EDITORIAL

Closure of a large patent ductus arteriosus in adults: first do no harm

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Patent ductus arteriosus (PDA) is an open communication between the upper descending thoracic aorta and the origin of the left pulmonary artery. It is the result of persistent patency of the foetal arterial duct and has an incidence of up to 1 in 2000 of the population born at term. At birth, the arterial duct is patent but spontaneous closure occurs during the first 24 h of life with contraction of the smooth muscle in the media. This functional closure is assisted by the approximation of the intimal cushions which protrude into the lumen. Complete closure usually occurs by the age of 3 weeks as a result of diffuse fibrous proliferation of the intima. But in 10%, this process may be delayed by several weeks. Closure usually begins at the pulmonary artery end and may remain incomplete adjacent to the aorta leaving an aortic ampulla from which the ductal ligament arises.¹ There is some variation in the morphology of a patent ductus, which may be long or at the other extreme, characterised by a window between the descending aorta and proximal left pulmonary artery. When there is persistent patency of the arterial duct, it histologically differs from the normal duct and from the adjoining great arteries.¹ It may therefore be considered a congenital abnormality of the wall although, currently, at least 15% of children presenting with a small to moderate arterial duct at our institution have been born prematurely. Calcification within the wall is encountered commonly but exclusively in adults.

In 1938, Robert Gross working at the Boston Children's Hospital, Boston, Massachusetts, USA, successfully ligated the arterial duct of a 7-year-old girl² and thus, began the progress of surgical treatment for congenital heart malformations. The era of transcatheter closure of patent ductus began tentatively in 1971³ and accelerated progress continued after 1985,^{4,5} and so this method became the treatment of choice for the majority of patients in most institutions. Because of the early introduction of surgical treatment that also antedated precise methods of diagnosis,⁶ the natural history of patent arterial duct is poorly documented. In infants with a large duct, death results from congestive cardiac failure, sometimes complicated by a chest infection or pneumonia.⁷ For those surviving infancy, death is likely to occur in their 20s to 40s from pulmonary vascular disease or left ventricular failure.

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Although unusual in a large duct, endocarditis is responsible for almost half of the deaths in patients who do not have surgical treatment. Campbell⁷ estimated that heart failure was the cause of death in 30% of all patients with PDA. Closure of a patent ductus by surgery or interventional cardiac catheterisation is very safe and hospital mortality has approached zero. When moderate or severe pulmonary vascular disease has developed preoperatively, late death may result from its progression. When operation is performed in adult life and when there is an advanced and long-standing chronic congestive heart failure, premature late deaths may not always be avoided because of irreversible left ventricular dysfunction.

In Western Europe and North America, it is unusual to be faced with a patient who has a large and untreated PDA and is beginning to develop pulmonary vascular disease or chronic left ventricular failure. Such patients are much more frequently encountered in the developing countries. In this issue, Yan and coworkers⁸ (see page 514) report the successful closure of a large patent arterial duct associated with pulmonary hypertension in 20 patients. An Amplatzer PDA occluder or muscular ventricular septal defect occluder was used temporarily to close the duct to assess the change in pulmonary arterial pressure, pulmonary vascular resistance and aortic pressure. With a satisfactory improvement and without immediate clinical deterioration of the patient the closure device was released. This interesting paper raises a number of important questions about the management of such patients.

By convention, patients with a pulmonary vascular resistance of >6 units when breathing 100% oxygen are considered unsuitable candidates for repair of congenital heart defects usually associated with left to right shunts.^{9,10} The reason for this is the higher operative risk of complication or death, the likelihood of progressive pulmonary vascular disease despite repair of the defect and improved symptoms and survival in patients with pulmonary vascular disease but the continuing capacity for right to left shunting—for example, across a patent arterial duct. Among the 20 patients undergoing PDA closure, 12 had a pulmonary vascular resistance of >6 units in oxygen and six of these had a resistance value >8 units, a figure that is generally considered to be indicative of severe pulmonary vascular disease. It seems probable that, despite the initial fall in pulmonary arterial pressure, many of these patients will develop progressive pulmonary vascular disease and eventually exhibit worse

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symptoms than they would have had if the PDA had remained open.

Another important aspect is the accurate measurement of pulmonary vascular resistance.⁹ For patients breathing oxygen, it is crucial to include values for dissolved oxygen in the calculation of resistance. Failure to include dissolved oxygen in the measurement of pulmonary blood flow results in an underestimation of resistance. Many cardiac catheterisation laboratories do not have the facilities for the measurement of oxygen consumption,⁹ and instead, assumed values from tables are used. This inevitably provides an additional potential source of inaccuracy in pulmonary vascular resistance measurements. It is not clear from the text if assumed oxygen consumption was used or if resistance measurements took dissolved oxygen into account.

So, while on the one hand, it is possible to safely close a very large arterial duct in patients with pulmonary hypertension, it seems inevitable that at least some will develop progressive and fatal pulmonary vascular disease and eventually be worse-off than they would have been in terms of symptoms and life expectancy. Some patients had resistance values >15 units. The key to the safe management of these patients is the accurate measurement of pulmonary vascular resistance and management based on a resistance value rather than direct measurement of pulmonary artery pressure, which is never an accurate reflection of resistance. Management based on the direct measurement of pulmonary artery pressure during duct occlusion, although in common practice, is inevitably flawed.

Finally, in patients such as these, it is arguable that successful duct closure should be followed by an aggressive therapeutic approach to reduce pulmonary vascular resistance using pulmonary vasodilator agents, if possible.

Such treatment is of course extremely expensive, rarely successful in achieving a normal resistance and inappropriate in many developing countries, and should be part of research protocols and audit. For the group of patients reported, it is important that follow-up should include measurement of pulmonary vascular resistance at a given period after closure of the duct. Such a follow-up study might well be worthy of publication and will help to enhance our understanding of pulmonary vascular disease in this group of patients. However, the management of patients with a raised pulmonary vascular resistance and patent arterial duct must always be based on sound and reproducible physiological measurements and the underlying principle of "first do no harm."

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