Second heart sound after pulmonary arterial banding operation

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After a pulmonary arterial banding procedure the phonocardiograms of 38 patients were correlated with haemodynamic and angiographic findings. Twenty-four patients had uncomplicated ventricular septal defect, 2 had single ventricle, 5 had transposition of the great arteries, 5 had atrioventricular canal defects, and 2 had coarctation of the aorta and ventricular septal defect. P_2 was separated from A_2 by less than 40 ms in 10 of the 11 patients with high pulmonary vascular resistance. Of 27 patients with nearly normal pulmonary vascular resistances and distal pulmonary artery pressures less than 50/20 mmHg (6.7/2.7 kPa), 24 had A_2 - P_2 intervals of over 40 ms. A narrow A_2 - P_2 interval with a satisfactory band was found in 2 patients with large right-to-left shunts. A_2 - P_2 interval did not change appreciably with age, and this measurement is a useful guide as to the effectiveness of pulmonary artery banding by one year after operation. If this interval is less than 40 ms, repeat catheterization should be carried out as such patients may have persisting pulmonary hypertension and an increased pulmonary vascular resistance.

Most patients have an easily heard splitting of the second heart sound (S_2) after a successful pulmonary banding. Some of the clinical reviews of the banding procedure fail to mention this change in the second heart sound (Dammann *et al.*, 1961; Stark *et al.*, 1969), while others (Takahashi *et al.*, 1968) have noted this splitting of the second sound as a characteristic clinical finding after banding. Aziz, Mesko, and Ellison (1972) stressed the potential value of the changes of the second heart sound after banding and presented data showing that with low pulmonary arterial pressures distal to the band, the split was over 40 ms while, if the pulmonary arterial pressure was raised, the splitting interval was less than 40 ms. This observation indicated that the phonocardiogram could serve as a non-invasive method for monitoring pulmonary arterial pressures after banding. However, only 17 subjects were studied by the above authors, all but 2 with adequate bands, and all subjects were over 2 years of age. Patients with pulmonary arterial banding have been followed with phonocardiograms for about 15 years at Winnipeg Children's Hospital. The phonocardiographic, haemodynamic, and angiographic findings of 38 banded patients are re-

viewed to assess the validity of using the phonocardiogram as a guide to the effectiveness of the banding procedure in reducing distal pulmonary arterial pressure.

Subjects and methods

Only patients in severe distress had pulmonary arterial banding. All had failed to respond to medical treatment. Several were desperately ill at the time of banding and the operation was performed in the presence of heart failure and lung infection. Only those patients who had post-banding cardiac catheterizations with measurement of distal pulmonary arterial pressure and with phonocardiograms obtained within a few months of catheterization are included in the present report. The 38 patients comprise about two-thirds of all the patients banded over a 15-year period. Twenty-four patients had uncomplicated ventricular septal defect, 2 had single ventricle, 5 patients had complete atrioventricular canal defects, 5 had transposition of the great arteries, and 2 had coarctation of the aorta as well as ventricular septal defect. The amount of constriction applied at operation was based on the cardiac response to banding

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and on the distal pulmonary arterial pressure (measured by pressure transducer in 33 patients and estimated by palpation in the others). An attempt was made to reduce the pulmonary arterial systolic pressure to about 40 mmHg (5.3 kPa). The mean band diameter was 7 mm, some infants developed striking cardiac slowing, with reduction of the PA pressures to 40 mmHg (5.3 kPa) and the surgeon accepted a lesser degree of constriction.

An index of pulmonary blood flow was obtained by using the pulmonary/systemic flow ratio. As an index of pulmonary vascular resistance, the pulmonary/systemic resistance ratio was calculated using mean pulmonary arterial and femoral arterial pressures. Serial film angiography was performed on all patients and measurements were obtained as indicated in Fig. 1. The minimal diameter of the pulmonary arterial segment underlying the band was measured; the distance from the pulmonary valve ring to the proximal part of the band, as well as the diameters of the pulmonary valve ring and of the aortic valve ring, were recorded. The average magnification factor in the angiographic system was 1.11, and was about the same for each subject, so that the measurements were not corrected for this distortion. Phonocardiograms were obtained using a Maico microphone at the left sternal edge in the second left intercostal space, using the Electronics for Medicine DR8 recorder in the 120-500 Hz range. The degree of splitting was obtained by

FIG. 1 Angiocardiogram. Arrow 1=plane of pulmonary valve; arrow 2=level of band. Distance between arrows is distance of band above pulmonary valve ring.

averaging the intervals between the aortic and pulmonary components of S_2 (A_2 - P_2 interval) for 5 consecutive heart beats. In addition, the height of P_2 divided by the height of A_2 was obtained to provide an index of the relative intensities of these sounds. Recorder speed was 75 or 100 mm/s. In children aged 3 years and over a carotid pulse curve was used to confirm the identification of A_2 (Fig. 2).

FIG. 2 Case 16—slight change in A_2 - P_2 interval with respiration. 100 mm/s paper speed, 20 ms time lines. Use of carotid pulse to identify A_2 . Sharpest and highest deflections arbitrarily used for timing A_2 and P_2 .

Results

Of the 38 patients, 15 were 2 years of age or younger, and the oldest patient was 9 years old. The mean age was 4 ± 3 years. Individual data are listed in Table 1.

The lumen of the pulmonary artery under the band was <10 mm in 29 of the 38 subjects, and <7 mm in 16 subjects. The systolic pulmonary arterial pressure was >50 mmHg (6.7 kPa) in 7 patients; 5 of these had banded segment diameters of 10 mm or more. The pulmonary/systemic resistance ratio was >0.30 in 11 patients and >0.60 in 5 of these. In 3 of these latter 5 patients, the banded segment was 10 to 14 mm in diameter.

Fig. 3A depicts the relations between raised pulmonary artery pressures or pulmonary vascular resistances (expressed as the resistance ratio), and A_2 - P_2 intervals, and Fig. 3B shows similar findings for the ratio of the intensity of P_2/A_2 . The values 0.30 for resistance ratio and 50/20 mmHg (6.7/2.7 kPa) pulmonary arterial pressure were chosen arbitrarily. Of the 11 patients with resistance ratios

of 0.30 or more, the A_2 - P_2 interval was <30 ms in 7, 36 to 38 ms in 3, and 45 ms in 1 patient. Of the 8 patients with pulmonary arterial pressures >50/20 mmHg (6.7/2.7 kPa), A_2 - P_2 was <40 ms in 7. Of the 14 patients with A_2 - P_2 intervals <40 ms, 11 had raised pulmonary arterial pressures and/or pulmonary vascular resistances (arbitrary values given above). Of the 24 patients with A_2 - P_2 intervals of 40 ms or greater, only 1 had a resistance ratio >0.30, and/or pulmonary arterial pressure >50/20 mmHg (6.7/2.7 kPa).

The pulmonary component of the second sound was of relatively greater intensity in patients with pulmonary hypertension, but the dividing line between those with raised pulmonary arterial pressures and resistances was less distinct for the ratio of P_2/A_2 intensity than it was for A_2 - P_2 interval. Of the 11 subjects with RP/RS ratios >0.30, 8 had P_2/A_2 intensity ratios of 1.0 or greater. Of the 27 patients with resistance ratios below 0.30, 22 of the subjects had P_2/A_2 intensity ratios <1.0. There were similar findings for those subjects with pul-

TABLE 1 Haemodynamic, angiographic, and phonocardiographic data

Case No.	P_2 - A_2 (ms)	Age (y)	Qp/Qs	Rp/Rs	Band (mm)	Band/aorta ratio	Band distance (mm)	Systolic gradient (mm)	PA pressure (mmHg) S	D	M	Desaturation (%)	P_2/A_2	RR (ms)	Lesion
1	5	4	0.78	1.11	11	0.85	15	10	85	45	75	9	5.10	605	VSD
2	29	2	0.83	0.95	6	0.40	15	64	46	24	36	24	1.02	546	VSD
3	20	5	0.74	1.21	10	0.56	22	15	105	45	70	11	6.62	773	VSD
4	40	2	1.00	0.27	7	0.47	50	72	22	14	16	8	0.58	420	VSD
5	56	1	0.69	0.19	5	0.31	8	80	12	7	9	6	0.25	448	VSD
6	48	2	0.97	0.21	6	0.60	15	70	22	10	15	8	0.25	395	VSD
7	66	4	1.53	0.12	8	0.38	17	73	22	10	16	0	0.31	468	VSD
8	72	5	1.47	0.25	10	0.42	30	62	34	11	20	4	0.40	669	VSD
9	65	4	0.65	0.25	9	0.50	12	80	20	8	13	1	0.10	560	VSD
10	50	5	1.40	0.23	10	0.48	12	77	38	12	27	10	3.00	628	VSD
11	20	4	0.70	0.15	6	0.35	17	92	15	9	11	10	1.58	642	VSD
12	24	6	1.25	0.64	14	0.74	18	17	80	35	60	17	2.40	746	VSD
13	21	1	1.33	0.13	6	0.27	18	56	20	8	13	20	1.13	400	VSD
14	38	8	1.81	0.32	15	0.79	13	12	83	28	47	1	2.50	560	VSD
15	80	5	1.14	0.15	7	0.50	23	60	24	10	14	5	0.57	540	VSD
16	71	4	1.22	0.16	4	0.19	9	74	23	8	14	1	0.28	600	VSD
17	41	1	1.30	0.25	7	0.47	11	54	36	12	24	6	0.40	388	VSD
18	46	1	0.71	0.27	4	0.33	11	80	15	6	12	21	0.22	448	VSD
19	48	1	0.72	0.18	4	0.41	15	74	14	7	9	8	0.65	494	VSD
20	49	2	2.20	0.10	7	0.42	10	64	26	11	18	20	0.95	410	VSD
21	61	2	0.89	0.17	4	0.32	9	85	15	7	10	7	0.50	495	VSD
22	53	4	0.92	0.16	8	0.43	15	63	17	7	11	6	0.24	694	VSD
23	27	4	0.48	0.35	8	0.40	16	86	21	12	17	15	1.00	486	VSD
24	33	4	1.39	0.17	6	0.25	21	63	27	9	18	1	1.20	438	VSD
25	54	3	2.31	0.19	9	0.45	18	33	47	17	32	2	1.08	773	AVC
26	80	5	1.00	0.14	12	0.36	30	83	20	8	14	16	0.35	545	AVC
27	55	5	1.70	0.13	8	0.34	20	82	28	10	15	10	0.32	739	AVC
28	36	2	0.54	0.32	4	0.27	2	70	20	7	13	11	0.14	360	AVC
29	21	3	1.64	0.33	8	0.43	16	35	55	22	35	8	3.11	396	AVC
30	55	8	1.90	0.17	8	0.44	4	50	40	15	25	10	0.14	620	SV
31	47	1	1.25	0.18	7	0.35	13	63	22	10	14	3	0.21	413	SV
32	25	8	0.83	0.40	5	0.31	17	45	37	11	22	25	0.35	607	TGA
33	36	9	1.56	0.38	11	0.46	15	57	58	26	48	22	0.54	672	TGA
34	35	7	0.57	0.29	6	0.32	12	85	17	10	12	50	0.05	520	TGA
35	45	2	0.61	0.75	6	0.32	7	51	54	30	38	42	1.12	480	TGA
36	60	1	0.90	0.28	6	0.39	16	120	15	8	12	21	0.31	533	TGA
37	43	2	0.79	0.26	4	0.21	17	104	31	11	20	1	0.96	507	COA
38	42	5	1.00	0.18	10	0.67	30	48	18	8	13	0	0.65	613	COA
Means	45	4	1.12	0.32	7	0.42	15	63	33	14	23	11	1.07	542	
SD	±17	±3	±0.46	±0.27	±2	±0.15	±6	±24	±22	±10	±16	±11	±1.40	±116	

Age—at time of heart catheterization and phonocardiogram; Qp/Qs—pulmonary/systemic flow ratio; Rp/Rs—pulmonary/systemic resistance ratio; band/aorta—ratio of band diameter to aortic valve diameter; P_2/A_2 —ratio of peak phonocardiographic deflections of P_2 to A_2 ; PA pressure: S=systolic, D=diastolic, M=mean; desaturation—pulmonary vein oxygen saturation (or 96%) minus arterial; VSD—ventricular septal defect; AVC—atrioventricularis communis; SV—single ventricle; COA—coarctation of aorta (plus VSD); TGA—transposition of great arteries. Cases 32 and 33 also had SV.

Conversion from Traditional to SI units: 1 mmHg≈0.133 kPa.

FIG. 3 (A) A_2-P_2 interval in patients divided into those with high or low pulmonary vascular resistance and those with high or low pulmonary artery pressures. (B) Ratio of intensity of P_2/A_2 in patients divided into those with high or low pulmonary vascular resistance and those with high or low pulmonary artery pressures.

monary hypertension defined as pulmonary arterial pressure $>50/20$ mmHg (6.7/2.7 kPa).

Four patients had both short A_2-P_2 intervals and P_2/A_2 intensity ratios >1.0 , yet had low pulmonary arterial pressures and resistances. Two of these patients had right-to-left shunts of 16 and 29 per cent of systemic flow.

The ratio of pulmonary to systemic blood flow was >1.5 in 8 patients. This was not associated with a greater degree of splitting of the second heart sound compared with those with little or no left-to-right shunt. The pulmonary arterial pressure was $>40/20$ mmHg (5.3/2.7 kPa) in 5 of these 8 patients and less than this value in the other 3.

The band was >15 mm above the valve in 17 patients, and 11 of these were over 3 years of age. The band was <10 mm above the valve in 6 patients and 4 were less than 3 years of age.

Arterial oxygen desaturation was calculated by subtracting the systemic arterial oxygen saturation from the pulmonary venous oxygen saturation. In 14 of the subjects, pulmonary venous saturations were obtained (mean value 96%); in the remaining 24 subjects it was assumed that pulmonary venous saturation was 96 per cent. Indicator dilution curves revealed a trace or greater right-to-left shunt in the majority of patients. The desaturation of arterial blood was greater than 8 per cent saturation in 10 of the 24 subjects with uncomplicated ventricular septal defects. In 4 of these 10 subjects the right-to-left shunt was associated with pulmonary hypertension and increased pulmonary vascular resistance, while in the remaining 6 subjects the right-to-

left shunt was apparently caused by the narrowness of the band.

Representative phonocardiograms are shown in Fig. 4 to 7. Fig. 4 illustrates the variability in A_2-P_2 intervals and P_2/A_2 intensity ratios in 3 patients with adequate bands. Case 35 with transposition of the great arteries had an A_2-P_2 interval exceeding 40 ms, but had raised pulmonary arterial pressures. Fig. 5 shows the very loud P_2 in 2 patients with severe residual pulmonary hypertension. Of 15 patients with phonocardiograms at age 2 to 3 years, and subsequently at least 2 years later at age 4 to 6 years, the mean A_2-P_2 interval changed from 40 to 45 ms ($P>0.10$) while RR interval changed from 565 to 570 ms ($P<0.05$). There seemed to be little change with age, for the A_2-P_2 interval increased by over 10 ms in only 3 of the 14 subjects, and the mean values at the two ages were not significantly different. In Fig. 5, Case 25 showed a wider A_2-P_2 interval with age, and Case 10 no change.

Some respiratory variation may occur in the A_2-P_2 interval after PA banding. The largest variation occurred in Case 38, in whom spontaneous closure of a large ventricular septal defect had occurred during the first year. This girl had an associated coarctation of the aorta, which was not repaired at the time of banding but was electively repaired at age 7 along with removal of the band. At cardiac catheterization at age 5, the right ventricular pressure was 66 mmHg (8.8 kPa), systolic pulmonary arterial pressure 18 mmHg (2.4 kPa), and left ventricular pressure 134 mmHg (17.8 kPa). The most notable feature of S_2 was the pronounced

FIG. 4 Phonocardiograms from 4 banded patients. Case 9 at age 4, Case 13 at age 1, Case 6 at age 2 years, all with large ventricular septal defects and normal PA pressures. Variable degrees of splitting of S_2 , variable ratio of intensities of P_2/A_2 . Case 35 has transposition of the great arteries and systolic PA pressure was 54 mmHg (7.2 kPa). Time lines 0.2 s in these and subsequent phonocardiograms.

respiratory fluctuation in the A_2-P_2 interval (Fig. 6). In most of the remaining subjects the A_2-P_2 intervals were either constant or changed by < 10 ms with different phases of respiration. No attempt was made to control respiration during the phonocardiograms on these patients, the majority of whom were under 5 years of age.

Linear regression analyses were carried out for the A_2-P_2 interval and the P_2/A_2 intensity ratios (Table 2). The highest correlation was for pulmonary arterial diastolic pressure and sound intensity ratio, and other significant correlations were found between resistance ratio, the ratio of band diameter to aortic diameter, and systolic pressure gradient across the band.

Discussion

As experience is gained in operating on small infants with large ventricular septal defects, considerably fewer pulmonary arterial banding procedures will be performed. None the less, in some

complex cardiac anomalies, such as single ventricle, transposition of the great arteries, and atrioventricular canal, the banding procedure is likely to remain a useful palliative procedure. It is in some of these patients that it may be difficult to enter the pulmonary artery at catheterization and an indirect means of assessing the pulmonary haemodynamics after banding would be most useful. In the series reported here, 4 patients had an inadequate band. Two of these subjects subsequently had pulmonary systemic resistance ratios of greater than 1.0, and are considered inoperable. In one subject the pulmonary/systemic resistance ratio was 0.64 and successful closure of the ventricular septal defect was carried out, but four years after operation the patient was in difficulty because of progressive pulmonary vascular disease. While 2 of these 4 patients moved away and were not seen again for 2 to 3 years after the banding procedure, and the parents of a third patient refused further operation, the phonocardiogram correctly identified these subjects as having persisting pulmonary hypertension and might have allowed earlier reinvestigation and effective surgery.

While the correlation between the level of pulmonary arterial pressure or pulmonary vascular resistance and the A_2-P_2 interval was not high, measurement of this interval was of practical importance. All but one of the subjects with a significant increase in pulmonary vascular resistance (pulmonary/systemic resistance ratio > 0.30) had an A_2-P_2 interval of less than 40 ms, allowing a recognition of 90 per cent of subjects with an increase in resistance. Of the 27 subjects with normal resistances, 3 had A_2-P_2 intervals of < 40 ms, a frequency of 11 per cent false positive identification of patients likely to have an increased pulmonary vascular resistance by using the 40 ms cutoff value.

The ratio of the intensity of P_2/A_2 was a less reliable index of rise in pulmonary arterial pressures or vascular resistance than was the A_2-P_2 interval. Of subjects with an increased pulmonary vascular resistance, 73 per cent had P_2/A_2 sound intensity ratios < 1.0, resulting in a false negative index in 27 per cent of the subjects. Eighteen per cent of the subjects gave a false positive result having P_2/A_2 ratios of < 1.0, in the absence of any increase in pulmonary arterial pressure or pulmonary vascular resistance.

One explanation of why the patient with a low pulmonary arterial pressure distal to an adequate band has wide splitting of the second heart sound is that the pulmonary valve does not close until the proximal pulmonary artery pressure falls from its high systolic level to a low diastolic value.

FIG. 5 Two patients with unsatisfactory results with severe persisting pulmonary hypertension. Case 1—very loud single S_2 ; Case 12—systolic ejection click (C), P_2 is much louder than A_2 and A_2-P_2 is only 24 ms.

Shaver *et al.* (1974) recently showed that the P_2 sound is delayed 33 to 89 ms beyond that point at which pulmonary arterial pressure falls below right ventricular pressure. Recognition of this time lag (called hangout) was possible with the development

FIG. 6 Phonocardiograms 3 years apart in 2 patients. Case 25— A_2-P_2 increased from 26 to 52 ms. Case 10— A_2-P_2 changed very little from 52 to 50 ms.

of practical catheter tip manometers. It was reported that the wide splitting of the second sound in patients with idiopathic dilatation of the pulmonary artery, mild pulmonary stenosis, and atrial septal defect, was not the result of prolonged right ventricular systole, but of an increase in this 'hangout' interval. Values for this 'hangout' interval will be of use in understanding the wide A_2-P_2 interval in patients with pulmonary arterial banding, but are not available. A reduced pulmonary arterial capacitance present in patients with pulmonary arterial banding might shorten the A_2-P_2 interval by reducing the P_2 'hangout' while a prolonged right ventricular systole and a low pulmonary arterial diastolic pressure would tend to delay pulmonary valve closure. Other factors likely to modify the A_2-P_2 interval include heart rate, distance of the band above the pulmonary valve ring, age, pulmonary blood flow and its ratio to systemic flow, magnitude of right-to-left or left-to-right shunts, closure of the ventricular defect, and respiration. The same subject was not compared at different heart rates, but no correlation was found between the A_2-P_2 interval and heart rates of different subjects. Aziz *et al.* (1972) reported an inverse linear correlation between the distance the band was above the pulmonary valve ring and the A_2-P_2 interval, but this correlation was not confirmed in the present study. Age seemed to influence this distance in our patients. Age would also influence the relative degree of constriction produced by the

FIG. 7 Phonocardiograms—Case 38 at age 2 and 6 years. There is a striking respiratory variation in the A_2 - P_2 interval. The patient had coarctation of aorta and the ventricular defect closed spontaneously.

band, for presumably the band remains fixed while other structures increase in size.

In addition, age might influence pulmonary vascular resistance, and, if previously high, some

TABLE 2 Correlation coefficients A_2 - P_2 and P_2/A_2

	A_2 - P_2 All patients (38)	A_2 - P_2 VSD only (26)	P_2/A_2 All patients (38)
Qp/Qs	0.18	0.19	—
Rp/Rs	-0.58*	-0.64*	0.73*
Band width	-0.10	-0.30	0.45*
Band/aortic diameters	-0.31	-0.44*	0.54*
Band distance	0.14	-0.01	0.16
Systolic gradient	0.49*	0.54*	-0.63*
PA diastolic pressure	-0.58*	-0.66*	0.81*
Arterial oxygen desaturation	-0.29	-0.53*	-0.06
RR interval	-0.02	-0.17	0.33*

* $P < 0.05$.

Conversion from Traditional Units to SI Units: 1 mmHg $\approx$ 0.133 kPa.

recession might be expected on the basis of anatomical studies (Dammann *et al.*, 1961). There was no strong correlation between the pulmonary/systemic flow ratio and the A_2 - P_2 interval in our clinical material except that a short A_2 - P_2 interval was found in 2 subjects with normal pulmonary arterial pressures and large right-to-left shunts. The reduced pulmonary flow in these patients might lead to a shortening of right ventricular systole.

Most of the patients studied by Aziz *et al.* (1972) were over 4 years of age. The important time to know whether a band has achieved the desired result of lowering pulmonary arterial pressures is within 6 to 12 months of creating the band and before there has been any progression of pulmonary vascular disease. The data on the patients presented here suggest that phonocardiography with the measurement of A_2 - P_2 interval is an excellent guideline as to the effectiveness of the band, even at age 1 to 2 years. Patients who have A_2 - P_2 intervals less than 40 ms one year after banding should have repeat catheterization studies in order to reassess their haemodynamic situation.

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References

- Aziz, K-U-D., Mesko, Z. G., and Ellison, R. C. (1972). Assessment of adequacy of pulmonary arterial banding by phonocardiogram. *British Heart Journal*, **34**, 1029.
- Dammann, J. F., Jr., McEachen, J. A., Thompson, W. M., Jr., Smith, R., and Muller, W. H. (1961). The regression of pulmonary vascular disease after the creation of pulmonary stenosis. *Journal of Thoracic and Cardiovascular Surgery*, **42**, 722.
- Shaver, J. A., Nadolny, R., O'Toole, J. D., Thompson, M. E., Reddy, P. S., Leon, D. F., and Curtiss, E. I. (1974). Sound pressure correlates of the second heart sound. *Circulation*, **49**, 316.
- Stark, J., Aberdeen, E., Waterston, D. J., Bonham-Carter, R. E., and Tynan, M. (1969). Pulmonary artery constriction (banding): a report of 146 cases. *Surgery*, **65**, 808.
- Takahashi, M., Lurie, P. R., Petry, E. L., and King, H. (1968). Clinical and hemodynamic effects of pulmonary artery banding. *American Journal of Cardiology*, **21**, 174.

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