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Intracardiac Phonocardiography:

RECENT OBSERVATIONS

By

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Our recent results obtained by intracardiac phonocardiography are reviewed. As one of the factors in production of murmurs abnormal turbulence within the heart and great vessels must be taken into account. As a morphological proof of this turbulence the so-called jet lesion can be considered. Some examples of normal humans, experimental dogs, human pathological states and that of the congenital heart diseases are demonstrated. The cases discussed on this occasion are those which presented various problems and interpretations are attempted based mainly on intracardiac phonocardiograms.

AT THE ANNUAL Meeting of Jap. Circulation Society in 1953 we have presented the method of intracardiac phonocardiography using dogs and have shown recordings of actual sounds obtained from heart cavity of man.¹⁾ It was emphasized then that this method is especially of value in the diagnosis of congenital heart diseases.²⁾³⁾¹⁴⁾ Our method is as presented in Fig. 1. It is essentially a method where the body serves as one electrical pole in the condenser microphone system as developed uniquely in Japan by the group under the late Professor Mashimo of Kyoto University.⁴⁾ However, since

in our apparatus a thin shielded wire was used within the catheter as a lead and one pole placed near the tip of the catheter, strictly speaking the two cannot be said to be the same. By means of this improvement, it became possible to pick up the changes in capacity of only the catheter tip. A year later Soulie⁵⁾ of France, by using a tiny electro-magnetic pressure gauge, and Lewis⁶⁾, three years later with a miniature barium titanate microphone at the catheter tip at almost the same time as Moscovitz⁷⁾ who utilized only the perpendicular forces to the axis of the same ceramic microphone, successfully recorded the intracardiac sounds of man. Independently of these workers Luisada, Liu⁸⁾ by using a Statham P-23D type transducer sensitive to high frequency waves situated outside body were also successful in recording these sounds by using only ordinary catheters. Luisada's method enables a simultaneous sampling of blood specimen and recordings of pressure waves but Lewis too has now made a commercially available double lumen catheter to be used for these purposes. We have tried using the double lumen catheter on many occasions

Fig. 1. Wiring diagram.

but presumably because of the penetration of fluid on to the lead section we have been successful in recording only a few cases. The intracardiac (i.c.p.g.) recordings to be shown here are those taken with the hand-made single lumen condenser microphone catheter.

In 1951 Gauer, Gienapp⁹⁾ introduced their intracardiac pressure gauge. When its phonocardiographic response was amplified and recorded as suggested by Maurice Rappaport the pressure waves presented only an overlapping first and second sounds as shown

Fig. 2. Gauer's transducer.

in the Fig. 2. Thus it is clear that it is unsuitable for the purpose of recording intracardiac heart sounds.

Immediately after our present method was published in 1954, two or three personal communications were received which questioned the possibility of blood flow murmurs being picked up by catheterization. Last year Eldridge and Hultgren¹⁰⁾, reported that due to catheterization, click-like sounds were artificially produced in the external phonocardiograms in 18 out of 57 cases and these extra sounds were found in the diastolic phase in 17 cases. Lewis also mentions a "knocking" sound which he interprets as being produced by the catheter tip striking the intracardiac wall. We too, have on rare occasions seen a regular occurrence of vibrations which we cannot help but recognize as related to the heart beat. The upper section of Fig. 3 is in the right atrium, and the center of the lower section is the chest sound. Above this a diastolic artificially produced noise, can be recognized at a glance. This sound does not travel to the chest wall. That complicated morphology of the heart cavity in addition to the cardiac movement would bring about the striking of the transducer in the tip of the catheter is to be expected and must be fully taken into account in the reading of the intracardiac phonocardiogram.

Jet Lesions

As one of the factors in the production

Fig. 3. See Tept.

of murmurs abnormal turbulence within the heart cavity can be considered. As a morphological proof the so-called jet lesions can be mentioned. This condition was formerly called Zahn's pocket, endocardial pocket, regurgitant lesion, etc.. Two years ago Edwards and Burchell¹¹⁾ gave a detailed report on numerous cases of jet impact. Papers concerned with this lesion point to certain regions as predominant sites as shown in Fig. 4. In our department 9 out of 15 cases were found to possess jet lesions. A typical case is a 37 year old patient who died of stomach cancer and whose heart had a very small circular ventricular septal defect of 3 mm diameter 4 cm below the aortic valve. Near the tricuspid valve on the opposite side on the right ventricle anterior wall a 7×6 mm oval shaped pocket-like sunken region was present. The surface was whitish, coarse and hard with a histological picture of a thickened endocardium with marked fibro-elastosis penetrating to the myocardium in part. Kitamura and Namba¹²⁾ of our department obtained intracardiac recordings of strong thrill and systolic murmur immediately after experimental V. S.D. was made in dogs after puncture of the septal muscular region with a trocar of 6 mm in diameter from the right ventricu-

SITES OF JET LESIONS

Fig. 4. Sites of jet lesions.

lar wall. The changes believed to be that of the jet lesions was evident as localized hyperemic changes even with only ten hours lapse of time after the operation. Histologically, the changes seen were limited mostly to that of the endocardium but some infiltration was also seen which suggested a transitional phase to the changes seen in jet lesions in man. It is evident therefore that the presence of turbulence in septal defects and valvular lesions can bring about these change. The next step was to make experimental aortic valve insufficiency and stenosis in dogs. After the production of aortic insufficiency a strong diastolic murmur became evident in the left ventricle. (Fig. 5) With aortic narrowing peripheral systolic

Fig. 5.

murmurs were heard. An ultrasonic detection method³⁾ was carried out with the cooperation of Dr. Wagai of the surgical department. (Fig. 6) A smooth basal line is seen when no stenotic murmurs are present but after experimental stenosis and together with the rise of murmurs the basal surface becomes irregular and a rapidly occurring and disappearing echo group ensues. (Fig. 7, 8) With the release of the stenosis these changes disappear. Whether this echo group arises from turbulence alone or is due to cavitation is not clear.

Fig. 6. Ultrasonic detection with aortic narrowing.

Fig. 7. Ultrasonogram during constriction.

Fig. 8. Ultrasonogram. release of the stenosis.

CASE REPORTS

Since my first report of intracardiac phonocardiography over 120 cases have been catheterized. However, since the use of a double lumen catheter is yet to become a practical and a reliable method, phonocardiographic catheter had to be inserted following routine catheterization with ordinary catheter. These separate insertions obviously presented difficulties in obtaining complete data in all cases. Only 46 cases which is tabulated in Table I will be presented at this time for discussion.

Before discussing these cases it must be mentioned that in man where there are no shunts, the atria, ventricles, pulmonary artery, and aorta each have characteristic internal phonographic recordings as reported in the first publication and these are as presented in the Fig. 9. There are no shunts in this case and from the top intracardiac phonocardiograms of the pulmonary artery, right ventricle, and the right atrium is seen. Characteristic points of pulmonary artery and the

TABLE I CLINICAL DIAGNOSIS BY STUDIED BY I.C.P.C.G.

Diagnosis	Number of Cases
<i>Congenital</i>	
VSD	7
ASD	7
PDA	3
Fallot	1
Pulm. St.	2
VSD+ASD	3
<i>Mitr. Disease</i>	7
<i>Aorti. Disease</i>	2
Basedow	4
Normal Findings	5
Others	5
<i>Total</i>	46

Fig. 9.

Fig. 10.

right ventricle is the slight systolic murmur and in the right atrium the second sound becomes rudimentary and instead the atrial sound marked. The next case is hyperthyroidism also without shunts presenting a large amount of systemic blood flow. The pulmonary artery, right ventricle and the right atrium is almost the same as the former case. Your attention is called however, on the Fig. 10 showing the process of the catheter being pulled out slowly from the right ventricle outflow to the inflow. In the outflow tract of the upper recording the second sound can be seen but in the lower inflow tract during normal systole an atrial sound is present. Even in the same ventricle the recordings become different according to the location of the catheter tip and with ventricular extrasystole of course, the atrial and the second sound cannot be found.

The left heart i.c.p.g. of man was first experienced by Luisada et al. We have had 2 cases and this is one of them. In the aorta a small first sound, moderate systolic murmur and a marked second sound was present. In the left ventricle a large vibration was seen at the beginning of the systole which was believed to be due to ejection. In the abdominal aorta the systolic murmur is somewhat delayed in reference to E.C.G. in the first beat and these vibrations become more and more rudimentary (Fig. 11, 12). The other case was that of combined aortic and mitral valvular disease. A remarkable early diastolic murmur from the left ventricle was seen in the oscilloscope.

Although intracardiac phonocardiogram holds many points of interest in the acquired heart disease group, it is in the diagnostic and pathophysiological study of congenital heart disease that this method is of the greatest value. This especially evident in the left to right shunt group in routine right catheterization. The following cases are those in which the discussion is based on the data obtained from right heart catheteri-

Fig. 11.

Fig. 12.

zation, which is tabulated in Table II.

Case 1

A 44 year old female patient with systolic murmur in the pulmonary artery was found to have no shunt with catheterization. The mean pulmonary artery pressure was 11 mm and the mean right ventricular pressure 19 mm mercury.

TABLE II RESULTS OF THE HEART CATHETERIZATION

No.	Disease	Name	Age Sex	Pressure (mmHg)					Oxygen (Vol %)					Shunt (l→r) (l/min)	Output (l/min)	
				r.a.	r.v.	l.v.	p.a.	art.	V.c.l.	V.c.s.	r.a.	r.v.	p.a.			art.
1	Pul. St.	S.O.	44 ♀	0	19		12		9	10	10	10	15		4.7	
2	V. S. D.	H.S.	25. ♀	4	13		15		12	14	13	14	17	2.9	6.1	
3	V. S. D.	T.T.	44. ♀	3	10		23		12	11	11	12	13	21	0.6	2.4
4	V. S. D.	A.S.	23. ♂	2	21		23			16	15	19	19	22	6.1	4.4
5	A. S. D.	K.H.	29. ♀	5	16		15			13	16	16	15	17	9.4	7.5
6	A. S. D. + ?	K.K.	31. ♀	4	34				12	11	15	13		17	1.5	5.3
7	Hypertension + A.S.D.	S.D.	46. ♂	9	19	26		160	10	9	16	16		18	12.	4.3
8	A.S.D.+V.S.D. + Pul. St.	T.A.	28. ♂	2	20		13		13	13	16	17	17	19	5.6	4.0
9	P.D.A. + A.S.D. ?	Y.M.	16. ♀	1	8		12		10	10	12	11	12	17	2.0	4.4
10	P.D.A. or Valsalva ?	K.H.	29. ♀	2	16		31		12	10	10	10 12	12	17	1.5	4.0

A diagnosis of simple pulmonary stenosis was made. The middle recording is the intracardiac recording within the pulmonary artery. (Fig. 13) It is a distinct diamond type ejection murmur with the first sound being rather rudimentary. The second sound in the pulmonary artery matches the pulmonic component of the second sound seen in the upper recording of the chest phonocardiogram. This murmur is not strongly evident within the right ventricle as seen in the lower recording. These findings of simple pulmonary stenosis were already reported by us 6 years ago.¹⁴⁾

Case 2

Next is a 25 year old female patient whose chest systolic murmur was in contrast to a strong shunt murmur within the right ventricle. (Fig. 14). This murmur was not present in the pulmonary artery or the right atrium. This strong shunt murmur of the right ventricle is recognized by every research group as being peculiar to V. S.D. This characteristic finding probably appears together with the forementioned jet stream especially when the defect is relatively small. Feruglio¹⁵⁾ states that the appearance of this

Fig. 13. Case 1.

Fig. 14. Case 2.

characteristic shunt murmur matches the surgical findings even when the shunt is so small as to defy detection of increase of oxygen content within the right ventricle by heart catheterization.

Case 3

A 44 year old female patient with arrhythmia absoluta and showing a protruding left pulmonary arch in a bilaterally enlarged heart is presented. A 1.2 vol % increase of oxygen content was present in the right ventricle. The upper recordings in the Fig. 16 are the chest phonocardiogram over the pulmonary artery and the lower intracardiac phonocardiogram. Such a marked third sound of the intracardiac phonocardiogram of the pulmonary artery is exceedingly rare and may be due to "knocking" of the catheter tip during the heart beat as mentioned earlier. However, since there is also a small vibration which matches this third sound in the chest heart sound it cannot be dismissed simply as an artifact. A click-like vibration is also seen in the mid-systolic phase and is believed to be an expanding sound of the dilated pulmonary artery. In the intra

cardiac sound of the right ventricle a shunt murmur as seen in the previous case is more evident at the time of the first and third cardiac cycles with the prolongation of the preceding diastole as mentioned above. Besides these findings you will note a mid-diastolic rumble. In the mingograph chest sound recordings this can be seen faintly as a so-called apical diastolic murmur.

Case 4

A 23 year old male presenting an increase of oxygen in the right ventricle of 4.1 Vol. % and showing a persistent left superior caval vein, is presented. The intracardiac sound gives a strongest shunt murmur in the right ventricle outflow and behind it is the mid-diastolic rumble as seen in the second recording. The position of the lowest recording is in the right ventricle inflow or the right atrium and shows a diastolic murmur including the atrial sound. In the upper-most recording within the pulmonary artery these diastolic murmurs are completely absent and shows a medium grade systolic murmur. As a case of V.S.D. it is probably due to a somewhat large shunt flow. The strong murmur seen at the right ventricle outflow tract is not clear at the inflow tract and the right atrium and this is interpreted as being due to the placement of the catheter tip on the jet stream at that time.

The chest heart sounds of A.S.D. have been given much attention since the reports of Barber, Magidson, et al.¹⁶⁾ The systolic murmur over the left second intercostal sternal line is due to a relative pulmonary stenosis brought about by increased pulmonary blood volume caused by this shunt. The strong splitting of the second sound is believed to be due to the delayed pulmonary valve closure accompanying prolonged ejection of the right ventricle. The mechanism is believed to be

Fig. 15. Case 3.

Fig. 16. Case 3.

similar to that of intracardiac pulmonary murmur in V.S.D. and pulmonary stenosis mentioned before. On the other hand, the mechanism of the faint apical diastolic murmur seen in 1/4 to 1/2 of A.S.D. cases is attributed by Feruglio¹⁷⁾ to relative tricuspid valve stenosis. We have also reported on this view in this assembly last year¹⁸⁾ and Lewis¹⁵⁾²⁰⁾ has reported cases where this murmur disappeared after successful surgery. In the 7 cases of A.S.D. discussed at this occasion all

presented mid-diastolic or pre-systolic murmurs at the tricuspid valve region whereas only a few were recognized with chest sound recordings.

Case 5

The first case of A.S.D. presented a shunt volume 1.3 times the systemic blood flow. When compared to the chest sound of the tricuspid region systolic murmur was absent in the right ventricle and the mid-diastolic murmur begins about 0.1 second later than the supposed position of the second sound. It is a diastolic murmur possessing a character of the so-called ventricle filling murmur. In simple A.S.D. we have not experienced extremely strong systolic murmur in the right ventricle. (Fig. 17, 18)

Case 6

The second case of A.S.D. is a 31 year old female whose catheterization was possible only to the right ventricle allowing only the confirmation of increase in the right ventricle pressure and A. S.D. A strongly predominant presystolic murmur in the right ventricle was seen. Although the presence of V.S.D. cannot be determined by the oxygen of the right ventricle alone the strong murmur suggests a small defect. Furthermore, in the right atrium the atrial sound and the first

Fig. 17. Case 4.

Fig. 18. Case 5.

Fig. 19. Case 6.

Fig. 20. Case 6.

Fig. 21. Case 7.

Fig. 22. Case 7.

sound is predominant and only a slight systolic murmur is seen. The chest recordings show no signs of this presystolic murmur of the right ventricle. If the supposition is extended further it may be said that this presystolic murmur arises locally due to a strong atrial contraction just when the tricuspid valve leaflets are at presystole, following upwards in a almost closing position due to a somewhat increased right ventricular pressure (Fig. 19, 20).

Case 7

The clinical picture of the next case 7 is a bilaterally enlarged heart with strongly impaired kidney function whose diastolic blood pressure is over 120 mm mercury. The systolic murmur is strongest at a point slightly inward from the apex in the chest wall. The increase of oxygen content of the right atrium to 6 vol% and the average pressure difference of the left and right atrium of

17 mm Hg was present. However, the shunt murmur was not seen in the right atrium but instead a slight systolic murmur and a diastolic murmur which increased somewhat at the presystolic stage was present in the right ventricle. With the shunt volume nearly 3 times that of systemic blood volume also seen in this case the cause of these murmurs is interpreted as being tricuspid in origin. (Fig. 21, 22)

Liu, Jacono²¹⁾ have reported systolic murmurs in the right atrium in 4 cases out of 6. In our experience only one out of 7 cases showed signs which we interpreted as being possibly a shunt

Fig. 23. Case 8.

Fig. 24. Case 8.

murmur. This finding was coincident to the fact that in A.S.D. jet lesions are rare as mentioned above. Even from the viewpoint of pressure gradient across the defect an existence of strong jet stream cannot be imagined. These observations lead the author to conclude that shunt murmurs are seldom seen in the right atrium in A.S.D.

Case 8

In the next case the diagnosis was made after the possible pathological conditions were listed which corresponded to the data obtained by catheterization. The systolic murmur of the pulmonary artery, strong shunt murmur of the right ventricular outflow and the diastolic murmur in the inflow tract fit into the picture very satisfactorily. (Fig. 23, 24)

Case 9

In the patent ductus arteriosus (P.D.A.) a 1.5 vol% rise of oxygen content in the right atrium and a 0.8 vol% oxygen gradient from the right ventricle to the pulmonary artery is present. This is a withdrawal from the distal end of the pulmo-

nary artery up to the inflow of the right ventricle. The shunt region is seen just where the upper tracing shifts to the middle row of the Fig. 25, and shows a continuous murmur when the catheter enters the right ventricular outflow, then the murmur disappears together with a series of extrasystoles. The return of the catheter to the inflow again causes the appearance of murmurs. When this finding is presented by increasing the gain it is seen in the second row of Fig. 26, as a continuous murmur. The diastolic murmur of

Fig. 25. Case 9.

Fig. 26. Case 9.

the right ventricular inflow seen in the lowest row is believed to be due to the filling murmur caused by A.S.D.

Case 10

The last case is also that of an external continuous murmur taken on the left sternal second intercostal space of a 25 year old female patient. Oxygen increase of 1.5 vol% was seen in the right ventricle and although a typical early diastolic decrescendo murmur was evident, insufficiency of

the pulmonary artery could not be confirmed by the pressure curve.

The tentative diagnosis of ruptured aneurysma of sinus Valsalva into right ventricle was also made. In the distal region of the pulmonary artery a short systolic murmur apart from the first and second sound and a long gradually decreasing murmur was present immediately after the second sound. The diastolic blood flow of the pulmonary artery also merits the consideration of P.D.A.. Why is this isolated appearance of the mid-systolic murmur present? Murmurs are accounted for by the inner diameter of the blood vessel and the blood flow volume but did the change in the inner diameter of the pulmonary artery both during systole and diastole bring about this condition? (Fig. 27, 28)

The cases discussed at this time were those which presented various problems and interpretations were attempted based mainly on intracardiac phonocardiograms.

SUMMARY

It can be said that P.D.A., V.S.D. and pulmonary artery stenosis show a definite pattern. A.S.D. also presents a general pattern. The apical diastolic murmur seen in A.S.D. can be attributed to the filling murmur near the tricuspid valves but the mechanism of the origin of mid-diastolic murmurs of V.S.D. has not as yet been discussed by anyone in length. By using the terms torrential mitral flow of the left ventricle origin McKusick has seemingly accepted the version of Carry-Coombs murmurs arising from the left ventricle as the cause of these mid-

Fig. 27. Case 10.

Fig. 28. Case 10.

diastolic murmurs.²²⁾ However, with intracardiac phonocardiograms the mid-diastolic murmur even in the case of V.S.D. are clearly recorded in the right ventricle. If it is interpreted that the sound originating in the left ventricle travels through the septal defect, McKusick's version may be correct. We have made experimental septal defects in dogs together with experimental aortic insufficiency to determine if it was possible to record from the right ventricle the murmurs arising within the left ventricle. As yet there has been no experimental findings which can support the presumption that murmurs of left ventricular origin travel to the right ventricle through the septal defect. To clarify these points an intracardiac phonocardiography of the left ventricle in V.S.D. would be helpful.

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