Changes in the Electrocardiogram and the Auscultatory Findings Following Surgical Repair of Ventricular Septal Defect

By

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Although the possibility of extracorporal circulation has given a considerable impetus to the surgical repair of ventricular septal defects, few reports deal with the postoperative ECG changes and the auscultatory findings. This apparent lack of interest is evidently due to the fact that the postoperative disappearance of the characteristic holosystolic murmur was regarded as indicating the success of the intervention, so that any further examination of acoustic phenomena was deemed superfluous. As regards ECG-tracings, it was held until quite recently that the curve of patients with ventricular septal defect was like that of normal persons, so that authors contented themselves with studying only the actual lesions due to ventriculotomy [7]. Recent investigations [3] have shown that not more than 15 to 20 per cent of patients with ventricular septal defect have a normal ECG, while the rest shows diagnostically and prognostically significant alterations that point to a left, right or combined ventricular overload. The present study was designed to ascertain how — irrespective of the original haemodynamic conditions — the electrocardiogram is influenced by the surgical repair of ventricular septal defect.

Material and Method

The material consisted of 25 cases of isolated ventricular septal defect — 12 boys and 13 girls between 5 and 15 years — who were examined before as well as 8 to 12 days and again 1 to 3 years after the operation. The data presented in the following are characteristic only of cases calling for reparative surgery and not of all patients suffering from the anomaly under consideration. Preoperative catheterization of the right heart was performed in all cases; in order to avoid the determination of oxygen uptake, a procedure fairly difficult and a possible source of error especially in the case of children, the value of relative shunt was estimated according to Mürtz [16]. The ECG was recorded from 12 leads, and the phonocardiogram in 3 frequency bands (with an ECG curve as reference tracing). Ventricular overload was assessed according to the criteria of Keith *et al.* [11]. The principal signs to show the success of the operation were a considerable increase in body weight (4 to 20 kg, with an average of 12 kg) and growth (13 to 30 cm, with an average of 26 cm). Except one child who exhibited a mild congestive failure a year after the operation, all the patients recovered and their circulation became sufficient.

Results

Electrocardiography

It is evident from Table 1 that symptoms of ventricular overload were observed in all patients prior to the intervention, and only in 50 per cent thereafter. Patients with no or only slight elevation of the pulmonary pressure (Group I) were separated from those suffering from considerable pulmonary hypertension (Group II). Members of the latter group had systolic pressures exceeding 50 mmHg which, however, still remained below the value of the systemic pressure, since otherwise (i. e. with veno-arterial shunt) they would not have been operated upon. The highest pressure in this group was 100/0 mmHg in the right ventricle, 100/40 mmHg in the pulmonary artery against a systemic pressure of 110/70 mmHg. Preoperative right ventricular overload was not observed in any member of Group I; left ventricular overload subsided in 50 per cent, and biventricular overload in more than two thirds of the cases. In Group II, the right ventricular overload ceased in 2 cases, while that of the left ventricle increased likewise in 2 cases after the operation. There were 7 cases of pulmonary hypertension in this group, and the postoperative ECG was normal in a single case only (statistically insignificant difference, $p > 0.9$), whereas, in Group I, the electrocardiographic signs of overload disappeared in 11 out of 18 cases ($p < 0.01$).

Table 1

Electrocardiographic signs of ventricular hypertrophy before, and 1 to 3 years after repair of ventricular septal defect

Ventricular load	Systolic pressure in right ventricle			
	< 50 mm Hg		> 50 mm Hg	
	before operation	after operation	before operation	after operation
Left ventricle	8	4	1	3
Both ventricles	10	3	3	2
Right ventricle	0	0	3	1
No load	0	11	0	1

It can be seen from Table 2 that incomplete or complete right bundle branch block was present in 20 per cent of the cases prior to operation, and was observed in all patients but one immediately thereafter. The number of incomplete bundle branch blocks which disappeared in the course of years was equal to those which had developed into complete block. While the latter condition occurred in a single patient before surgery, it appeared in nearly 70 per cent of them after the operation. The development of block

seemed to be independent of both the site and the size of the defect.

As can be seen from Fig. 1, the depth of the Q-wave obtained in left precordial leads was in close connection with, and its amplitude was proportional to, the shunt volume. Accordingly, the amplitude of the Q-waves decreased significantly after the operation ($p < 0.001$; Fig. 2).

As regards repolarization, the T-waves in lead V_1 exhibited considerable postoperative modifications (Table 3). They were negative in one third of the cases before, and in more than two thirds immediately after, the operation. Of 25 patients, 21 had negative and 4 diphasic T-waves 1 to 3 years after the intervention, so that none of the patients displayed a positive T-wave at that time, although their number was 7 prior to surgery. The difference was significant statistically ($p < 0.05$) in respect of the period

TABLE 2

Right ventricular conduction disturbance before, 1—8 days and then 1—3 years after repair of ventricular septal defect

	Right bundle branch block		
	none	incomplete	complete
Before surgery	20	4	1
1—8 days after surgery	1	10	14
1—3 years after surgery	5	3	17

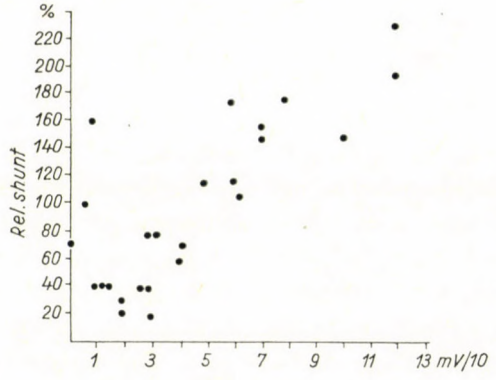

FIG. 1. The depth of the Q-wave in lead V_6 is proportional to the size of the arterio-venous shunt in patients with ventricular septal defect

immediately following the intervention, and insignificant ($p > 0.1$) in that of subsequent periods.

Phonocardiography

With the exception of one patient whose preoperative high pulmonary pressure was accompanied by an ejection murmur only, a characteristic harsh pansystolic stenotic murmur of increasing and then decreasing intensity was registered in all the patients before the operation (Table 4). Phonocardiograms taken immediately after the intervention are less reliable on account of the thick bandage around the chest. It was only in a single case that a holosystolic stenotic murmur was observed 1 to 3 years after the intervention. In 50 per cent of the cases a short protosystolic ejection murmur, and in a third no murmur whatever, was registered. In 4 cases the murmur remained pansystolic but had become more subdued, and its pattern was no longer diamond-shaped: it had developed into a holosystolic murmur of unchanging or decreasing intensity.

Preoperative examinations revealed a widely split second heart sound in

TABLE 3

T-wave in lead V_1 before, 1—8 days, and 1—3 years after repair of ventricular septal defect

	T-wave in lead V_1		
	negative	diphasic	positive
Before surgery	8	10	7
1—8 days after surgery	18	4	3
1—3 years after surgery	21	4	0

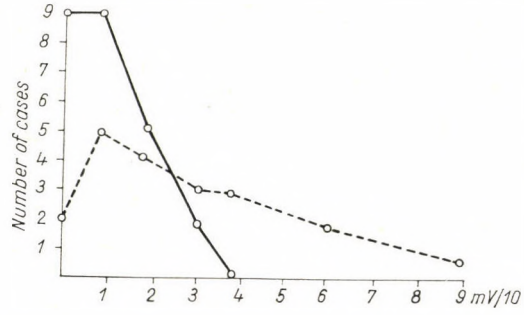

FIG. 2. The amplitude of the Q-wave in lead V_6 diminishes after repair of the ventricular septal defect. Solid line = before operation. Dotted line = 1 to 3 years after operation

about 70 per cent of the cases; the interval between the two components reached or exceeded 0.04 sec, a phenomenon that appeared in more than 90 per cent of the patients after surgery (Table 5).

strable owing to the gradual reduction of the right ventricular muscle mass. The fact that the percentage of electrocardiographic disorders did not significantly decrease after the operation of patients with high preoperative

TABLE 4

Character of heart murmur before, and 1—3 years after repair of ventricular septal defect

	Number of cases	
	before	1 to 3 years after
	operation	
Holosystolic stenosal	24	1
Holosystolic non-stenosal	—	4
Ejection	1	12
No murmur	—	8

DISCUSSION

Ventricular hypertrophy was observed in every one of our patients, whereas — according to literature — the ratio of patients with a normal ECG amounts to 15 to 20 per cent [3]. This discrepancy is presumably due to that we only examined surgically treated patients and there is no indication for surgery in mild cases in which no ventricular hypertrophy develops. All signs of left or bilateral ventricular hypertrophy disappeared in most members of Group I after the operation. As regards Group II, only the right ventricular hypertrophy subsided after the intervention, whereas the hypertrophy of the left ventricle became — according to the ECG tracings — more frequent, or rather more easily demonpulmonary pressure is an additional fact indicating that the intervention should be performed before pressure in the pulmonary artery had become too high. A comparison of our results with those obtained by VINCE and KEITH [18] shows that the changes observed in our material were not spontaneous phenomena but induced by the surgical repair of the ventricular septal defect.

The appearance of incomplete or complete right bundle branch block in the period immediately following the operation is — as stated also by FISHER *et al.* [7] and MARANHAO *et al.* [14] — obviously due to an operative lesion of the subendocardial conduction system. There were a few cases in which the right bundle branch block was transitory, a phenomenon pre-

sumably caused by some small haemorrhage or oedema [17]. Our findings offer no support to the view of BRISTOW *et al.* [4] that there exists a close correlation between the location of the defect and the frequency of block. While LAUER *et al.* [13] observed the development of complete atrio-ventricular block in about 20 per cent of their surgically treated patients, there was no such case in our material, since this complication does not occur with up-to-date surgical techniques [17].

The frequency of a deep Q-wave in left precordial leads, especially in V_6, has been noted by MARSICO *et al.* [15], BURCH and DEPASQUALE [5], WOOD *et al.* [19], VINCE and KEITH [18], as also by BLONDEAU [3], a phenomenon attributed to a hypertrophy of the ventricular septum by MARSICO *et al.*, and to a diastolic overload of the left ventricle by CABRERA [6]. Our observation that the depth of the wave is proportional to the volume of the left-to-right shunt, and that it is reduced after the operation, supports the latter assumption. Like BLONDEAU *et al.*, in V_1 the T-wave was initially negative in a third of our cases. The postoperative inversion of originally positive or diphasic T-waves was evidently due to an injury of the septum at operation [7]. If the inversion of an originally positive T-wave occurs several years after the operation, it is caused by a gradual regression of right ventricular hypertrophy.

TABLE 5

Interval between the two components of the second heart sound before, and 1—3 years after repair of ventricular septal defect

	Number of cases	
	before operation	1—3 years after operation
Single second sound	3	1
— 0·03″	4	1
0.04″—0.06″	18	23

A postoperative holosystolic stenotic murmur was observed in a single case only; development of a residual shunt, unavoidable in about 10 per cent of the cases [12] was proved in our case by the dye-dilution curve. The origin of the ejection murmurs observed several years after the intervention is somewhat obscure. Their haemodynamical prerequisite (relative stenosis induced by a muscular hypertrophy of the right outflow tract) had probably been present before the operation but was suppressed by the harsh holosystolic murmur; it might

be maintained by hypercirculation, a frequent phenomenon in children and adolescents. It should be noted that BLEIFER *et al.* [2] further Van HAUWAERT and NADAS [8] observed similar murmurs in patients with a small ventricular septal defect; it may have been elicited postoperatively by a haemodynamically insignificant residual shunt. The mechanism of postoperative holosystolic but not stenotic murmurs is unelucidated. This type of murmur was recorded in 4 patients; in one case it showed the highest intensity parasternally in the 4th left intercostal space. It may have been caused by a moderate tricuspid insufficiency induced by a transitory detachment of the median leaflet of the tricuspid valve in the course of the operation. In the other 3 cases, the murmur in question was most intensive apically; yet, signs of left heart failure indicative of functional mitral regurgitation were absent. It is probable that the murmur under review, the like of which was observed by HOLLMAN *et al.* [9] in numerous cases where the ventricular septal defect was not accompanied by pulmonary hypertension, was due to an incomplete closure of the defect or defects. Such incomplete closure was verified in a single instance out of a total of 29 cases treated by BECK *et al.* [1]. That the second heart sound was widely split in two thirds of our cases must have been due to a diastolic overload of the pulmonary circulation [10]. Not only did the intervention fail to arrest this phenomenon, but — with the exception of two cases — it appeared in all patients several years after the operation. The cause of this unexpected sequel was evidently the fact that, while the cessation of the arterio-venous shunt reduced the pulmonary flow, the surgical injury gave rise to a right bundle branch block manifesting itself with a delayed closure of the pulmonary valve.

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SUMMARY

In 25 patients with successfully repaired ventricular septal defect the electrocardiographic and phonocardiographic changes have been observed immediately and then 1 to 3 years after the operation.

Ventricular hypertrophy ceased in the major part of patients whose preoperative pulmonary pressure had been less than 50 mm Hg. The ECG of patients with pulmonary hypertension did not return to normal. The amplitude of the deep Q-wave in lead V_6, which points to a diastolic overload and is proportional to the size of the arterio-venous shunt, significantly decreased after the operation. The surgical injury gave rise to incomplete or complete right bundle branch block in nearly every case, and a complete block was present in the overwhelming majority of the cases 1 to 3 years after the operation. The T-wave in lead V_1 usually became negative after surgery.

The harsh holosystolic stenotic murmur disappeared after correction of the defect; the auscultatory findings had become normal in the majority of the patients, while only a short ejection murmur was heard in some cases. The latter phenomenon, as also the sporadically observed non-stenosal holosystolic murmur, may have been due to the existence of a haemodynamically insignificant residual shunt.

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