A Case Report of the Use of Inflow Occlusion and Moderate Hypothermia for a Pulmonary Valvotomy: Anaesthetic and Surgical Management

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INTRODUCTION

Pulmonary valvotomy was one of the earliest cardiac operations attempted. Indeed the success with which it was done did much to dispel the myths of the impossibility of operating on the heart, and ushered in the subsequent advances in cardiac surgery. This is a report of a recent case, followed by a discussion of the relative merits of different techniques.

Case report

The patient was an eighteen-year-old male weighing 34 kg. He had been admitted for investigations for failure to thrive in 1984, when a pulmonary stenosis was noted. Angiography done in February 1985 revealed a pure valvular stenosis. The right ventricular pressures were 120/0 mm Hg and the pulmonary artery pressure 23/12 mm Hg.

He was admitted for a pulmonary valvotomy, his major complaint being of moderate limitation of exercise. On physical examination he was of small stature and had signs of pulmonary stenosis and right ventricular hypertrophy. Features of right ventricular hypertrophy were present on his ECG and chest X-ray. Premedication consisted of 5 mg morphine given IM one hour preoperatively. After connection of ECG monitoring and placing peripheral venous and arterial lines, anaesthesia was induced with droperidol 2.5 mg, morphine 14 mg, diazepam 2.5 mg, and alcuronium 15 mg. He was then intubated and ventilated with 50 per cent oxygen in nitrous oxide. During normothermic periods 0.2 per cent trichloroethylene was added. He was given 20 mg dexamethasone and a central venous pressure monitoring line placed via the right internal jugular vein. In addition, oesophageal and rectal temperature probes, and a oesophageal stethoscope were placed. He was then put in an ice bath, taking care to avoid contact of the ice with his extremities. No additional vasodilators were given. After 80 minutes when the oesophageal temperature dropped below 33 °C, he was removed from the ice bath and the operation commenced. The temperature-after-drop continued below 32 °C. During cooling he was cardiovascularly stable with minimal change in heart rate or blood pressure. He did develop T waves on the ECG.

Through a median sternotomy the pericardium was opened to reveal a hypertrophied right ventricle. The right atrium was normal and so was the left ventricle and atrium. A palpable systolic thrill across the pulmonary valve was felt. Tapes were put round the Superior Vena Cava (SVC), the Inferior Vena Cava (IVC) and the pulmonary artery. The tapes around the SVC and IVC were snugged and after a few heart beats the pulmonary artery was opened longitudinally just distal to its origin. The pulmonary valve was found to have a diameter of approximately 3 mm. The commissures split easily on digital pressure.

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opening the valve to 12 mm. There was no subvalvular stenosis palpable. The pulmonary artery was closed using 5/0 Proleme and haemostasis achieved. The snares on the SVC and IVC were released. Unfortunately pressures in the right ventricle and pulmonary artery could not be measured to determine if any gradient still existed.

Just prior to the inflow occlusion isoprenaline 4 µg and sodium bicarbonate 32 mmol were given, in order to maintain normal cardiac conduction during the ischaemic time. The total occlusion time was eight minutes and the rhythm on the ECG remained sinus throughout. When flow was restored the systolic blood pressure was 70 mm Hg but rapidly improved to 90 mm Hg after 4 µg isoprenaline and 40 mmol sodium bicarbonate and 500 mg calcium chloride. Post-operatively the patient was then warmed using vacolitres of warm saline and heated blankets. It took 90 minutes for his temperature to exceed 35° C whereupon he was transferred to the intensive care unit for post-operative ventilation. No inotropic support was needed. He was extubated on day three and made an uneventful recovery. When seen in the out-patients six weeks post-operatively he was asymptomatic. He was able to walk briskly without any dyspnoea. There was no murmur heard and no changes on his chest X-ray although the ECG still showed evidence of right ventricular hypertrophy.

DISCUSSION

Pure pulmonary stenosis may present a wide spectrum of severity, ranging from a near atresia that presents in infancy, to a relatively mild variant that was present in this case. There are three techniques for the surgical correction of pulmonary stenosis. The first and earliest was to split the stenosis indirectly via the right ventricle. It has the advantage that it is the quickest technique, but in at least 25 per cent of cases it results in no drop in the pressure gradient between the right ventricle and pulmonary artery.

The second technique is the one used in this case. It consists of hypothermic circulatory arrest followed by valvotomy under direct vision. It was introduced by Swan and Blount in 1954. They reported very low mortality figures if the stenosis was not associated with other cardiac defects. Elective circulatory arrest for periods of up to eight minutes under moderate hypothermia have been used in neurosurgical operations without causing neurological deficit.

The third technique is direct valvotomy while the patient is on cardio-pulmonary bypass. It was first reported by McGoon and Kirklin in 1958 who claimed two advantages. Firstly, it allows repair of unsuspected associated defects (principally an atrial septal defect). Secondly, it allows the infundibular hypertrophy to be corrected. However, it has been well documented that the infundibular hypertrophy regresses if an adequate valvotomy has been done. No substantial series has been done comparing the risks and benefits of using inflow occlusion against extracorporeal circulation. Some consider the inflow occlusion technique as the method of choice, while others consider it be used only as a life-saving measure in palliating a severe stenosis in infancy.

Little has been written on the anaesthetic management of these cases. The general principles followed in this case were to maintain pulmonary perfusion by avoiding undue myocardial depression and maintaining the filling pressure at pre-operative levels. In those cases where an infundibular hypertrophy might be present, excessive sympathetic activity will worsen the cardiac output in a manner analogous to that in HOCM.

Just prior to the period of circulatory arrest sodium bicarbonate and a small dose of isoprenaline were given so as to marginally increase the heart rate. This would hopefully maintain normal conduction during the ischaemic period, as it did in our case. In conclusion we would confirm that direct pulmonary valvotomy using inflow occlusion and moderate hypothermia is an acceptable and relatively simple technique.

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