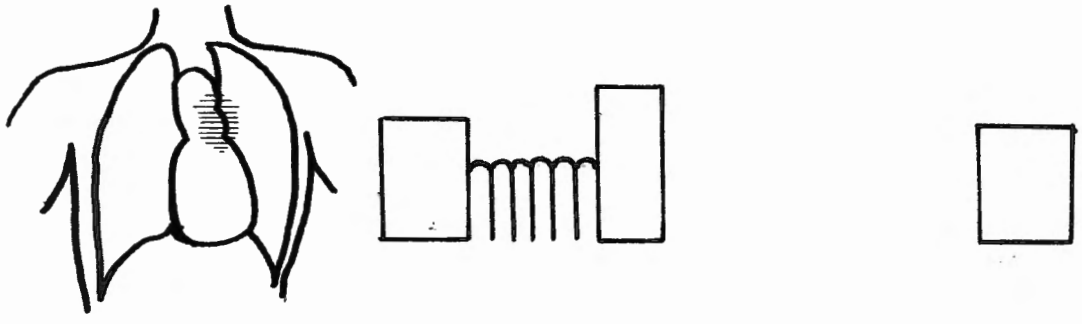

Fig. 7. Diagrammatic representation of the common type of non-organic pulmonic systolic murmur.

Other Non-organic Murmurs

The tricuspid valve area has no characteristic non-organic murmur other than those which have already been discussed. As was pointed out by King (53) in his excellent monograph, "The Safety Valve Function of the Right Ventricle of the Heart," damage to the tricuspid valve rarely occurs except in conjunction with damage to other valves. Because of the inherent strength of this valve, single murmurs localized to the tricupid region are very rare. Functional murmurs are seldom encountered; diagnosis can usually be made on the basis of the other symptoms, as for instance, anemia.

General Discussion

It will be observed that many of the non-organic bruits discussed above have been intracardiac or intravascular in origin. Their differential diagnosis may seem difficult until it is realized that there are several characteristics by which they may be interpreted, viz.: they are soft, short, and blowing, and often vary with the position of the patient; they are frequently evanescent and fleeting; moreover, characteristic signs of the actual process--anemia, cavities in the lung, et cetera--are often present to aid in the diagnosis. (36, 17)

No discussion of non-organic murmurs would be complete without mentioning those systolic bruits associated with fever, exercise and hyperthyroidism. In all cases the mechanism is the same--that is, increased velocity of blood flow through the heart chambers.(13) No one valve area is afflicted in particular, although most sounds are located in the aortic and apical regions. (111) Per se, they do not indicate organic damage to the heart nor have they been proven to indicate valvular orifice dilatation.(82, 35) Such murmurs have even been used to diagnose the cause! Levine (58) recites several cases in which the presence of a puzzling systolic murmur led him to search for other signs which led to a diagnosis of anemia, or hyperthyroidism, or lung pathology.

Logically, the question arises concerning the significance that should be attributed the puzzling systolic murmur which has no associated signs of organic heart disease, yet, which is not associated with fever, hypertension, hyperthyroidism, anemia, tachycardia, or lung disease. Because Levine and White (58, 111) feel that loud murmurs are invariably associated with noticeable organic signs and symptoms, murmurs of greater than grade II intensity are removed from consideration. What significance, then, should be attributed to such sounds?

Levine (59) states unequivocably that murmurs of

grade I intensity should be disregarded completely unless there is an antecedent history of Rheumatic Fever or Chorea. He says that a history of frequent nosebleeds, vomiting, sweats, or unexplained illnesses should incline the physician to be cautious and regard the murmur as organic. Maliner and Okin (65), working with epinephrine and amyl nitrite, estimated that 71 per cent of the children thought to have functional murmurs in reality had some underlying organic condition. Rednick (82), in an analysis of 268 systolic murmurs which went to the post mortem table, found that only 22 per cent were free from organic cardiac damage; 50.7 per cent showed definite valvular deformity. Of the 268 autopsies only 16.4 per cent had symptoms of cardiac disease before their final illness.

These statistics would seem to indicate that the diagnosis of non-organic heart murmurs is made too frequently. Contratto (24) is in complete agreement with this statement, and laments the number of people who pass through life without adequate treatment. Blumenthal (11) differs, and reports that in his clinical experience "if we find in a heart of normal size and rhythm a systolic murmur with absence of any sign that would indicate that it is definitely organic in origin, then we conclude that the heart is perfectly normal."

MacKenzie (64), in his long-continued follow-up studies, agreed; for, he said that he has seen many individuals with very loud rasping systolic murmurs for thirty years who never suffered from heart decompensation. He also says that "the estimation of the sign of functional murmurs is not based on the murmur itself but on the functional efficiency of the heart muscle and on the presence or absence of other signs of cardiac affections."

A very confusing factor in evaluating these murmurs is the reliance to be placed on insurance statistics. These have led the insurance companies to conclude that there is no such thing as a harmless murmur, and on that basis they either rate up heavily or reject afflicted applicants.

McCrudden (67) states that there is a definite decrease in life expectancy with all apical systolic murmurs even though they are unaccompanied by any sign of organic disease or history. Starr in 1933 (101), and Hunter in 1937 (51), presented statistics prepared from all the existent insurance policies in the United States, which indicated that the relative mortality increased progressively when the murmur was constant, when it was transmitted, and when there was a history of Rheumatic Fever. They also reported that, contrary to McCrudden's experience, inconstant systolic murmurs had a relative

mortality of less than the expected. For this reason they considered inconstant murmurs to be physiologic. All agreed, however, in the statement that transmission, history (if significant), and cardiac hypertrophy were the best methods of evaluating the significance of a murmur.

The conclusions, then, to be reached from this discussion are: (a) murmurs occurring at the base of the heart in the pulmonic valve area and which are not transmitted should always be regarded as inconsequential; (b) inconstant apical systolic murmurs should be regarded as nearly always non-organic; (c) constant apical systolic murmurs are usually non-organic; (d) transmission of the murmur should lead the physician to be cautious in his prognosis; (e) a history of Rheumatic Fever or Chorea should be regarded as highly significant.

The Organic Systolic Murmurs

The commonly encountered organic systolic murmurs are those due to mitral regurgitation, aortic stenosis, dilatation or atheroma of the aorta, pulmonary stenosis, and interventricular septal defect.(23) Less commonly heard are those murmurs due to patency of the foramen ovale, and the ductus arteriosus, and the bruit which Perry (77) ascribed due to subaortic stenosis.

Lesions of the tricuspid valve occur but rarely, and then in conjunction with damage to other valves.

It was MacKenzie (64) who made the statement the significance of murmurs rests in whether they portend heart failure. This is obviously true and little clarification is needed except as it applies to the present discussion. The significance and interpretation of non-organic bruits have already been discussed; clearly the problem to be presented is an evaluation of the characteristics of murmurs which are known to lead to decompensation. It has been obversely stated that these murmurs are recognized by the early signs of a failing heart, viz., hypertrophy, dilatations, increased blood pressure, accentuation or masking of the heart sounds, and other related phenomena. Consequently the remainder of this paper will be devoted to a discussion of the characteristics of the various heart murmurs and their recognition. Most of the data presented will be taken from the works of White (111), Lewis (61), MacKenzie (64), Adams (2), and Norris and Landis (75) unless of a controversial nature where due credit will be given.

Mitral Regurgitation

The murmur due to mitral regurgitation is best heard at the apex. It is probably the commonest of the

organic murmurs although there is considerable difference of opinion among authorities. In an examination of 20,000 inductees in the Pacific Northwest, Wilbourne and Ceccalini (112) found that 83.1 per cent of the valvular defects were mitral in origin, 50 per cent of the total being due to mitral regurgitation. Moreover, the same study showed that approximately 25 per cent of all the murmurs encountered were due to mitral regurgitation.

Insurance statistics of the relative mortality caused by mitral regurgitation are interesting. McCrudden (67) states that the relative mortality of this valvular defect is far above standard risk and increases progressively with hypertrophy and a history of Rheumatic Fever. Rodgers (92), Grosvenor (45), and Rockwell (91) confirmed these figures.

(See Chart)

	RELATIVE MORTALITY		
	RODGERS	GROSVENOR	ROCKWELL
MITRAL REGURGITATION	165%	166%	151%
WITH MILD HYPERTROPHY	253	209	184
WITH HISTORY OF RHEUMATIC FEVER	455	309	391

Chart 2. Statistics showing the progressive increase in mortality with the symptoms listed.

Anderson (4), considering these figures, stated that murmurs of this nature carried too much risk to be insurable but suggested that the applicant's occupation

be considered before the relative mortality be judged. This statement hits at the crux of the problem: do these statistics reflect the relative mortality from mitral regurgitation alone, or do they reflect other conditions which have no relation to the subject under discussion? Lyons (63) feels that considerable reliance should be placed on these figures, and it is with him that we agree.

Niehaus (72) states that there are two causes which result in organic murmurs: (a) cardiac enlargement from degenerated myocardium (relative mitral regurgitation) which results in dilatation of mitral ring and from a pulling down of the chordae tendinae preventing closure, and (b) inflammation of the mitral valve resulting in distortion. The typical causes of this latter are Rheumatic Fever and severe infections such as Tonsillitis, Pneumonia, and Scarlet Fever.

Site: Most authorities state that the point of maximum intensity is exactly at the apex, though some prefer a point a little above and to the right. Transmission to the back or axilla depends upon the intensity. Confusion with the murmur of aortic stenosis can best be avoided by carefully locating the point of maximum intensity, according to Baker, Sprague and White (5) who state "the clue to these cases lies in the fact that the

loud harsh systolic murmur heard at the apex is also heard at the aortic valve area although perhaps less loudly." They feel that the mitral regurgitant murmur is frequently well heard at the lung bases and in the left axilla and that this happens frequently enough to be of diagnostic value.

Time: The typical murmur is exactly systolic and usually masks the first sound in whole or in part. It is always diminuendo in character, fading away during the first pause.(62)

Character: The quality of an organic mitral murmur may vary from a gentle blowing to a harsh rasping bruit. It may be whistling or musical. The intensity is ordinarily greater than grade II, being roughly proportional to the severity of the lesion.(111, 58) The pitch is moderately high. The murmur is constant and unaffected by posture and respiration.(72) It may be more clearly heard with a bell stethoscope, though little difficulty should be experienced hearing the murmur with a Bowle's type. Occasionally firm pressure on the chestpiece will make the murmur more clearly heard.

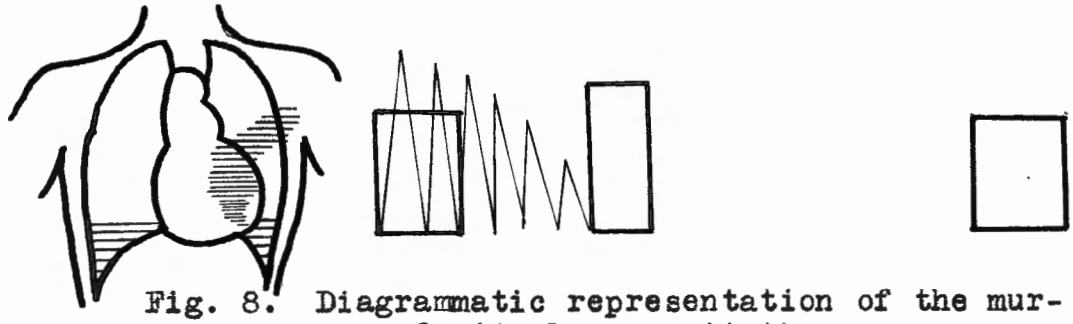

Fig. 8. Diagrammatic representation of the murmur of mitral regurgitation.

Aortic Stenosis

Stenosis of the aortic valve is the least common cause of murmurs in that area.(112) Statistics on the causes of systolic murmurs in this region are incomplete but it is estimated to be less than five per cent of the total organic murmurs.(2)

The causes of aortic organic bruits are listed by Adams (2) as being (a) dilatation of the aorta following arteriosclerosis, with or without hypertension; (b) the result of syphilis; (c) stenosis of the valve; (d) aneurysm of the aorta; and (e) transmitted murmurs. The interesting pathology of the typical lesions of this area is quite beyond the scope of this paper, but suffice to say, mere roughening of the valve is considered insufficient to produce a murmur.(105) If a cusp is thickened or calcified and projects into the blood stream during systole, then a turbulent flow will be set up. In other cases the valve remains normal but the proximal part of the aorta becomes dilated from a generalized weakening following syphilis or arteriosclerosis.

Site: The maximum intensity is often encountered in the aortic area in the second right intercostal space. Transmission is usually up into the carotids into the neck. Frequently the murmur is audible over the entire chest and may be noted in the back.

Time: The typical murmur is holosystolic, occupying the whole of systole and masking the first heart sound.(7)

Character: The characteristic murmur is rasping and harsh, very occasionally it may be musical. The intensity is high and apparently close to the ear. The sound is remarkably constant and is rarely affected by posture, respiration or exercise. The character of the second aortic heart sound may be useful in distinguishing valvular heart disease from diseases of the aorta: diminished heart sounds are indicative of the former.(58, 7)

Diagnostic phenomena which led White and Wood (117) to exclaim that not a case of aortic stenosis when proven by post mortem had been misdiagnosed in ten years, are the hypertrophy, the typical pulse, the characteristic thrill and the diagnostic blood pressure. Obversely, Levine (58) feels that there are many cases of early aortic stenosis which are missed because the maximum intensity of the bruit is at the apex. In answer to this, Sir Thomas Lewis (60) states unequivocably that ". . . to diagnose aortic stenosis on the basis of the systolic murmur at the aortic cartilage is unpardonable." On the other hand, Willius (115) affirms that "a systolic murmur that is confined to the aortic area is almost without exception

indicative of disease of the aorta or the aortic valve, namely, aortic stenosis, aortic sclerosis or aortitis." To this last we must agree.

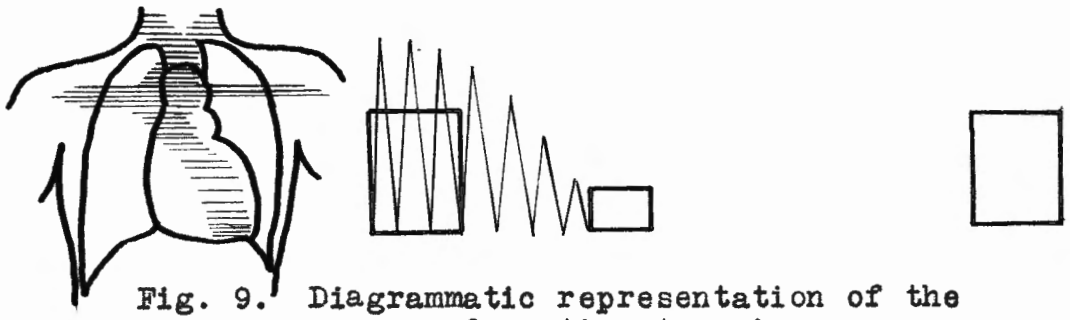

Fig. 9. Diagrammatic representation of the murmur of aortic stenosis.

Other organic systolic murmurs are largely congenital in nature with the possible exception of the relatively rare tricuspid valve regurgitation. Tricuspid valve injury, as was discovered by King (53) acts much the same as a safety valve and occurs in combination with other valvular damage.(68) Relative tricuspid valve regurgitation occurs also in combination with dilatation of the other valves.(53)

As a general rule, these sytolic bruits are rare and readily diagnosed by concurrent signs and symptoms of cyanosis and dyspnea. Except in the case of pulmonary stenosis murmurs occur over the body of the heart and not in the valve areas.

The typical murmur of pulmonary stenosis occurs throughout systole and is localized over the valve area. It may be blowing or harsh in quality, less intense and

variable with full inspiration, unchanged by posture. Thrill is often associated. Transmission may be any place on the chest or back.

The bruits of a patent interventricular septum has been named Rodger's Murmur after its discoverer. It is relatively common and has been compared by Norris and Landis (75) to the whirring of a grinding machine. It is very loud and harsh, transmitted widely, constant, and unchanged by position, respiration, or exercise. Heart sounds are typically unchanged and unaffected.

Other congenital heart lesions which have been listed have diastolic components which lead to their proper discussion under continuous murmurs.

CHAPTER IV

THE DIASTOLIC MURMURS

Except for a diastolic cardio-respiratory murmur similar in all respects and characteristics to the systolic cardio-respiratory bruits previously discussed, diastolic murmurs are always pathologic and point to an important disorder of the heart.(2) Sullivan (104) even discounts the cardio-respiratory bruit when he says: "The presence of a diastolic murmur always establishes the diagnosis of organic heart disease." Willius (115) of the Mayo Clinic reports that these murmurs are important and ". . . are most commonly identified with aortic insufficiency and with mitral stenosis." Wood and White (117), however, reported in 1923 that many diastolic murmurs due to dilated hearts have been falsely diagnosed mitral stenosis and suggested that perhaps the average physician was a shade too anxious to call any diastolic bruit that he heard either aortic regurgitation or mitral stenosis. Wood and White stated that ventricular hypertrophy of a high degree seemed to be the main factor in such cases. Fisher (33) in 1894 reported five cases of such a mistaken diagnosis.

Wilbourne and Ceccalini (112) analyzed the statistics from the examination of 20,000 inductees: their

figures seem to show that mitral stenosis and aortic insufficiency are the cause of the larger share of diastolic murmurs. The American Heart Association (23) lists these two murmurs as the important bruits. Sullivan (104) considered that the protodiastolic and mesodiastolic murmurs may be the result of either semilunar valve regurgitation or atrial-ventricular valve stenosis and these are decrescendo in type.

Mitral Stenosis

Mitral stenosis may be relative or absolute.(10) In relative stenosis enlargement of the ventricular cavity is held to blame. Fisher (33), Phear (78), and Bramwell (14) have all reported cases. Bramwell emphasized the fact that the production of an obstructive murmur depended on the degree of obstruction relative to the rate of flow through the obstructed orifice and suggested that the mitral orifice was too small for blood flow when the rate of venous return was increased. Contrary to the White, Lewis, MacKenzie opinion, Bramwell did not consider this an organic lesion.

Weinstein (107) recorded that the anatomic character of his series of apical diastolic murmurs without structural mitral disease was due to (a) marked dilatation of all the chambers, and (b) myocardial lesions. The

disappearance of the murmur terminally suggested to him that a certain amount of myocardial power is necessary for their production. (Velocity) White (110) stated that functional mitral stenosis occurs with marked ventricular hypertrophy in rare instances of tumor or thrombus large enough to obstruct the blood flow and in aortic regurgitation when the regurgitant stream forces back the anterior cusp of the mitral valve. (See Flint Murmur under Aortic Regurgitation.)

Absolute or organic mitral stenosis most commonly occurs as the result of the healing of endocarditis following Rheumatic Fever according to Bland, White and Jones.(10) Here, there is actual deformity of the mitral valve. Cabot (17) reported that a history of Rheumatic Fever was obtained in 78 per cent of the mitral stenoses proven by necropsy, chorea 6 per cent, and acute Tonsillitis in 7 per cent. Histories of Scarlet Fever, Diphtheria, and similar acute infectious diseases were obtained in the remainder.

The typical pathology is due to the scarring of the valve cusps and fibrosis of the chordae tendinae. Two processes in particular are responsible for defective function--White (111): one of these is the fusion of the cusps at their edges causing both stenosis and regurgitation; the other is fusion of the chordae tendinae with

shortening which further distorts the valve. In very chronic cases the fusion of the cusps proceed to the typical "fish mouth" valve. Occasionally the valve calcifies or becomes perforated or becomes overgrown with bacterial vegetations all of which contribute to the murmur.(27)

From the discussion above it is easily seen that pure "stenosis" or pure "regurgitation" does not occur. The customary finding is both, with one phase predominating.

Studies of cases of mitral stenosis in which there were varying degrees of heart block have clearly demonstrated that the presystolic murmur of mitral stenosis is produced by contraction of the auricle.(39, 22) The murmur recorded was not produced by contraction of the muscle itself, but was caused by the additional impetus given the blood by the auricle, and it was produced only when the blood passed through the valve. Stead and Kunkel (102) verified this statement and added that the intensity of the bruit depends on the difference between the auricular and ventricular pressure at the time of auricular contraction. Early, they state, in diastole the difference is marked and the murmur loud: in the latter portion of long diastolic pauses the difference is much less and the murmur either faint or absent. They

also reported that the first sound was accentuated as a result of displacement of the atrial-ventricular valves to the ventricles by auricular contraction. Wolferth and Margolies (116) also encountered this phenomenon and commented upon it.

The presystolic accentuation of the mitral diastolic murmur was formerly described as crescendo in character, but the crescendo is actually an auditory illusion as shown by phonocardiograms; the illusion is due to the combined presence of a sudden accentuation of a murmur that has suddenly died away and the sharp first heart sound that terminates it.(27, 109) It was formerly believed that the auricular portion of the murmur was the entire bruit: this is false and the distinction is generally credited to MacKenzie (64) who was the first to note that the presystolic element disappeared at the onset of auricular fibrillation.

There are no characteristics of the murmur that will not change completely with increased pathology.(10) Nevertheless, an attempt will be made to describe the typical bruit heard in the clinic.(60)

Site: The usual location is apical with maximum audibility slightly to the right and above the apex. Transmission is sharply limited to an area two or three

centimeters in diameter, a characteristic which may frequently cause the murmur to be missed. Contrary to the experience encountered with other organic murmurs the bruit of pure stenosis is not propagated.

Time: Mid-diastolic and late diastolic murmurs are typically encountered. The presystolic element may be missing in auricular fibrillation.(60)

Quality: The murmur is so low pitched and rumbling in character that Sir Thomas Lewis (61) stated that physicians recognize it by "its low pitched abruptly ending noise, much as one recognizes a dog by its bark." It changes very little in quality with posture and exercise. Wood and White (117) state that failure to recognize and diagnose mitral stenosis in most instances is due to failure to hear or interpret correctly the pathognomonic apical mid-diastolic rumble. Duroziez (28) in 1862 gave the first recognizable description of the sound as a part of a rhythm which he represented phonetically as "ffout-ta-ta-rou," but he and Potain (79) erred in ascribing it to asynchronism of aortic and pulmonic valve closure rather than to the opening snap of the mitral valve (the claquement d' overture de la mitrale of Rouche). According to Wolferth and Margolies (116), evidence indicates that this opening snap is produced by sudden limitation of the opening movement of the stenosed

valve which occurs in early diastole. This opening snap and the following rumble with the illusory crescendo is the characteristic sound of mitral stenosis. (61, 111)

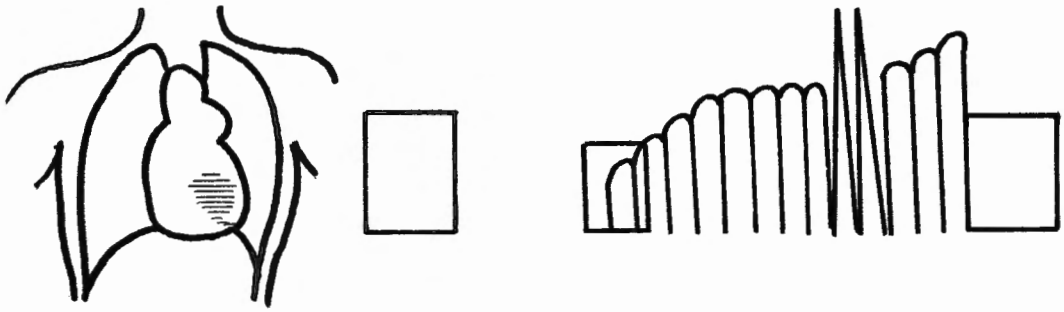

Fig. 10. Diagrammatic representation of the murmur of mitral stenosis.

In conclusion, White states that the presence of an apical diastolic rumbling murmur, with or without apparent presystolic crescendo should lead one to suspect mitral stenosis even though aortic regurgitation has not been ruled out.(111)

Aortic Regurgitation

Aortic regurgitation occurs as a result of (a) structural insufficiency of the aortic valve caused by Rheumatic Fever, Syphilis, or Arteriosclerosis, and (b) as a result of relative insufficiency following aortic dilatation, hypertension, thyrotoxicosis, and severe anemias. Organic murmurs heard in this region may also be present as a result of transmission.

Bellet (8) states that retroversion of the right anterior aortic valve leaflet rather than the usual assigned cause, i.e., rupture or tear of the aortic leaflet, should be emphasized as the commonest cause of the loud musical diastolic murmurs of this region. This lesion, first suggested by Hodgkin (49) in 1829, was found to be present in six of Bellet's patients who went to autopsy.

The typical pathology and the relationship to the murmur is similar to that discussed earlier in this paper.

The auscultatory characteristics that distinguish aortic regurgitation from other diastolic murmurs are (a) a blowing, rarely musical, quality either high or low pitched; (b) an onset with, or immediately following, the second heart sound; (c) a maximum audibility over the midsternum and immediately to the left of it in the third and fourth intercostal spaces, and usually with wide transmission to the apex, left axilla, and the neck; (d) a better perception with a diaphragm type chestpiece; (e) a better perception with the patient upright; (f) nonaccentuation of heart sounds; and (g) a continuous, holodiastolic murmur which decreases gradually in intensity. (110) Levine (58) insists that "almost invariably a systolic murmur will be heard at the base of the heart and

generally there is (one) at the apex as well." He considers the apical systolic murmur as due either to relative mitral insufficiency or to a concomitant mitral endocarditis.

The Austin Flint murmur, named after its discoverer (34), is a presystolic murmur which frequently accompanies the holodiastolic murmur of aortic regurgitation and is best heard at the apex. Its importance lies in the danger of confusion with the rumble of mitral stenosis. Herman (48) and Thayer (105, 106) theorize that the murmur is caused by the force of the regurgitant stream from the aortic valve expending itself against the anterior mitral curtain. White (111) feels, however, that the best explanation lies in a functional dilatation of the mitral valve resulting in mitral insufficiency and consequent regurgitation. This has much less evidence in support than the previous theory, and has not been generally accepted in the literature.

Other Diastolic Murmurs

Pulmonic insufficiency when it occurs is due to increased pulmonary vascular tension, and structural disease of the valve is exceedingly rare.(17) The most common cause is mitral stenosis: when due to mitral stenosis the murmur is known as the Graham Steell murmur. It is

characterized by being soft and blowing in quality, similar to one phase of the murmur of aortic regurgitation from which it is indistinguishable without the aid of other clinical findings.

Tricuspid stenosis is similar to mitral stenosis, except for the location at the lower end of the sternum. Diagnosis cannot be made on the basis of auscultation alone; the presence of other signs is necessary.

Summarizing, all diastolic murmurs are serious and merit careful investigation for even if not due to actual valvular disease they almost always indicate the presence of some serious cardiac disturbance.

CHAPTER V

THE CONTINUOUS MURMURS

Continuous murmurs may be heard over the heart in combined mitral stenosis and regurgitation, combined aortic stenosis and regurgitation, combinations of valvular lesions--particularly with the tricuspid valve--and in certain extra-cardiac lesions. The former have been discussed: the latter will be discussed under the heading of venous hums, patency of the ductus arteriosus, and arteriovenous communications.

Probably the most common and the least important cause is the mechanism giving rise to the venous hum in the neck. This murmur is very similar in nature to the systolic Eustace-Smith murmur already discussed, except that the venous hum is continuous and often even audible. (111) It has no significance and is readily recognized by the high pitch, low intensity, "mill-wheel" characteristics. Unfortunately the murmur may be transmitted to the lower portion of the sternum and be confused with patency of the ductus arteriosus.

Patency of the ductus arteriosus has a continuous bruit, frequently accentuated in systole, and heard best in the first and second interspaces to the left of the sternum.(20,21) It has a typical "machinery" quality

which aids greatly in its recognition.(111) The murmur ordinarily is localized sharply to the region indicated. Unless accompanied by cyanosis, dyspnea, or clubbing of extremities, it has little serious significance.

Arteriovenous communications possess a shrill continuous murmur usually with systolic accentuation. (21) Its interpretation is usually quite easy.

CHAPTER VI

CONCLUSIONS

In summary, the following conclusions may be reached from the evidence presented in this paper:

1. Heart murmurs are produced by a turbulence in the blood flow which sets surrounding tissues in vibration. Fundamentally the problem of evaluating the bruit depends upon determining whether the turbulence is produced by structural disease of the valve, dilatation of a heart chamber or great vessel, or decreased viscosity of the blood associated with increased velocity of the flow.

2. The stethoscope remains the best instrument for the determination of bruit characteristics.

3. The qualities of a murmur which are of the greatest importance in its interpretation are location, time, intensity, transmission, and duration. The effects of posture, exercise and the quality of pitch are secondary.

4. Systolic murmurs are difficult to interpret and to attach significance; however, they should be carefully evaluated by the characteristics listed. Constancy, transmission and intensity are factors which should influence the physician particularly. A history

of Rheumatic Fever, and the presence of hypertrophy are elements which lead to a grave prognosis. Any symptom of decompensation should color the interpretation given a murmur, no matter what the characteristics of the bruit are.

5. Diastolic murmurs are always indicative of organic heart disease. Interpretation and significance are decided by the same factors as those listed above.

6. Continuous murmurs are usually easy to interpret by the standards listed.

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