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Editorial Comment

Pulmonary arterial angioplasty: heed the message – why bother?

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'False facts are highly injurious to the progress of science…'
Charles Darwin

If we read between the lines of the review by Bergersen et al.,[1] they tell us that angioplasty of the branches of the pulmonary trunk is fraught with ineffective dilation and complication, and is not for the faint-hearted. Their report is but a snapshot of the leading experience in the World on this subject. What can we learn? We can learn that, with procedure times of several hours, periods of fluoroscopy in excess of an hour, the need for transfusion of blood in three-tenths, and admission to intensive care of one-quarter of their patients, that poor results in their hands are unlikely to be due to lack of technique or perseverance. Indeed, I salute these latter qualities with admiration. Once again, therefore, we must conclude that we are defeated by the anatomic substrate.

The indications for pulmonary arterial angioplasty are threefold:

- To reduce right ventricular hypertension, thus avoiding long term adverse effects of hypertrophy, adverse myocardial metabolism, and potential ischaemia on right ventricular function.
- To balance the flow of blood into the lungs, improving the matching of ventilation to perfusion, avoiding the deleterious effects of disproportionate growth of the lungs, particularly in those patients who have undergone thoracotomy, of leading to scoliosis, with the inevitable concomitant effect on pulmonary function, which is compounded by the coexistence of the initial cardiac problem.
- To encourage flow into distal pulmonary arteries, thereby favourably influencing secondary pathology such as pulmonary valvar regurgitation, reduction in right-to-left shunting at atrial or ventricular level, where it is an issue, and improving systemic cardiac output.

And, in so doing, to improve exercise tolerance.

It is obvious, then, to conclude that any assessment of efficacy of relief of pulmonary arterial stenosis should include:

- Comparison of haemodynamics before and after the intervention, particularly the ratio of the right ventricular to the systemic pressure.
- Comparison of the distribution of the flow of blood into the lungs prior to and after the angioplasty, using isotopic techniques, or more recently, the technique of magnetic resonance imaging.
- Assessment of pulmonary regurgitation, ventricular volumes and output before and after angioplasty using magnetic resonance imaging.
- Objective demonstration of change in exercise capacity, such as walk testing, uptake of oxygen, and anaerobic threshold.

But the history of angioplasty of the pulmonary arteries tells us that there is not a single paper on the subject with reliable objective data using these methods to assess efficacy. Why?

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Deep down, we have a sincere desire to make pulmonary arterial angioplasty successful in the setting of multivessel disease. In this regard, witness the development of high pressure balloons, cutting balloons, rotablators and stents. We can probably influence the diameter of a vessel. But we are far less likely to influence the greater picture, specifically the haemodynamics, the physiology, and the symptoms. We rationalise against the more objective tests above. They are time-consuming, need to be repeated, and may involve the need for general anaesthesia, intravenous injections, and radiation. In fairness, some tests, particularly exercise and those involving general anaesthesia, cannot be universally applied. Like all investigations, there are issues of resolution, interpretation and reproducibility. Our efforts admirably are to be pastorally supportive to the patient and family, who with this disease by definition have their lives dominated by visits to hospital and inconvenience. So we look elsewhere for assessment, but in so doing we pull the wool over our eyes.

Intuitively, we see a stenosed vessel as the enemy, and fight its resistance to dilation with the ever-invasive tools of angioplasty described above, misguidedly measuring the result of the fight by comparing the diameters of the vessels, and the gradients across the stenotic areas, both of which are potentially seriously flawed as determinants of success. We learn from Bergersen et al.[1] that representative diameters of the vessels are often very small, no more than 2 millimetres or so. We also learn that, when using diagnostic catheters of 4 and 5 French gauge, considerable pressure dampening is to be expected, so measurements of gradients are unhelpful. Furthermore, in the setting of unilateral disease, with compliant vessels in the other lung, or in patients with a ventricular septal defect, pressure decompresses through the compliant lung or the septal defect, making measurements of gradients close to useless. Although the technology needed to provide quantitative imaging continues to improve, we accept the physical potential for error in measuring small changes in diameter, particularly relevant in distal vessels. Moreover, we acknowledge that the calibre of the vessels may increase in a dimension not witnessed by planar imaging. Recoil is also acknowledged, the sucker punch for interventionists, with the "successful" result of today proving to be a Pyrrhic victory tomorrow.

Inadvertently, surely never deliberately, we have been choosing tools for assessment to suit our ego. In so doing, however, could it be that we have not critically assessed the results? Back we go to the future. Quod erat demonstrandum.

Reference

1. Bergersen L, Gauvreau K, Lock JE, Jenkins KJ. Recent results of pulmonary arterial angioplasty: the differences between proximal and distal lesions. Cardiol Young 2005; 15: 597–604.

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