II. Clinical Aspects

MODOERATOR: DR. ROBERT P. GRANT

SOME NEWER OR POORLY RECOGNIZED
AUSCULTATORY FINDINGS OF THE
HEART

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A SUMMARY of a miscellaneous group of
clinical auscultatory findings is presented.

Ventricular Tachycardia. A succession of
sounds (usually of low frequency) are
commonly heard and are almost specific for ven-
tricular tachycardia. These extra sounds are
presumably due to wide splitting of the first
and second sounds and gallop rhythm (fig. 1).
When these sounds are combined with previ-
ously emphasized features, such as the changing
intensity of the first heart sound, the very
slightly irregular ventricular rate, and the lack
of response to carotid sinus pressure, ventricu-
lar tachycardia can be even more strongly
suspected at the bedside.

Ventricular “Knock” Sound. In mitral in-
sufficiency a sound in early diastole is produced
by the impact of an enlarged heart against the
anterior chest wall in early diastole. Simultane-
ously the palpating hand feels a prominent
impulse. This sound may be louder than even
the first or second heart sound and is often
misinterpreted as an opening snap or gallop.
The mechanism of production is felt to be
analogous to the pericardial “knock” sound
with constrictive pericarditis. Whereas the
constricting pericardial shell is responsible in

pericarditis, the chest wall is the restricting
structure with the ventricular knock (fig. 2).

Right-Sided Diastolic Murmurs of Severe
Aortic Insufficiency. Diastolic murmurs best
heard along the right sternal border rather than
the left immediately suggest unusual disease in
the region of the aortic valve or in the first
portion of the ascending aorta (fig. 3). One of
the following conditions is likely: aortic dis-
section or aneurysm, aortic cystic medionecro-
sis such as seen with the Marfan syndrome,
aneurysm of a sinus of Valsalva, syphilitic
aortic regurgitation.

Systolic “Whoop.” This phenomenon, so
named because of its similarity to the “whoop”
of whooping cough, has been observed in 3
patients, all without heart disease. Each had
a systolic click, which suddenly might be re-
placed by the “whoop” sound in systole. In
the case presented (fig. 4) the sound appeared
clinically to have no relation to position or
phase of respiration. The condition is benign.

Splitting of Second Heart Sound with Right
Ventricular Failure. As emphasized by Lea-
tham, valuable information can be obtained
by careful auscultation with particular refer-
ence to splitting of sounds. (1) A normal
increase in degree of splitting of the second
heart sound occurs with inspiration. (2) In
congenital right bundle-branch block without
right ventricular failure, wide splitting of the
second sound occurs because of delayed pul-
monic valve closure (fig. 5). In this case a
further increase in the degree of splitting
occurred with inspiration, as in normal subjects.
(3) In right bundle-branch block with right
ventricular failure, there is no increase in
splitting with inspiration. This suggests that in
right ventricular failure a point is reached when
the normal augmentation of right ventricular
filling associated with a prolonged right ven-

This is the afternoon session of a symposium held
in Cincinnati on Friday, Oct. 26, 1956. Abstracts of
the prepared talks were submitted by the speakers.
The discussion was stenotyped. The proceedings of
the morning session, devoted to the discussion of
mechanisms, appeared in last month’s issue of this
journal.
tricular systole fails to take place. For example, in a patient with mitral incompetence without right ventricular failure, wide splitting of the second sound due to premature aortic valve closure increased in inspiration due to a normal delay in the pulmonic component (fig. 6A). In mitral incompetence with right ventricular failure increase in splitting did not occur with inspiration, presumably due to absence of delay in the pulmonic valve component (fig. 6B).

Murmmur of Tricuspid Stenosis. An increase in intensity of the presystolic murmur may occur with inspiration (fig. 7).

In the same patient the simultaneous recordings of right ventricular and right atrial pressures by means of a double lumen catheter demonstrated that during expiration there was virtually no gradient across the tricuspid valve prior to atrial systole, but during inspiration as right atrial filling was augmented, the gradient appeared coinciding with the increase in the presystolic murmur.

Murmmur of Tricuspid Incompetence Masquerading as Mitral Incompetence in Patients with “Tight” Mitral Stenosis. A significant number of patients are denied surgery for mitral stenosis because a systolic murmur of tricuspid incompetence is misdiagnosed as indicative of mitral incompetence. Such patients frequently have larger hearts than usually seen with mitral stenosis. The systolic murmur may be grade III or IV (or even louder) and may be heard over the mitral as well as the tricuspid area. If due to tricuspid incompetence, the intensity of the murmur is likely to increase on inspiration (fig. 8). The murmur of mitral incompetence, on the other hand, tends to decrease with inspiration.

Mitrll Stenosis. The act of turning is important in “bringing out” the diastolic rumble of mitral stenosis. Textbooks emphasize the importance of the left lateral position in auscultation for the murmur of mitral stenosis. However, after finding the maximum arial impulse, it is of even greater importance to listen to the patient during the act of turning to the left lateral position. Sometimes the typical diastolic murmur is heard only for 6 to 10 beats as the patient turns, and then the murmur wanes quickly. If one waited until the patient had actually turned the murmur might be missed.

Discussion

Dr. Harold D. Levine (Boston): It is not necessarily calcific constrictive pericarditis in which the early diastolic sound occurs; you can get it whether or not there is calcification. It may be due to the so-called “cocktail shaker”
Fig. 3. Proven aneurysm of sinus of Valsalva in a 48-year-old woman with clinical features of severe aortic insufficiency. The murmurs are heard better along right sternal border (RSB) than along the left (LSB). SM, systolic murmur; DM, diastolic murmur.

Fig. 4. A loud systolic "whoop" (W), in patient with a normal heart, waxes and wanes in intensity (upper 3 continuous tracings). Lower tracing: systolic click (C) heard intermittently between first and second sounds (S₁, S₂).

Fig. 5. Congenital right bundle-branch block. Exaggerated asynchrony of the 2 components of the first and second sounds resulting in abnormally wide splitting. With inspiration (upward deflection of the baseline) there is further delay in pulmonary valve closure and further widening of the second sound. (This and the phonocardiograms displayed in figures 6 and 7 were taken by Dr. Perloff during his rotation through Dr. Robert P. Grant's department at the National Heart Institute.)

The motion of the heart and it may have a mechanism similar to that of the ordinary gallop.

Although the changing intensity of S₁ in paroxysmal ventricular tachycardia is quite diagnostic, there are cases of nodal tachycardia with bundle-branch block that show the same phenomenon.

I have studied a considerable number of cases of tricuspid regurgitation, mostly functional, in patients with congestive heart failure and found no change in intensity of the murmur with inspiration. There was already maximal flow across the valve and inspiration failed to produce further increase.

Dr. Aldo A. Luisada (Chicago): The correlation of the early diastolic sound in constrictive pericarditis (and in amyloidosis of the heart and cardiac fibrosis, as well) with reference recordings leaves no doubt that sudden checking of ventricular filling is the cause—whether or not we call it a protodiastolic gallop. With calcification of the pericardium the sound is higher pitched and louder.

Dr. Mariano M. Alimurung (Manila): First I am wondering about Dr. Harvey's insistence that what he terms "ventricular knock" is not a gallop. Secondly, are there any new clues to the differentiation of the Graham Steell murmur from that of aortic regurgitation?
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Fig. 8. Patient with proved tight mitral stenosis. A systolic murmur (SM) of tricuspid incompetence increases strikingly with inspiration.

pericarditis. It often is very loud, in fact often the loudest of the heart sounds. Perhaps we are stretching a point.

In deciding whether a diastolic murmur at the left sternal border is a Graham Steell murmur or represents aortic regurgitation, all the peripheral signs of the latter condition must, of course, be sought. One rule we hold to is that, if the diastolic murmur is at all audible to the right of the sternum, then it indicates aortic insufficiency.

Dr. McKusick (Baltimore): The view that hearing the diastolic murmur best along the right sternal border narrows the possibilities as to the cause of the aortic regurgitation is a special case of Balthazar Foster's rule. This "rule" is based on the premise that the direction of radiation of an aortic diastolic murmur is an indication of which cusp is primarily involved. This premise seems to have been proved unsound by the studies of Kerr and Palmer. I have not been impressed with unusual radiation of the aortic diastolic murmur in patients with the Marfan syndrome.

Splitting of Heart Sounds and a Classification of Systolic Murmurs

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Splitting of the first and second heart sounds, each into 2 major components, can be recognized by auscultation in most normal subjects. Assuming that mitral and tricuspid valve closures are mainly responsible for the first
sound,2 and aortic and pulmonary valve closures for the second, splitting of these sounds may be attributed to slight asynchrony in onset and duration of left and right ventricular systole; this has been confirmed by pressure pulses taken simultaneously from 2 ventricles and from the 2 great vessels.

Simultaneous high-frequency phonocardiograms (resembling the findings of auscultation) from the mitral and tricuspid areas show that the 2 major components of the first sound are normally separated by a narrow interval of 0.02 to 0.03 second. The first component is maximal at the mitral area, the second at the tricuspid area suggesting that mitral closure normally precedes tricuspid closure and this is confirmed by the relation of aortic ejection (indirect carotid tracing) to the first component rather than the second. Delay of right-sided events will also be found to account for splitting of the second sound in normal subjects (fig. 9).

Increase in right-sided delay from complete right bundle-branch block is associated with abnormally wide splitting of the first sound best heard at the lower left sternal edge (tricuspid area). The electrocardiogram of “partial right bundle-branch block” (QRS less than 0.12 second) is not associated per se with abnormal splitting of either the first or second sounds and we have shown that there is no delay in onset of the right ventricular pressure pulse in these cases. Splitting of the major high-frequency components of the first sound is seldom obvious in complete left bundle-branch block,18 partly because the normal slight delay in tricuspid closure makes it more difficult for mitral delay to cause obvious separation. Another explanation may be absence of delay in onset of left ventricular systole in left bundle branch,14, 15 although the total duration of left ventricular systole is prolonged. Paradoxical closure of the tricuspid valve before the delayed and accentuated sound of mitral closure may be demonstrated in mitral stenosis by taking simultaneous phonocardiograms from the mitral and tricuspid areas.

The presence of an early systolic ejection sound may simulate wide splitting of the first sound but the extra sound, which is probably an exaggeration of normal ejection vibrations, is maximal at the base, and a simultaneous phonocardiogram from the tricuspid area shows that it is preceded by both mitral and tricuspid components of the first sound. The pulmonary ejection sound is maximal in the pulmonary area and decreases in intensity during inspiration. It is associated with dilatation of the pulmonary artery from increased flow in left-to-right shunts, from mild or moderate pulmonary valve stenosis, or from pulmonary hypertension when it occurs a little later owing to the prolonged isometric time of the right ventricle. An aortic ejection sound from dilatation of the aorta is often associated with coarctation of the aorta, pulmonary atresia, aneurysm, hypertension, arteriosclerosis, and aortic valve disease.

Splitting of the second heart sound in the pulmonary area during the inspiratory phase
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of respiration is an obvious physical sign in most children and young adults, and was recognized by Potain (1866). Simultaneous phonocardiograms (fig. 9) from the pulmonary and mitral areas, together with a carotid tracing, show that the earlier component is due to aortic valve closure, and is the only one normally transmitted to the apex, and the later component is due to pulmonary valve closure and is normally isolated to the pulmonary area and immediately below it. In the expiratory phase of continued respiration the 2 valves close almost simultaneously. During inspiration the increased filling of the right ventricle from the extrathoracic venous reservoir selectively prolongs right ventricular systole, delaying closure of the pulmonary valve by as much as 0.08 second. Transmission of the pulmonary component to the apex is found when this area is over a dilated right ventricle as in atrial septal defect, or, when pulmonary closure is accentuated from pulmonary hypertension.

Abnormally wide splitting of the second sound, even in the expiratory phase of respiration, is caused by delay in pulmonary valve closure from complete right bundle-branch block, selective increase in right-sided flow as in atrial septal defect (or anomalous pulmonary venous return), and in pulmonary stenosis. In atrial septal defect the wide split (0.04 to 0.05 second) is almost fixed, scarcely varying with respiration, since little further filling of the right ventricle can be accomplished (fig. 10). There is no appreciable change in the width of the splitting directly attributable to pulmonary hypertension unless a right-to-left shunt abolishes the selective increase in right-sided flow. Pulmonary stenosis, whether valvular or infundibular, causes the greatest delay in pulmonary valve closure (up to 0.16 second or more) and the width of the splitting bears a close relation to the right ventricular systolic pressure. The splitting may be difficult to appreciate, since aortic closure is liable to be drowned in the pulmonary area where the systolic murmur is loudest, and the late pulmonary closure sound may be soft, but it can be heard and recorded in about 85 per cent of cases (fig. 11). When the pulmonary stenosis is associated with a ventricular septal defect and right-to-left shunt (Fallot’s tetralogy) pulmonary flow is further reduced and the pulmonary valve closure sound is almost invariably absent. Abnormally wide splitting of the second sound is also found in mitral incompetence, and is caused by shortening of left ventricular systole due to diminished resistance to left ventricular outflow. This is probably a factor in causing a slight increase in

Fig. 10. Atrial septal defect with left-to-right shunt. Wide fixed splitting of the second sound due to delay in pulmonary component. Midsystolic ejection murmur (SM) due to increased pulmonary flow. (Courtesy, Brit. Heart J.)

Fig. 11. Pulmonary stenosis (RV 65/5 mm. Hg). Midsystolic ejection murmur goes up to aortic closure but ceases before late soft pulmonary closure.
the width of splitting in ventricular septal defect (with left-to-right shunt) although the increased flow is affecting both ventricles.

Prolongation of left ventricular systole causes reversal of the normal order of valve closure, and thus paradoxical splitting of the second sound. This may be recognized by noticing that the split is maximal in expiration, decreasing on inspiration owing to the usual delay in pulmonary closure; the best example is complete left bundle-branch block. Paradoxical splitting of the second sound also occurs in severe aortic stenosis and in some cases of patent ductus arteriosus with left-to-right shunt.

Systolic Murmurs. Two varieties may be recognized: (1) Ejection—midsystolic—due to flow of blood through the pulmonary or aortic valve; (2) regurgitant—pansystolic—due to mitral or tricuspid incompetence, or, ventricular septal defect or patent ductus with left-to-right shunt.

Ejection systolic murmurs swell to a peak about midsystole and invariably finish before the second sound—or at least before the sound of closure of the valve where the murmur originates. This gap is to be expected, since flow must have almost ceased before valve closure. These murmurs originate at the aortic or pulmonary valve under the following condition, alone or in some combination: Stenosis of valve or infundibulum, valvular damage without stenosis, dilatation of vessel beyond, and increased flow or rate of ejection.

Aortic systolic murmurs (fig. 12) may be well transmitted to the apex; the presence of a silent interval between the end of the murmur and the second sound is a useful method of differentiation from a mitral systolic murmur. When the order of closure of the arterial valves is paradoxical as with severe aortic stenosis or left bundle-branch block the murmur may reach the earlier pulmonary closure but always stops before the later aortic closure. The greatest difficulty in recognizing the shape of the murmur is experienced when aortic closure is soft or absent as in rare cases of severe aortic stenosis.

In aortic incompetence a systolic murmur of similar shape (sometimes even accompanied by a systolic thrill) results from a stroke output which is increased both in rate and volume, across the deformed valve, while the dilated aorta beyond may be a third factor. In coarctation of the aorta the murmur due to ejection of blood through the stenosed vessel is of the same shape but may be shifted a little later.

In pulmonary stenosis (fig. 11), because of prolonged right ventricular systole and late closure of the pulmonary valve, the murmur may pass and drown the earlier aortic closure sound but always stops before the pulmonary closure sound when it can be recorded (vide supra).

The ejection murmur occurring with dilatation of the vessel beyond the valve in pulmonary hypertension is frequently initiated by an early systolic ejection sound.

Increased flow into the pulmonary artery is a frequent cause of pulmonary ejection systolic murmurs. One of the best examples is atrial septal defect (or anomalous pulmonary venous return); others are anemia, thyrotoxicosis, pregnancy, and increased stroke volume from slow rates as in complete heart block. Faint ejection vibrations can be recorded at the base in most subjects. When these become audible they probably represent the majority of innocent systolic murmurs. These murmurs are always short and soft on auscultation, finishing well before the second sound and frequently possessing a grunting or musical quality. They are most frequent in children, become louder with increased blood flow as with exertion or
be an ejection systolic murmur (and a pulmonary diastolic murmur) in the pulmonary area from dilatation of the pulmonary artery. The same mechanism accounts for the disappearance of the typical continuous murmur in patent ductus with high pulmonary resistance. In some of these cases there is a pansystolic murmur in the pulmonary area suggesting that the left-to-right shunt may be systolic only, and can be attributed to the difference in pressure and resistance between the 2 great vessels being confined to systole.

**Discussion**

**Dr. Levine:** I was surprised by Dr. Leatham's statement that he had felt a systolic thrill in cases without actual aortic stenosis.

**Dr. Leatham:** Yes. We observed 3 cases of syphilitic aortic incompetence in which there was a systolic thrill but no stenosis found at necropsy.

**Physician:** Would you comment on the intensity of the pulmonary closure sound in pulmonary stenosis?

**Dr. Leatham:** In mild pulmonary stenosis it is little if at all reduced in intensity. Possibly the dilatation of the pulmonary artery favors conduction of the sound to the surface of the chest in these cases. I find the loudness of the pulmonary component of the second sound little help in deciding severity of the stenosis; it varies a great deal with the anatomy of the chest wall, degree of dilatation of the pulmonary artery, anatomy of the stenotic valve, and so on. I find the delay in pulmonary valve closure a much more valuable index of severity.

**Dr. Franklin D. Johnson (Ann Arbor):** In connection with the splitting of heart sounds, it might be well to point out what a wonderful instrument the human ear is. I am talking now purely about things that we hear with the stethoscope. The question comes up, How close together can 2 sounds be and yet be appreciated as 2, not 1? One can investigate this with the shutter of a camera which makes 2 similar sounds, 1 of opening and 1 of closing, separated by an interval that can be set. Even at an interval of a fiftieth of a second (.02 second) 2 sounds can be distinguished; at shorter intervals, they tend to merge. But it is an individual...
matter. I am sure some people can appreciate a split as close as .01 second.

Dr. Joseph K. Perloff: The slide of congenital right bundle-branch block shown earlier (fig. 5) demonstrates splitting of the first heart sound. Of the 2 components the first is louder at the apex and the second at the sternal edge. So it is thought that the sources are mitral closure and tricuspid closure, respectively.

Effect of Respiration and Upright Position on the Interval Between the Two Components of the Second Heart Sound and that Between the Second Sound and the Mitral Opening Snap

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When the pulmonary valve closes later than the aortic valve, the interval between the 2 components of the split second sound seldom exceeds 0.04 second in normal adult individuals. However, it may occasionally exceed this value in healthy teen-agers, as well as in conditions accompanied by an increased blood flow through the right ventricle and in the presence of right bundle-branch block. In these situations, the second component of the split second sound may be confused with the opening snap of the mitral valve.

The duration of the interval between the second sound and the opening snap usually measures 0.07 to 0.11 second. However, this interval can be as short as 0.03 or 0.04 second, especially in patients with a very reduced mitral valve area or rapid heart rate. An early opening snap may be confused with the second component of the second heart sound.

It was thought that these two situations could be distinguished more readily if the conditions of the examinations were altered. The following reasoning was applied: Conditions that increase the flow of blood into the right heart lead to a prolongation of the interval between the 2 components of a split second sound as they cause further delay in the closure of the pulmonary valve. The same conditions will cause increased filling and pressure in the left atrium* and thus an earlier opening of the mitral valve. On the other hand, conditions that decrease the flow of blood into the right heart lead to a shortening of the interval between the 2 components of the second sound caused by the late closure of the pulmonary valve. Since these conditions will also lead to a decreased filling of the left atrium, they will be expected to cause a later opening of the mitral valve and to increase the interval between the second sound and the opening snap.

Nine patients with established mitral stenosis and an audible opening snap and 15 persons with a split second sound due to late closure of the pulmonary valve in whom the interval between the 2 components of a split second sound exceeded 0.04 second, were studied. Phonocardiograms were recorded from the point where the opening snap or the second component of the second sound was heard best, using a Sanborn Twin-Beam Phonocardiograph with logarithmic setting of the amplifier. The

* Ed.: At least in normal subjects inspiration might be expected to have opposite effects on filling of the 2 sides of the heart.
recordings were made during quiet normal respiration and during held inspiration, at first in the recumbent position, then immediately after standing up.

The following results were obtained: The effect of held inspiration and held expiration on the interval between the 2 components of the second sound conformed with the well-known results of previous observations, namely lengthening in inspiration and shortening in expiration.11,16 The opening snap showed a tendency to appear later after prolonged expiration and earlier after prolonged inspiration. However, these variations did not appear to be consistent and, moreover, the results were obscured by variations of the heart rate which took place during respiratory maneuvers. The effects of changing heart rate on the timing of the opening snap have been described by several investigators; they consist of earlier appearance of the snap after a short diastolic interval and a later appearance after a long

Fig. 15. A. Phonocardiogram and lead II of the electrocardiogram of a 34-year-old man with atrial septal defect. The calculated left-to-right shunt was 6.62 L. per minute and the right ventricular pressure was 49/8 mm. Hg. Note the wide splitting of the second sound (0.06 second) in the recumbent position and a decrease in splitting (0.03 second) in the upright position. The heart rate is almost identical in both tracings. B. Phonocardiogram and lead II of the electrocardiogram of a 24-year-old woman with atrial septal defect. The calculated left-to-right shunt was 8.96 L. per minute, and the right ventricular pressure 42/10 mm. Hg. The electrocardiogram shows an incomplete right bundle-branch block with QRS duration of 0.11 second. Note the wide splitting of the second sound (0.07 second) in the recumbent position and a decrease in splitting (0.04 second) in the upright position. The heart rate is nearly identical in both tracings.

Fig. 16. A. Phonocardiogram and lead II of the electrocardiogram of a 42-year-old woman with rheumatic heart disease and mitral stenosis. The calculated mitral valve area (Gorlin’s formula) was 0.6 cm.², and the pulmonary artery pressure 56/28 mm. Hg. Note that the duration of the interval between the second sound and the opening snap (OS) measures 0.06 second in the recumbent and 0.09 second in the upright position. The heart rate is nearly the same on both occasions. B. Phonocardiogram and lead II of the electrocardiogram of a 43-year-old woman with rheumatic heart disease and mitral stenosis. Calculated mitral valve area = 1.2 cm.²; pulmonary artery pressure = 37/23 mm. Hg. Note that the duration of the interval between the second sound and the opening snap (OS) measures 0.09 second in the recumbent and 0.11 second in the upright position. The heart rate is more rapid in the upright position.
diastolic interval. No variations of the timing of the opening snap were noted during respiration by Mounsey; however, presence of variations was reported by Schoelmerich and Gehl. In the upright position shortening of the interval between the 2 components of the second sound and lengthening of the interval between the second sound and the opening snap occurred in nearly all examined cases. The changes produced by standing were more marked than those produced by changing the respiratory position (fig. 14A). A shortening of the interval between the second sound and the opening snap in the upright position was described earlier by Schoelmerich. In the present study, the greatest differences in the duration of the discussed intervals were seen when the phonocardiogram taken in the recumbent position during held inspiration was compared with the phonocardiogram taken in the upright position during held expiration. However, unequivocal differences were usually noted between the phonocardiogram taken during quiet respiration in the recumbent and upright position. These differences frequently took place without any change in heart rate (figs. 15 and 16A). Figure 16B shows a prolongation of the interval between the second sound and opening snap in the upright position at the time when the heart is more rapid. In this case one would expect an earlier appearance of the opening snap with a more rapid heart rate, but the effect of upright position apparently nullifies the expected effect of the increased heart rate.

Clinical Survey

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Spectral phonocardiograms (SPCG) will be used to illustrate the following comments. In these displays of sound, time is on the abscissa and frequency on the ordinate; intensity is represented as degree of blackness in given portions of the display. These records appear to have the following advantages, all resulting from the spreading out of frequency: 1. Fine splitting, i.e., separation of the components of complex transients, can be more easily identified—to an interval of .01 second or less under ideal circumstances. 2. Timbre is represented—a feature which renders the method useful for the study of musical murmurs (see below). 3. A loud and a faint murmur can be displayed in the same record in something more nearly approaching their true intensity proportions. 4. Artifacts are more easily identified.

The method has, however, not yet come of age, from the standpoint either of instrumentation or of clinical application. In my opinion an adequate instrument is not presently available commercially. Desirable for such an instrument are the following: (1) flexibility in the manipulation of the time axis without influencing the frequency scale—so that these sounds can be stretched out for closer analysis; (2) possibly photographic display for improved intensity grading; (3) improved analysis in the low frequency range—below 60 or 100 c.p.s.; and (4)—at least for a research instrument—facilities for putting multiple channels of information on the record, e.g., 2 spectral phonocardiograms, conventional oscillographic phonocardiogram, carotid pulse, etc., in addition to the electrocardiogram, which is the barest essential for any instrument.

Musical Murmurs. The SPCG provides a physical definition for musicality in murmurs: instead of the rather diffuse frequency pattern of the usual noisy murmurs, harmonic pattern characterizes musical murmurs. Justification for dividing murmurs into noisy and musical types is provided by 2 rather distinctive features of musical murmurs as a genus: 1. A structural member which is thrown into vibration can be identified post mortem in most, perhaps all instances—retroverted aortic cusp, aberrant ventricular tendon, stenotic aortic valve diaphragm, rubbing pleuropericardial surfaces, etc. Although fluid factors and boundary structural factors are obviously of significance in the generation of both noisy and musical murmurs, fluid factors seem dominant in the case of noisy murmurs and the structural factor in musical murmurs. 2. Musical murmurs include some of the loudest varieties of cardiovascular sound; e.g., the musical murmur of retroverted aortic cusp or of calcific aortic stenosis may be audible at a distance from the body.
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Fig. 17. The counterparts in musical instruments of the generators of musical murmurs in man. A. Bass viol (violin family in general); analogous to musical pleuropericardial murmurs. B. Harmonica; not strictly analogous to retroverted aortic cusp, since the reed faces into the wind not downstream. Reed a is activated on blowing and alternate reed b on sucking. C. Trumpeter’s lip; analogous to calcific aortic stenosis. D. Aeolian harp; analogous to anomalous chordae tendineae. E. Reed of grass. More like a downstream reed but differs in that the fluid stream passes on both sides of the vibrating member.

By analogy of the generator to musical instruments (fig. 17), musical murmurs can be divided into 4 major varieties and a fifth mixed group: 1. The retroverted aortic cusp resembles the reed instruments in general principle but differs from all modern instruments of this type in the fact that the reed faces downstream not upstream. 2. Calcific aortic stenosis resembles the trumpeter’s lip plus mouth-piece—a combination capable of generating musical tones (fig. 18). 3. Aberrant tendons of the ventricle resemble the Aeolian harp. 4. Rubbing pleuropericardial surfaces may, under some circumstances, resemble the violin group of instruments. 5. Certain musical peripheral vascular murmurs, some arterial, some venous, some probably arteriovenous, cannot be precisely analogized in this manner.

Examples of each type are presented. We have not studied the heart sounds in a patient proved to have aberrant tendon of the ventricle of the moderator band type, which Huchard described as a cause of musical murmur, but have found musical elements in the systolic murmur of ventricular septal defect. These may be produced by aberrant tricuspid chordae tendineae, which sometimes span the defect.

We have had a varied and interesting experience with musical murmurs that we believe are extracardiac in origin. Some of these, we think, fall in the group of murmurs just referred to by Dr. Harvey as “cardiac whoop.” The features of this musical murmur (fig. 19) are as follows: (1) presumed innocence, judging from the usually healthy state of the subject; (2) late systolic timing; (3) association with a systolic click; (4) variation with respiration and with increased vigor of the heart beat—specifically accentuation with inspiration and following exertion or emotion; (5) development following pericarditis, such as the benign nonspecific variety; and (6) in some cases at least, the presence of mild pectus excavatum in the subject. The SPCG shows harmonies and the conventional PCG a regular periodic pattern at the rate of the fundamental. The evidence for extracardiac, specifically pleuropericardial, origin is (1) the clinical setting (point 5 above), (2) the association with systolic click (point 3) which from other evidence is judged to be of pleuropericardial origin in many instances, and (3) the fact that the murmur may extend over the second heart sound slightly into diastole (cf. fig. 19).

In partial occlusion of arteries a continuous musical murmur in which the fundamental displays the pattern of an arterial pressure pulse may occur (fig. 20). (In other instances a more complex pattern is present.) Uterine souffle may show the same pattern. Venous hums are occasionally musical (fig. 21). The mechanism of the musical murmur of partial arterial obstruction may be identical to that of the “musical murmur” of bronchial asthma, which in the sound spectrogram is characterized by harmonics; the principle of Bernoulli,
SYMPOSIUM ON CARDIOVASCULAR SOUND

**Fig. 18.** A. Tone of 440 c.p.s. tuning fork and, on right, 60 c.p.s. calibration signal. B. Musical sound produced by blowing on a reed of grass. Note that the second harmonic is louder than the first, which is presumably the fundamental. This was also the situation in the case of the musical tone produced by a silk thread across a water-conducting tube. C. Trumpet mouthpiece. The tones are not steady and impurities in the form of additional partials are present in certain notes. D. Violin with portamento (glide) from about 150 c.p.s. up to about 440 c.p.s. A brief segment of speech sound is also present and a 60-cycle calibration. E. Full trumpet. Here the tones are steady and pure. F. Musical sounds of a bass viol. G. Recording of musical sound produced by blowing on isolated oboe reed. These are analyses of the same sound on 3 different frequency scales. Below 720 c.p.s. there is nothing except wind noise. The fundamental, located at 960 cycles, appears in the analysis to 1,440 c.p.s. A total of 3 overtones is demonstrated in the third analysis.

as adapted to this situation by Rodbard, is probably operative. In general it appears that it is in the musical murmurs that Rodbard's "flitter" is most clearly involved as a mechanism; however, it seems to the speaker that to view these murmurs as a composite of a large number of closely timed transients is of doubtful usefulness although in a sense perhaps accurate.

**Miscellaneous Auscultatory Phenomena.** With inspiration not only may the diastolic murmur of tricuspid stenosis be greatly accentuated but also the interval between the second sound and the onset of the murmur is likely to be shortened. Increase in right atrial filling with inspiration is a satisfactory explanation.

Occasionally in patients with aortic stenosis and regurgitation one hears over the lower sternum a continuous machinery murmur simulating, in quality, that of patent ductus arteriosus, although the same patient demonstrates the usual double to-and-fro murmur in the aortic and pulmonary areas. The mechanism of the continuous murmur, which also in
the SPCG simulates that of patent ductus arteriosus, is unclear.

The murmur in one case of rupture of a sinus of Valsalva into the right atrium was continuous in character simulating patent ductus arteriosus on stethoscopy although in the phonocardiogram the intensity-frequency peak of the murmur was later than in patent ductus arteriosus. The murmur disappeared completely after successful surgical closure of the communication.

In a case of mitral obstruction due to left atrial myxoma the mitral first sound was snapping and moderately delayed (Q-S₁ = 0.08 second) and the pulmonic second sound was accentuated as in conventional mitral stenosis. A possible opening snap was present. Further phonocardiographic experience is necessary before it can be stated that the simulation of mitral stenosis by myxoma extends to the opening snap.

With ventricular septal defect the systolic murmur is likely to be holosystolic* when pulmonary hypertension is not present but becomes limited more and more to the earlier portion of systole as pulmonary hypertension progresses and may disappear entirely with severe degrees of pulmonary hypertension.

That a late systolic click may occur on the basis of pleuropericardial adhesion is suggested by its occurrence following clinical acute pericarditis, of rheumatic origin, for example. Clinical import derives from the possibility of mistaking the click and second sound for the second sound-opening snap combination of mitral stenosis.

* There is a tendency to use pansystolic and holosystolic interchangeably, to indicate a murmur filling the entirety of systole. It may be argued that holosystolic is preferable since pan- carries a connotation of "each." Pansystolic could mean a murmur occurring with each systole, whereas holosystolic can mean only a murmur filling systole.
Clinical Quantification of the Intensity of Heart Sounds and Murmurs: The Transmission of Basilar Systolic Murmurs

Eugene Lepeschkin, M.D., Burlington, Vt.

Quantification of Intensity. The introduction by Dr. Samuel Levine of a system of grading systolic murmurs according to their loudness has been helpful in the quantitative treatment of auscultatory phenomena. However, some confusion is still present due to the use of the original 6-grade system by some cardiologists and of a 4-grade system by others. Of the 62 cardiologists present at this meeting, 44 indicated that they use 6 grades, 8 that they use 4 grades, and 10 that they use no system of grading. Still, the identification of the grade of a given murmur is a highly individual process, and it would be of advantage to develop a more objective means of measurement. Ten years ago I built the first model of an attenuating stethoscope, in which the opening between the bell and earpieces could be gradually occluded until the murmur or heart sound could no longer be heard; the loudness of the murmur then corresponded to that of the audiometer sound, which could be just heard through the same opening. The instrument was perfected in 1950,29 and through consultation with Dr. Levine it was found that each grade of his system corresponded to an increase in loudness of approximately 12 decibels.

However, the stethoscope had several disadvantages. The opening was so fine that it was easily plugged by dirt or moisture, and the clearance of the moving parts was so critical that the instrument could not be mass-produced. Moreover, it could not attenuate sounds of an intensity more than 60 decibels because...
the sounds of greater loudness were conducted through the wall even when the opening was closed. Finally, attenuation was always accompanied by a change in pitch. For these reasons, the amplifying electric stethoscope* was calibrated and modified to provide an amplification (or attenuation) of +15 to −100 decibels. This modified instrument avoids the disadvantages enumerated above, and, in addition, has the advantages of being able to amplify subthreshold murmurs and of eliminating the occasionally very disturbing sounds produced by rubbing of the stethoscope tubes against each other or the clothing of the listener.

The amplifying stethoscope we used has a peak sensitivity at about 500 c.p.s., similar to that of a binaural stethoscope with membrane chest piece, and it was calibrated at this frequency by means of a Western Electric audimeter. Measurements in normal persons showed that the loudness of the first heart sound ranges from 20 to 60 decibels at the point of maximal intensity, the loudness of the second heart sound ranges from 30 to 70 decibels at the aortic and 25 to 65 decibels at the pulmonic area, while the loudness of systolic murmurs ranges from −20 (inaudible range) to 40 decibels at the point of maximum intensity. Certain high-pitched, musical systolic murmurs may reach a loudness of 50 decibels. In general, persons overweight or those with pulmonary emphysema showed lower values for loudness than thin persons and children. Correlations with pulse pressure, blood viscosity and conductivity of the chest for a calibrating sound source are being made.

Propagation of Murmurs. One interpretation

* Maico Co., Minneapolis, Minn.
for the observation that the systolic murmur of aortic stenosis is well heard over the subclavian and carotid arteries is that it is carried by the blood stream, just as distant sounds may be carried by the wind. The velocity of sound in air is about 330 M. per second, and a moderately strong wind of 10 M. per second, blowing in the direction of the listener, would reduce the distance traveled by the sound per second by about 3 per cent. In the case of blood, however, the systolic velocity is at the most 0.4 M. per second while the sound velocity in water is around 1,400 M. per second, and this would lead to reduction of only 0.03 per cent in the distance traveled by the sound. It is therefore improbable that flow of blood in the direction of the point of auscultation should cause a measurable increase in the intensity of the murmur heard at this point. This conclusion was confirmed by an experiment in which a latex rubber T-drain was perfused with water and a point of stenosis was produced in the lower branch at its origin from the top section of the “T” (fig. 22). The clamps were adjusted so that the flow through the stenosis was equal to that past the stenosis. The intensity of the murmur was measured along the surface of the tube by means of the calibrated amplifying stethoscope described above. As can be seen from figure 21, the attenuation of the murmur is only 10 decibels within a distance of 10 cm. downstream from the stenosis, while upstream from it the attenuation is 70 decibels along the same distance. In this upstream section, in which water flows quietly past the stenosis, the attenuation is practically the same upstream or downstream, although the velocity of flow in this section is equal to that in the section downstream from the stenosis. The propagation of the murmur downstream from the stenosis with almost undiminished intensity can therefore not be due merely to the direction of the flow. The best explanation for this propagation is that the murmur is not conducted from the point of stenosis but actually recreated at each point of the tube wall by the vibrations in the disturbed flow pattern set up at the stenosis and continuing down the entire length of the tube.

In 5 cases of aortic stenosis, the average decrease in the intensity of the systolic murmur from the aortic area to the right carotid artery (a distance of about 20 cm.) was 15 decibels while the peak intensity showed a delay of 0.06 second (fig. 23). This corresponds to a conduction velocity of about 3.5 M. per second.

![Fig. 22. Loudness of murmur, in decibels above hearing threshold, in the vicinity of a stenosis (S) in a perfused rubber tube. Direction of flow is indicated by arrows.](image1)

![Fig. 23. Phonocardiogram of a patient with rheumatic aortic stenosis, demonstrating delay in the maximum of systolic murmur at the right carotid artery with respect to the aortic area.](image2)
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similar average delay was found earlier by Kerr and Harp.^{30} If the murmur were transmitted to the carotid artery by conduction at the speed of sound, the difference of intensity would have been much greater and no appreciable delay would have occurred, as in the case of a loud murmur of pulmonary stenosis (fig. 24). On the other hand, if the murmur were caused by the turbulent blood ejected through the stenosis arriving at the carotid artery, the delay would have been much greater. The speed of conduction of the murmur actually approximates that of the pulse wave, as previously suggested by Kerr and Harp.^{30}

Discussion

DR. LEATHAM: In grading murmurs why not fractionate? One can say that a murmur is “grade 4 out of 6,” written “4/6,” or “grade 2 out of 4,” and so on. There is then no confusion as to whether a base of 4 or 6 grades is used.

DIFFERENT TYPES OF DIAMOND-SHAPED BASAL SYSTOLIC MURMURS

CHRIST ARAVANIS, M.D., Chicago, Ill.

Seventy patients with loud aortic systolic murmurs were investigated by clinical, roentgenologic, and graphic means.

The tracing of the suprasternal notch, the carotid tracing, and the aortic kymogram presented data (slow rise, flat top or anacrotic notch, multiple vibrations) that were helpful in the differential diagnosis between obstructive stenosis and minor, nonobstructive aortic lesions, or relative aortic stenosis.

The study revealed that no statistical significance could be attached to the amplitude of the murmur and its distance from the first and second sound, or to the amplitude of the second aortic sound. The shape of the murmur, on the contrary, seemed important: 87.5 per cent of cases with relative stenosis had an early-diamond (i.e., early peak) and 66.7 per cent of cases with organic stenosis had a late-diamond (i.e., late peak). A late beginning and a late termination of the murmur, and a small, delayed second aortic sound are more often found in organic, obstructive valvular stenosis than in relative or nonobstructive stenosis.

Discussion

DR. DAVID H. LEWIS (Philadelphia): Am I correct in understanding that the differentiation of relative from organic aortic stenosis—on which this study was based—was made from the shape of the carotid pulse? I take it you do not have tracings of intra-arterial or aortic pressure.

DR. ARAVANIS: It has been demonstrated that the shape of the pulse in the substernal notch corresponds closely to that of the aortic pressure pulse. Furthermore, it has been demonstrated that the severer the aortic stenosis, the greater is the delay in the peak of the pulse wave.

Heart Sounds in Total Anomalous Pulmonary Venous Return and in Ebstein’s Anomaly

P AtliCk A. Ongley, M.D., Boston, Mass.

Complete Transposition of the Pulmonary Veins. The general clinical picture associated with complete transposition of the pulmonary veins is well known. Pulmonary flow is much greater than normal and so murmurs due to increased flow through the great veins and through the tricuspid and pulmonic valves can be expected: (1) A systolic murmur over the pulmonic area; (2) a mid-diastolic-presystolic
murmur over the tricuspid area; and, (3) frequently a continuous murmur or venous hum over the great veins. There is often a third sound secondary to the increased early right ventricular filling and a fourth sound may result from increased force of right atrial contraction. With the rapid heart rates encountered in infants and children a summation gallop may result.

These characteristic sounds and murmurs have been encountered in those cases of complete transposition of the pulmonary veins in which drainage is into either the right or left superior vena cava, into the coronary sinus, or directly into the right atrium (fig. 25). We have not heard these murmurs when drainage has been below the diaphragm into the portal vein or the ductus venosus.

Ebstein's Anomaly. Ten cases of this anomaly have been studied clinically and during the past 5 years phonocardiograms have been recorded in 6 of them. The findings have been fairly consistent (fig. 26).

The first sound is of normal intensity and is best heard at the apex. The second heart sound is best heard at the lower left sternal border or at the apex and it is definitely diminished at the pulmonic area. In spite of a marked degree of right bundle-branch block the second sound was either narrowly split or unsplit in 3 of the patients and only normally split in 3 others. A prominent third sound was present in all patients. A fourth sound was occasionally present. In those patients with a short diastole the third and fourth sounds may be summated.

All patients showed a systolic murmur of moderate intensity, of medium frequency, and of either decrescendo or crescendo-decrescendo configuration. It was best recorded about the mid or lower left sternal border.

All patients showed a presystolic murmur which because of the prolonged P-R interval

![Fig. 25. Total anomalous pulmonary venous drainage. Prominent first sound (1); normal second sound (2); low-intensity third sound (3); medium frequency, moderate intensity, systolic murmur (SM); medium frequency, moderate intensity, presystolic murmur (PSM).](image)

![Fig. 26. Ebstein's anomaly. Prominent first sound (1); well split second sound (2); prominent third sound (3); decrescendo, medium frequency, moderate intensity, systolic murmur (SM); mid-diastolic murmur (DM), following the third sound, and the P wave of the electrocardiogram.](image)
was not crescendic in quality. In some patients this murmur was quite intense and had a rather "scratchy" quality on auscultation.

Middiastolic murmurs at the apex were infrequently encountered and were of low intensity and medium to low frequency.

Discussion

Dr. Luisada: Was atrial septal defect present in these children?

Dr. Ongley: Of course. All cases of total anomalous pulmonary venous return, to survive, must have a septal defect.

Dr. Luisada: And the Ebstein cases?

Dr. Ongley: Some have an interatrial communication; some do not.

Dr. Luisada: The reason I ask is that in our cases of plain atrial septal defect 1 out of 3 or 4 showed a murmur in presystole completely separate from the first sound and usually recorded at the base in either the pulmonary or aortic area. We thought this murmur might be produced by the passage of blood through the defect.

Dr. Ongley: We have felt that the murmur you refer to in atrial septal defect is tricuspid in origin. On catheterization the pressure differential between right atrium and right ventricle is more impressive than that between left atrium and right atrium.

Dr. McKusick: I have an isolated observation in total anomalous venous return and some observations on Ebstein's anomaly. This recording (fig. 27) is from a 21-year-old patient who for years was followed as probably having a patent ductus arteriosus, atypical in that the

Fig. 27. Intense murmur has greatest amplitude and frequency span in the region of the second heart sound (II) as in the murmur of patent ductus arteriosus. Recorded under midlavigle in first right interspace.
murmur was maximal under the right clavicle, where this recording was made. The murmur was harsh and continuous, with none of the fickleness of a venous hum, and, in brief, with all the auscultatory features of patent ductus. Cardiac catheterization and angiocardiography have established the diagnosis of the "figure-eight," "cottage-loaf" or "snow-man" type of total anomalous venous return.

The striking gallop, occasionally double and resulting in quadruple rhythm, is the feature which has impressed us about the Ebstein cases. This feature seems quite characteristic and among patients with cyanotic heart disease immediately suggests this particular malformation to the stethoscopist. Midsystolic clicks also occur producing rather bizarre combinations. In the second place I am doubtful that tricuspid regurgitation accounts for the systolic murmur in all cases of the Ebstein anomaly. A circumscribed midsystolic murmur (fig. 28) with the shape of an ejection systolic murmur suggests that forward flow through the malformed tricuspid valve may be occurring. Blount 31 infers from intracardiac pressure patterns that the pretricuspid chamber contributes to propulsion of blood into the pulmonary artery.

CONCLUDING COMMENTS

FRANKLIN D. JOHNSTON, M.D., Ann Arbor, Mich.

I first became interested in sound records a great many years ago. As a matter of fact, the interest in sound records was the thing that originally got me into the Heart Station with Dr. Wilson, and I took a great many of them for a number of years. But about 10 years ago I had nearly decided that sound records were not worth doing, at least very often. Then Dr. Leatham and his associates in England, and other people, began to publish beautiful tracings, and not only publish the tracings but point out from these tracings the very interesting auscultatory findings that can be derived from them. As a matter of fact, and I think this is true of many other people in this country, our interest in sound tracings has been revived.

It is not the tracings themselves that are
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really important; it is the things that they teach us. You see a few of these records and the next time you see a patient you are paying attention to the splitting of the first sound and the splitting of the second sound, and the chance of making a tentative clinical diagnosis of left branch block, let us say. I think that is the value of phonocardiography and I think it is wonderful.

EDITORIAL SUMMARY AND CONCLUSIONS

No attempt will be made to summarize the numerous and interesting clinical observations that were reported in this part of the symposium.

In the concluding remarks Dr. Johnson emphasizes the educational value of phonocardiography. Its usefulness in 2 other respects should be indicated: 1. Inasmuch as phonocardiograms provide information that eludes the best-trained ear, they have scientific value. They permit precise time measurements and demonstrate sounds of low-frequency content beyond the perception of the ear. Other types of meaningful information may be provided by new methods, such as spectral phonocardiography, now under study. 2. Phonocardiography provides valuable documentation, particularly in cases of valvular heart disease, bearing on the course of disease and the effects of cardiac surgery. It keeps the clinician honest. Recently Dr. William Dock made the cogent statement: “We should no more think of sending a patient for valve surgery without a phonocardiogram than one would in the ‘old days’ think of sending a patient to Trudeau without a chest x-ray.” Dock further stated in his Porter lecture:

The permanent objective records supplied by phonocardiograms are as valuable in managing heart disease as blood smears in leukemia, chest films in pulmonary tuberculosis, or electrocardiograms in coronary disease. Perhaps their greatest value is in teaching us to be better doctors at the bedside and in the office, and by making us less prone to error when the phonocardiogram is not available.

Part of the advantage of objective documentation is the possibility of studying the records at leisure, and again and again. Ability to represent the auditory impression on the printed page is no mean aid to education and scientific progress.

The objective of phonocardiographic research should be to provide precision instruments and methods that can supplement, not merely embellish, the bedside diagnostic findings.

SUMMARY IN INTERLINGUA

Non es essayate summarisar le multe e interessante observationes clinic reportate in iste parte del symposio.

In su remareas conclusori, Dr. Johnson sublinea le valor pedagogic del phonocardiographia. Su utilitate ab 2 altore punctos de vista deberea esser signalate. (1) In tanto que phonocardiogrammas provide information que escappa al plus experte aure, illos possede valor scientific. Illos permitte precise mesurationes de tempore e demonstra sonos de frequentias infra le limine de perception per le aure. Altere typos de information significative va possibilmente esser obtenite per medio del nove methodos, per exemplo le phonocardiographia spectral, que es currentemente sub investigation. (2) Le phonocardiographia provide un documentation de valor—specialmente in casos de morbo de valvula cardiac—con respecto al curso del morbo e le effectos de chirurgia cardiac. Illo protege le honestitate del clinico. Recentemente Dr. William Dock faceva le pertinente constatation: “Nos debe tanto paucio pensar a inviar un patiente al chirurgia valvular sin un phonocardiogramma como in dies passate on haberea pensate a inviar un patiente a Davos sin un roentgenogramma thoracic.” In su conferentia Porter, Dock diceva in plus:

Le permanente documentation objective providite per phonocardiogrammas es tanto utile in le tractamento de morbo cardiac como frottis de sanguine in leucemia, roentgenogrammas thoracic in tuberculose pulmonar, o electrocardiogrammas in morbo coronari. Le plus grande valor del phonocardiogrammas es forsan que illos insenia nos a esser meiior medicos in le situation clinic e in le sala de consulta e que illos reduce le possibilitate de nostre committer errores quando phonocardiogramma non es disponibile.

Un parte del avantage de un documentation objective es que on pote studiar lo deliberate e repetitemente. Le possibilitate de representar
le impression auditori in forma imprimite non 
est un detatle de neglible adjuta in le instruc-
tion e in le progresso scientific.

Le objectivo de recerca phonocardiographie 
deberea esser le provision de instrumentos de 
precision e de methodos exacte que servi a 
supplementar e non solmente a adornar le 
constatationes diagnostic del clinico.

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