Haemodynamic changes during paroxysmal tachycardia in a patient with Wolff-Parkinson-White syndrome

P. Jebavý, J. Hurych, K. Bergmann, and J. Wolf¹

From the Institute for Cardiovascular Research, Budějovická 800, Praha 4-Krč, Czechoslovakia

The paper describes the haemodynamic changes in one patient with Wolff-Parkinson-White syndrome during a sudden attack of supraventricular tachycardia. During tachycardia, there was an unusual increase in cardiac output and a manifestation of a left-to-right shunt. This shunt was not detectable at rest at normal heart rate.

The Wolff-Parkinson-White syndrome is usually considered a benign curiosity of electrical myocardial activity, and most papers deal with this syndrome from this point of view. Okel (1968) reviewed several cases of sudden death in these patients, and up to the present only a few patients have been subjected to haemodynamic examination during a sudden attack of tachycardia (Ferrer *et al.*, 1949; Saunders and Ord, 1962; Benchimol *et al.*, 1965b). This paper describes the haemodynamic changes and a manifestation of a left-to-right shunt in one patient during a paroxysm of tachycardia.

Case report

The patient was a 16-year-old ice-hockey player. He suffered from frequent bouts of tonsillitis, but there was no history of rheumatic fever or chorea. At the age of 7 a systolic murmur was found, but the patient gave no history of breathlessness even during strenuous exercise. Three years previously he had several paroxysms of very fast heart rate, these attacks being provoked either by exercise or by a blow over his chest.

Physical examination showed an asthenic type of man. No praecordial pulsation or heavy apex beat were palpable. A loud systolic murmur was heard over the upper part of the sternum and over his left upper sternal margin. There was no propagation of the murmur. The rest of the physical findings were entirely normal.

Chest x-ray, spirometry, blood count, and all biochemical tests were normal. Electrocardiographic examination showed sinus rhythm, PQ interval

0.1-0.12 sec., QRS complex 0.1 sec., typical 'delta wave', and an incomplete right bundle-branch block.

The phonocardiogram showed a systolic murmur of a slight spindle-like character ending closely before the second heart sound. The murmur reached maximal amplitude over the pulmonary area where it was indiscernible from the first heart sound; the second sound was slightly split.

On the basis of clinical examination, a preliminary diagnosis of atrial septal defect and Wolff-Parkinson-White syndrome was made.

The haemodynamic examination at rest revealed normal pulmonary arterial pressure; there was, however, a small (8.2 mm. Hg) systolic pressure gradient across the pulmonary valve. The blood samples taken from the pulmonary artery, right heart, and the superior and inferior venae cavae showed no difference in oxygen content, and the dye dilution curve (pulmonary artery injection-aortic arch sampling) showed no early recirculation. Cardiac output was 9.0 l. calculated both according to Hetzel *et al.* (1958) and according to Stewart-Hamilton.

During catheterization, when the resting measurements were taken, a paroxysm of supraventricular tachycardia occurred. In the 70th minute of tachycardia the catheter was moved from the pulmonary artery and placed in the superior vena cava, and the paroxysm was abolished by a 4.5 kV DC discharge.

The haemodynamic changes during the paroxysm of tachycardia are summarized in the Table. During the initial phase of tachycardia (heart rate 192/min.), the cardiac output increased from the control value of 9.0 l./min. to 18.9 l./min. and the pulmonary arterial mean pressure increased from 7.3 to 14.7 mm. Hg. The right ventricular end-diastolic pressure remained at zero level and the systolic pressure gradient across the pulmonary valve disappeared. The dye dilution curve showed a slight early recirculation (Fig. 1).

¹ Medical Department of Physical Training, Prague Municipal Health Institute Spálená 12, Praha 2, Czechoslovakia.

TABLE Haemodynamic data

		Pressures (mm. Hg)													
Time	Heart rate /min.	Aorta			Pulmonary artery			Right ventricle		Right atrium	Pulm. blood flow (l./min.)	Stroke vol. (ml.)		Qs — % Qp	
		Syst.	Diast.	Mean	Syst.	Diast.	Mean	Syst.	Diast.			RV	LV		
	84	109	75	88	10.3	3.0	7.3	18.5	0	—	9.0	120	120	0	
Tachycardia 0'															
5'	192	106	91	94	18.4	8.1	14.7	18.8	0	—	18.9	98	75	24.2	
20'	228	—	—	—	23.6	9.5	17.7	23.7	0	—	17.1	75	40	46.3	
25'	216	—	—	—	—	—	—	—	—	—	21.3	98	27	72.5	
35'	216	—	—	—	25.0	10.6	—	25.6	0	—2.0	—	—	—	—	
Cardioversion 70'															
	78	—	—	—	—	—	—	—	—	—	6.8	88	88	0	

During the 20th minute of tachycardia the heart rate reached 228 beats a minute, the pulmonary arterial mean pressure reached 23.6 mm. Hg, and the dye dilution curve (pulmonary artery injection-aortic arch sampling) showed a definite pattern of early recirculation. The pulmonary blood flow (calculated according to Hetzel *et al.*, 1958) was 17.1 l./min. and the ratio of shunted to pulmonary blood flow was 46.3 per cent. This ratio reached 72 per cent during the 25th minute of tachycardia.

After cardioversion the heart rate was 78 a minute and the cardiac output was 6.8 l./min. The dye dilution curve (superior vena cava injection - aortic arch sampling) no longer showed early recirculation. To prevent another attack of tachycardia, repeat catheterization of the pulmonary artery was abandoned.

Discussion

There are two main questions that should be discussed in this case: (1) the considerable increase in pulmonary and systemic blood flow during the initial phase of the tachycardia; and (2) the manifestation of the left-to-right shunt during the later phase of the paroxysm.

Most papers describing the reaction of cardiac output during paroxysms of tachycardia or during artificial pacing of the human heart show that the cardiac output is either reduced (Benchimol *et al.*, 1965b) or remains unchanged (Saunders and Ord, 1962; Ferrer *et al.*, 1949). An increase in cardiac output during tachycardia has been described only by Benchimol, Ellis, and Dimond (1965a).

The decrease or the lack of substantial increase in cardiac output during tachycardia, due either to a spontaneous attack of tachycardia or to artificial pacing is usually explained by the shortening of the filling period or by the resistance to ventricular filling.

This resistance might be different from that in tachycardia due to exercise. The explanation of the unusual increase in cardiac output during supraventricular tachycardia in this case might be the fact that the patient was a trained young subject with a good cardiac contractility and a good ventricular compliance. Another explanation might be the fact that the patient had a very low resistance in both pulmonary and systemic circulation. This could contribute to a better venous return.

During tachycardia the most striking feature was the dye dilution curve showing an early recirculation typical for a left-to-right shunt. In our set-up (pulmonary artery injection-aortic arch sampling) we are unable, however, to locate the exact level of the shunt. Though in the dye dilution curve 2 (Fig. 1), the recirculation is almost indistinguishable in the descending part of the curve, no doubt is left about the existence of the early recirculation in curves 3 and 4. The calculated left-to-right shunt, 46 and 72 per cent of the pulmonary blood flow, according to Carter *et al.*, 1960, is of considerable importance.

The manifestation of this shunt during tachycardia is conditioned by an increase in the pressure gradient between the sites of the performed shunt. Therefore, both persistent ductus arteriosus and ventricular septal defect (assuming no pressure gradient across the aortic valve) seem to be less probable, since the systolic pressure gradient between the aorta and the pulmonary artery diminished during tachycardia.

Using the diastolic pressure in the pulmonary artery as an index of left atrial pressure (Kaltman *et al.*, 1966), we assume an increase in the pressure gradient between the left and right atrium during tachycardia. The calcu-

FIG. 1 Dye dilution curves by injecting cardiogreen into the pulmonary artery (PA-Ao) and into superior vena cava (VCS-Ao). CO, cardiac output; SV, stroke volume; I, quantity of indicator in ml.

lated pressure gradient between the left and right atrium increased from 3 mm. Hg at rest to 12.6 mm. Hg during tachycardia.

During tachycardia, the right atrial pressure remained unchanged in spite of the great increase in pulmonary blood flow. This might be explained by a better compliance of the right atrium in comparison with the left atrium (Ekelund and Holmgren, 1967); these authors have shown that in normal subjects during graded exercise the pulmonary wedge pressure increased with increasing heart rate whereas the right atrial pressure slightly decreased. The compliance of the left atrium was shown to be much inferior to that of the right atrium also by Little (1949).

We have no direct evidence about the level of the shunt, but we assume a left-to-right shunt at the atrial level. The manifestation of the shunt could be explained by a different compliance of the left and right heart during the paroxysm of tachycardia. On the basis of our observation we assume that the increase in heart rate and cardiac output either by

FIG. 2 Haemodynamics. CO, cardiac output; SV pulm, right ventricular stroke volume; SV syst, left ventricular stroke volume; $\dot{Q}_s/\dot{Q}_p\%$, shunt as a percentage of the pulmonary flow; P_{PA} , pulmonary arterial pressure; $\bar{P}_{RA}$, right atrial pressure mean.

exercise or by electric stimulation of the heart might be useful for proving the presence of intracardial shunts. The use of higher frequency electric stimulation seems to be more advantageous because of the possibility of a simultaneous angiographic examination. This assumption, however, requires verification by further experimental studies.

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