The Straining Procedure as an Aid in the Anatomic Localization of Cardiovascular Murmurs and Sounds

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Most heart murmurs diminish in intensity during straining. Those derived from the right heart and pulmonary circulation regain intensity immediately after straining. Those from the left heart and systemic circulation recover only after an appreciable delay. The murmurs of patent ductus arteriosus and of mitral stenosis each showed a modified, but characteristic response. These murmur changes were correlated with recorded dynamic cardiovascular changes produced by straining.

CERTAIN commonly used diagnostic technics are based on the relationship of murmur intensity to the rapidity and volume of blood flow. By exercise, amyl nitrite, and other means, the general circulatory velocity is increased to accentuate murmurs. If the blood flow in the right side of the heart and pulmonary artery could be altered differentially from that in the left side of the heart and aorta, then the time of murmur accentuation should provide a clue to its anatomic origin. Such a differential alteration in circulatory dynamics is provided in the relaxation period immediately following cessation of voluntary straining.

In a previous study the electrokymograph was tested as an apparatus for recording the acute circulatory disturbances produced by straining. A marked difference was shown between the recovery pattern of the aorta and that of the pulmonary artery (figs. 1–4). During straining the increase of intrathoracic pressure causes an immediate reduction of venous return to the heart. The output of the right heart decreases, the pulmonary reservoir is depleted, and the output of the left heart falls. With relaxation, the intrapulmonic pressure returns to the atmospheric level. The venous blood which has been dammed back peripherally now rushes first into the right side of the heart. The previously reduced pulsations of the pulmonary artery become vigorous at once, and the lesser circulation is restored. The output of the left side of the heart and the aortic pulsations become maximal only after refilling of the pulmonary blood reservoir. Thus, in the period of relaxation, there is a difference in the time of maximal volume flow in the two sides of the heart.

Based on these changes the following predictions were made: (1) Murmurs should be altered, becoming faint during straining and gaining intensity after relaxation. (2) Murmurs originating from the right side of the heart or pulmonary artery should become intensified very early following relaxation. (3) The restoration of intensity of murmurs arising from the left side of the heart or aorta should be delayed. (4) The differences in time of return of the two types of murmurs should be as definite as the differences in the recovery patterns of aortic and pulmonary arterial pulsations which are demonstrated in figures 1–4.

This investigation was undertaken to test these predictions and to study the behavior of murmurs and other sounds of cardiovascular origin during the straining procedure.

METHODS

Numerous patients with murmurs of various types were tested in the wards and clinics, and it was quickly found (1) that the murmurs could, in most instances, be reduced in intensity by the straining procedure and (2) that the time of return of murmur intensity varied. In order to demonstrate these changes graphically, recordings were made, in various combinations, of the heart sounds, electrokymogram, electrocardiogram, and intrapulmonic pressures. In each of the patients selected as a subject for these recordings, the anatomic location of the murmurs seemed unquestioned. Some patients had the characteristic findings of acquired valvular dis-
Fig. 1.—Normal subject L.J. Simultaneous recordings of aortic knob (upper), brachial arterial pressure (middle), and intrabronchial pressure (lower). During straining, aortic pulsations and pulse pressure are reduced, and pulse rate accelerates. After relaxation, return of amplitude of aortic pulsations and pulse pressure is gradual, and tachycardia changes to bradycardia after a few beats. (Figures 1–4 have appeared in a previous publication. 1)

Fig. 2.—Normal subject L.J. Pulmonary artery pulsations (upper), brachial arterial pressure (middle), and intrabronchial pressure (lower). During straining, pulmonary arterial pulsations are reduced. After relaxation, maximal pulsations return immediately. Changes in pulse pressure and pulse rate as in figure 1.
ease. Others were proved to have pulmonary or peripheral arteriovenous aneurysms, or one or another of the various forms of congenital heart disease.

Recording Methods. The electrokymographic equipment used in this study was of the type described in detail elsewhere. Intrapulmonic pressure was recorded by a strain-gage manometer. The sound apparatus was of standard commercial type.

All tracings were recorded on a Sanborn Tribeam instrument.

Investigative Procedure. When electrokymograms were feasible, the patient was seated before the fluoroscope and braces were applied to reduce extraneous movement. The microphone was strapped in position at the point of maximal murmur intensity. The straining procedure was a modification of the Valsalva maneuver, produced by blowing against a mercury manometer. On command, the breathing was arrested in a midrespiratory position and a short control period recorded. The subject then strained against the mercury column to a pressure of 20 or 30 mm for a sufficient time to alter the murmur. He then relaxed but did not resume respirations until the poststraining recording was completed. Throughout this period the subject kept his glottis open. Employing this standard procedure, electrokymographic tracings of the pulmonary artery or aortic knob were recorded, along with the heart sounds, and intrapulmonic pressure or electrocardiogram. In certain instances the electrocardiogram was used as the timer instead of the electrokymogram.

Such a procedure as that described above required a certain degree of patient cooperation. Some patients, especially infants and young children, could not be expected to follow the necessary instructions. Some were unable to do so because of dyspnea or weakness. Still others unknowingly supported the mercury column with oral compression, and ac-
Routinely raised their intrapulmonic pressure only momentarily, or not at all. This latter difficulty rarely caused serious trouble once we became aware of its existence. Finally, in about one individual of every 5 examined the murmur did not appreciably change in intensity despite evidently satisfactory cooperation.

Thus, production of murmur changes was difficult or impossible in some individuals. Likewise, recording of audible changes was not always satisfactory. In this study we were constantly required to demonstrate objectively that which was often much more easily heard than recorded. For example, the fainter basal systolic murmurs and the higher-pitched aortic diastolic murmurs often gave characteristic responses to straining when heard with a stethoscope, but did not show very well on tracings. Under "Results," only those murmur changes which have been recorded objectively in patients with lesions of known anatomic origin are presented.

**RESULTS**

_Effect of Straining upon Murmurs_

In general, the results bear out the predictions. When straining is effective, a definite

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**Fig. 5.**—*A*, Ventricular septal defect. Sound recordings (upper), pulsations of aorta and of pulmonary artery (middle), and intrabronchial pressure (lower). The intensity of the systolic murmur is reduced during straining. After relaxation, the intensity returns progressively, in parallel with the recovery of amplitude of aortic pulsations. The return of maximal pulmonary arterial pulsation precedes the return of murmur intensity. Note also straining tachycardia and late poststraining bradycardia.

_B*, Pulmonary A-V aneurysm. Sound recordings (upper), electrocardiograms (middle), and pulsations of the aorta and of the pulmonary artery (lower). The intensity of the continuous murmur is reduced during straining. Murmur intensity recovers immediately upon relaxation, as does the pulmonary artery pulsation. Aortic pulsations show later return. (See also fig. 6.)
reduction in murmur intensity is the rule. In patients with uncomplicated right- or left-sided lesions the two types of poststraining response can be clearly differentiated. The murmurs of mitral stenosis and patent ductus arteriosus consistently show modifications of the typical pattern of left-sided response. Patients with advanced aortic insufficiency show the typical pattern of a left-sided murmur, but fail to show certain changes of pulse rate characteristic of straining. The murmurs of patients with complex congenital lesions show variable responses as would be expected.

**Differentiation of Murmurs of Right and Left-Sided Origin.** The ability of the straining procedure to aid in the anatomic localization of murmurs was tested in patients with murmurs of unquestionable right- or left-sided origin. Subjects with murmurs of *proved right-sided origin* were difficult to obtain due to the rarity of isolated tricuspid or pulmonary valve lesions. Thus, studies were confined to one patient with pulmonary arteriovenous aneurysm and one with a large mediastinal tumor compressing the pulmonary artery. The lower tracings of figure 5 show results in a patient with pulmonary arteriovenous aneurysm. The continuous murmur becomes less intense during straining and recovers immediately after relaxation. The electrokymograms confirm that this restoration of intensity coincides with the recovery of pulmonary arterial pulsations, and clearly precedes the return of aortic pulsations. These tracings exemplify the right-sided type of response expected for a murmur known to arise from a lesion of the pulmonary arterial tree. The right-sided murmur response is also shown in figure 6.

The control group of patients with *murmurs of proved left-sided origin* included subjects with the following conditions: aortic insufficiency, aortic stenosis, mitral insufficiency, mitral stenosis, uncomplicated ventricular septal defect, patent ductus arteriosus, peripheral arteriovenous shunts, and coarctation of the aorta (both for the precordial and collateral circulation bruits). When patients with these lesions were tested, records showed left-sided response patterns in which the murmur intensity was reduced during straining and recovered grad-

![Figure 6](http://circ.ahajournals.org/)

**Fig. 6.—**Same patient as in figure 5. B. Sound recordings (upper) with amplification increased in comparison with records of figure 5. B. Electrocardiogram (middle) and intrabronchial pressure (lower). Note recovery of murmur intensity immediately upon relaxation.

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response to straining. In patients with advanced aortic insufficiency the expected changes in pulse rate were not seen. In other patients with mitral stenosis and patent ductus arteriosus, the typical pattern of left-sided response of the murmur was modified. These results deserve special mention and consideration.

1. Aortic Insufficiency: The characteristic left-sided type of response was regularly observed in association with the murmur* of aortic insufficiency (fig. 7). While the murmur itself responded in a typical manner, the pulse rate changes characteristic of effective strain-

* In this murmur especially, changes easily heard with a stethoscope were often difficult to record.

Fig. 7.—Aortic insufficiency. The diastolic murmur (M) diminishes in intensity during straining. It returns to maximal intensity on the sixth and seventh beats after relaxation. Neither straining tachycardia nor poststraining bradycardia is present. The second sound shows reduplication. The two components show independent recovery in the poststraining period. After relaxation, the first component is large in beat number 2; in beat 7 the components are equal; in beat 10 the second component is maximal (see text).

ing were not found in these patients. Normally, the pulse rate increases steadily from the onset of straining. This tachycardia persists for several beats after relaxation, and is followed by a rather abrupt bradycardia. These rate changes are well demonstrated in figures 1, 2, 3, and 4, particularly in the latter two. They are not present in figure 7, and were not seen in six of seven other cases of aortic insufficiency. This absence of pulse change is presumably the result of an abnormality of the reflex mechanisms. We emphasize that the patients tested were considered to have advanced lesions, and had been chosen for their outstanding auscultatory and peripheral signs.
2. Mitral Stenosis: This left-sided murmur tended to vary from the typical response pattern. In the early phases of the study when attention was directed particularly to the presystolic component of the murmur, most patients apparently failed to show a significant decrease of murmur intensity with straining. Successful production of rate changes during the procedure suggested that failure to strain could not account for the lack of murmur alteration. Indeed, these rate changes evidently explain the failure of the presystolic component of the murmur to show the usual strain pattern.

It is well known that an increase of rate accentuates a presystolic murmur. This accentuation is not primarily due to the general increase of blood flow, but is more closely related to the shortening of diastole that accompanies the increased rate. A presystolic murmur is louder with a short diastole, and softer or even absent with longer diastolic periods. This well-known phenomenon is demonstrated in the lower tracing of fig. 8, which shows the record of a patient with mitral stenosis and a loud presystolic murmur. During the recording, auricular premature beats oc-

Fig. 8.—A, Mitral stenosis. The intensity of the presystolic murmur (M) is seen to correlate with changes in pulse rate. Contrary to the usual left-sided response, the intensity increases during the tachycardia of straining and decreases during the bradycardia of late poststraining (see text and fig. 9, A).

B, Mitral stenosis. The opening snap sound (3) remains unchanged throughout the procedure. The presystolic murmur (M) is consistently louder with the premature beats (A) than with the normal beats. Murmur intensity is reduced during straining. After relaxation, in the premature beats the murmur is easily seen, and its intensity is sequentially increased. Conversely, in the normal beats the murmur is barely detectable and does not appreciably increase in intensity (see text).
curred, and it is obvious that the presystolic murmur is much louder for the premature beats than for the beats following the longer pauses. Clearly, in producing a presystolic murmur, the effect of auricular systole is lessened after a long diastolic period. Such a period permits passive auricular emptying and ventricular filling to a point where auricular systole adds similar premature beats. However, the flow effect is minor, relative to the effect produced by the difference in duration of diastole.

Obviously, therefore, in the average patient with mitral stenosis, the rate changes produce effects opposing the blood flow changes. The tachycardia during straining and the bradycardia during relaxation will tend to neutralize, relatively little impetus to blood movement. Thus, changes in rate, through their effect on the duration of diastole, appear to influence the intensity of presystolic murmurs to a greater degree than do changes in volume of blood flow during straining. That the increase of flow during the poststraining period has some effect can be seen by comparing the earliest premature beat of relaxation with sequential for the presystolic component of the murmur, the expected influences of altered blood flow. This is seen in the upper tracing of fig. 8 which shows the straining procedure in a patient with mitral stenosis. Straining has been effective, as evidenced by the development of characteristic rate changes and alteration of heart sounds. However, the presystolic murmur has not been decreased during straining, but is

![Graph and Diagram](http://circ.ahajournals.org/)

**Fig. 9.**—*A,* Mitral insufficiency. Typical left-sided response. The murmur intensity continues to increase during the bradycardia of the late poststraining period. (Contrast with Fig. 8, *A.*)

*B,* Mitral stenosis. The early diastolic component of the murmur (*E*) is reduced in intensity during straining and augmented in the late poststraining period in a typical left-sided response pattern. The changes in the presystolic component of the murmur (*P*) are also of the left-sided type, but are less conspicuous (see text). The opening snap sound (*3*) disappears during straining and returns on the third beat of the relaxation period.
somewhat decreased in the later relaxation period when bradycardia occurs. The upper tracing of figure 9 is included for comparison to demonstrate the typical left-sided response of a mitral systolic murmur. Note that this murmur continues to increase at the time of slowing in the poststraining period. The lower tracing of figure 9 shows the record of a patient with mitral stenosis in whom the intensity of the poststraining period. This apparent paradox is a reversal of the effects expected to result from changes of blood flow on the left side of the heart. It is apparently a pulse rate phenomenon, contingent upon the fact that auricular systole is less effective in accelerating the flow of blood after a long diastole, when the ventricle has already been passively filled, than after a short diastole, when the ventricle is

diastolic murmur decreases during straining and increases during later relaxation. Of the various portions of this diastolic murmur, the presystolic element shows less change than does the rest of the murmur. It is to be noted that the rate changes are less marked in this individual than in the others described above.

To recapitulate, in mitral stenosis, the behavior of the presystolic component of the murmur is variable. It may actually increase during straining and often does diminish in the less well filled. The early diastolic component of the murmur of mitral stenosis responds in the characteristic left-sided manner.

3. Patent Ductus Arteriosus: In this condition the elevation of aortic pressure in the late poststraining period caused murmur accentuation in a characteristic left-sided manner. As an additional phenomenon, regularly observed in our three patients, the murmur became even fainter in the immediate poststraining period than it had been during late straining (fig. 10).
The sharp rise in pulmonary artery pressure immediately after relaxation had undoubtedly resisted the inflow of blood from the aorta. Theoretically, this pattern could occur in any patient with a murmur originating from a left to right shunt, but was definite only in the patients with patent ductus arteriosus. In patients with ventricular septal defect, a similar phenomenon was suggested in some records (uppermost tracing of fig. 5), but was not a consistent or clear-cut finding.

**Effect of Straining Upon Murmurs in Congenital Heart Disease.** In certain patients with congenital heart disease, a study of the murmurs by the straining procedure is unsatisfactory. This has been especially true of individuals with cyanotic malformations and atrial septal defects, since those old enough to cooperate are often unable to strain because of dyspnea or weakness. Hence, in these particular lesions, the number of patients available for a control group is inadequate. Despite this inadequacy, some preliminary observations should be reported, pending further study.

In the cyanotic group, certain complex lesions may produce murmurs which cannot be distinguished by the straining procedure as clearly left- or right-sided in origin. The systolic murmurs of the tetralogy of Fallot and the Eisenmenger complex may not permit differentiation between that sound component arising from the ventricular septal defect and that produced on the pulmonary side. Five such patients have been tested, and the results were variable, as would be expected. Where the murmur was affected by straining, the intensity recovered partly early and partly late in post-straining.

Two patients with the tetralogy of Fallot were studied postoperatively after anastomoses of the Blalock type. The continuous murmurs of the anastomoses were not decreased in intensity during straining, preventing any demonstration of a recovery pattern. While murmurs originating from a peripheral artery ordinarily give a left-sided pattern, in these patients the aorta itself receives considerable right-sided blood and the pattern should therefore be correspondingly altered. These two patients failed to cause murmur alteration despite apparently adequate efforts to strain. A patient with atrial septal defect similarly failed to produce murmur changes. These observations have led us to speculate upon possible mechanisms by which one could account for temporary maintenance of blood flow at the site of murmur origin despite a reduction in venous return of peripheral blood to the right atrium. The suggested explanations have had varying degrees of plausibility.*

In contrast to other types of congenital lesions, patients with patent ductus arteriosus, uncomplicated ventricular septal defect, and coarctation of the aorta have proved very satisfactory for study. These murmurs show a characteristic left-sided response pattern with the modification in patent ductus arteriosus as discussed above.

**Effect of Straining upon Cardiovascular Sounds Other than Murmurs**

Phonocardiograms made during the straining procedure show that heart sounds are also altered, becoming fainter during straining and regaining intensity following relaxation. This finding may prove of considerable practical value in the study of the various so-called "extra" heart sounds. While the present experiment was directed primarily toward the investigation of murmurs, certain observations were made regarding other sounds of cardiovascular origin. These results will be mentioned only briefly here, and will be subjected to further study.

1. **Split Heart Sounds.** If split second heart sounds arise from asynchronous closure of the left and right valves, then the pattern following straining will show differences in the recovery of the two components of the split sound.

* For example, in atrial septal defect, the volume of blood shunted from the left to the right atrium might increase during straining to compensate for diminished venous return from the periphery. As a result, blood flow through the right side of the heart and pulmonary artery would be sustained sufficiently to prevent a significant change in the murmur. The reservoir of the enlarged pulmonary vascular bed could constitute a source for such a flow. Obviously, this maintenance of flow could continue only briefly, but might delay the reduction of murmur intensity beyond the capacity of the subject to maintain effective straining.
This is seen in figure 7, where, following relaxation, there is an immediate increase of the initial component of a split second sound. Subsequently this first component subsides, and the second component increases in intensity. It is believed these changes indicate that the initial component of this split second sound was of pulmonary semilunar origin.

Certain studies by Wolferth and Margolies6, 5 were concerned with the nature of splitting of the first heart sound. They concluded that "(1) the split first sound has a right ventricular and a left ventricular component, and that (2) separation of these components is due to asynchronism in certain early phases of cardiac contraction in the two ventricles." When tested by the straining procedure, split first sounds show a difference in recovery of the two components. This demonstrates that the components are of separate origin and confirms the earlier work.

2. Opening Snap Sound. The mechanism of the opening snap sound (claquement d’ouver- ture de la mitrale6) has been studied by Margolies and Wolferth.7 (This sound is often referred to as the third heart sound of mitral stenosis.) Their evidence is in accord with the hypothesis that the sound is produced by the sudden limitation of the opening movement of a stenosed mitral valve. This movement occurs when auricular pressure exceeds ventricular pressure, but before blood of significant amount flows from auricle to ventricle. Thus, the snap is heard following the second sound, and precedes by a short interval the earliest part of the diastolic murmur. The behaviour of the sound with straining is compatible with this theory of its production. If the sound depends on the sudden checking of a movement of the valve en masse, then there should be some element of "all or none," i.e., a "threshold" where the valve will move with enough suddenness and force to cause the sound, and below which it will fail to produce this effect.

As seen on the lower tracing of figure 8, there is an opening snap sound which not only fails to disappear during straining, but shows no significant decrease or increase during the procedure. In the lower tracing of figure 9, the opening snap sound has disappeared during straining, and returned on the third poststraining beat with almost the same intensity as on any subsequent beats. In some instances this sound has shown a decrease in intensity during straining, with or without disappearance of the sound. However, when it disappears and reappears, there seems to be a threshold effect rather than a more gradual change.

3. Other Miscellaneous Sounds. We have some evidence that other sounds (gallop sounds, systolic clicks, semilunar opening clicks, and physiologic third heart sounds) might be altered by the straining procedure so as to differentiate right and left origin in a manner similar to that described above for murmurs. However, further study of these various sounds is necessary before their behaviour during straining can be considered established.

Discussion

The dynamic alterations of the circulation in response to increased intrathoracic pressure are reasonably uniform and predictable. During straining, cardiac filling is impaired and cardiac output is diminished. After straining, the right side of the heart fills first, and the blood flow in the lesser circulation is promptly accelerated. Filling of the left side of the heart and restoration of the flow in the systemic circulation occurs only after an appreciable interval required for the restoration of the pulmonary blood reservoir. In these studies it is shown that variations of the intensity of murmurs conform to the dynamic physiologic changes. A reduction in intensity of all types of murmurs is the rule during the period of effective straining. When a definite reduction occurs, it is rarely difficult to differentiate the prompt return of murmurs arising in the pulmonary circulation from the delayed return of murmurs arising in the left side of the heart or systemic circulation. As described in the results, modified responses were characteristic of the murmurs associated with certain lesions.

This material is not easily adapted to quantitative analysis. Complete records were made on 38 subjects with a wide variety of cardiovascular lesions of known anatomic location. These were selected from a much larger number of individuals in whom the effects of straining
were evaluated as a simple clinical procedure. Selection was based upon the opinion of the examiner that the murmur was of suitable pitch and intensity to show on the records, that the changes induced by straining were sufficient to be easily demonstrated objectively, and that the particular lesion had not already been demonstrated repeatedly in our records. Most of our subjects had lesions of the left side of the heart and systemic circulation. Numerically, murmurs produced by uncomplicated lesions of the right side of the heart and lesser circulation are relatively rare. In this study, only 2 such subjects were recorded, one with a pulmonary arteriovenous communication, and one with a lung tumor compressing the pulmonary artery at its bifurcation. In addition, the "physiologic pulmonic systolic" murmur of children and young adults was repeatedly found to respond according to the right-sided pattern as heard with a stethoscope. Since this study was directed toward lesions of known anatomic origin, we rarely attempted objective records in these subjects, and when attempts were made, we had little success in recording the murmur, even in the control period.

In almost every textbook of physical diagnosis or heart disease, references are made to the influence of respiration upon one or another type of murmur. In the main, these references apply to changes produced by variations of the anatomic relations of the chest wall to the vascular structures. The accentuation of the murmur of aortic insufficiency in full expiration, and the appearance of some cardiorespiratory murmurs only in certain respiratory positions of the chest wall may be cited as examples of this anatomic relationship. References to murmur changes resulting from alteration of circulatory dynamics incident to respiration are much less frequent. For example, Moyer and Ackerman describe a murmur of pulmonary arteriovenous communication which was loud during inspiration and barely audible in expiration. The physiologic basis for this variation is not discussed. White believes that the "physiologic pulmonic systolic" murmur is probably associated with a dilatation of the pulmonary artery under the increased pulmonary artery pressure during full expiration or during the Valsalva experiment. Levine and Harvey likewise refer to increased pulmonary artery pressure during the Valsalva experiment as the possible causative factor in the production of a systolic murmur in a patient with an undiagnosed heart disorder. Actually, both the net pressure and the stroke change in diameter of the pulmonary artery are regularly reduced during straining, hence a dynamic basis for the murmur accentuations described by White and by Levine and Harvey is not apparent.

To our knowledge this is the first controlled study of the effects of straining upon murmurs. This method should provide a teaching exercise useful in the application of a complex physiologic response to problems in clinical medicine. It should provide a foundation for research studies of the mechanism of certain heart sounds of obscure origin. Finally, it should be helpful clinically in the anatomic localization of murmurs of uncertain etiology.

**SUMMARY**

1. Patients with heart murmurs associated with a wide variety of cardiovascular lesions were subjected to a standardized straining procedure. Heart sounds, border movements of the great vessels, intrapulmonic pressures, and electrocardiograms were recorded.

2. Murmurs were reduced in intensity during straining. The response after relaxation depended upon the anatomic location of the lesion. (a) Murmurs derived from the pulmonic circulation returned immediately. (b) Murmurs derived from the left side of the heart and systemic circulation returned only after an appreciable delay. (c) The presystolic component of the murmur of mitral stenosis and the murmur of patent ductus arteriosus each showed a characteristic modification of the left-sided response. (d) Murmurs associated with complex intracardiac shunts responded variably.

3. These changes in murmur intensity were correlated with the sequence of dynamic events during and after an increase in intrathoracic pressure.

4. Sounds other than murmurs were also affected, and a brief discussion of these is included.
5. Observations of the effect of straining upon murmurs and sounds should be useful in teaching and in the investigation of the nature and origin of obscure heart sounds and murmurs.

ACKNOWLEDGMENT

The authors wish to express appreciation to Dr. Charles C. Wolferth for his many helpful suggestions in the course of this study.

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The Straining Procedure as an Aid in the Anatomic Localization of Cardiovascular Murmurs and Sounds
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Circulation. 1950;1:523-535
doi: 10.1161/01.CIR.1.4.523

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
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