EDITORIAL

Collaterals: how important are they?

Gerald S Werner

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The question of "how important collaterals are" is a rhetorical one. The essential value of collaterals is clearly demonstrated in patients with a completely preserved left ventricular function despite a totally occluded coronary artery. There is even the anecdotal argument that collaterals might be so well developed that the revascularisation of an occluded coronary artery might not be required, but clinical data suggest that revascularising a chronic coronary occlusion provides a survival benefit.[1 2] Furthermore, the quantitative assessment of collateral function demonstrated that <5% of occluded arteries in patients without a prior myocardial infarction receive collateral supply equivalent to an open epicardial artery.[3]

Recovery of functionally impaired myocardium after revascularisation can only be expected with a minimum collateral supply to maintain myocardial viability.[4] In the absence of collaterals, no viable myocardium will be found,[5 6] but there is a dispute about whether the extent of collaterals is directly related to viability and the potential for functional recovery.[7 8] The history and circumstances of the development of a chronic occlusion are the major determinants of how extensively myocardial function will be impaired. The occlusion may occur acutely, with symptoms of an acute myocardial infarction leading to irreversible myocardial damage and a border zone of hibernating and stunned myocardium. We could show by invasive assessment of collateral function that collaterals in patients with a recent myocardial infarction take about 3 months to develop to a functional capacity similar to that of collaterals that must have been present at the time of an occlusion and were capable of preserving myocardial function.[9 10]

Collateral development does not depend on the presence of viable myocardium, as collaterals develop because of increased shear stress along preformed interarterial, arteriolar connections and do not require an ischaemic stimulus.[11] Therefore, well-developed collaterals may also be observed with non-viable myocardium. When the occlusion occurs gradually with symptoms of preinfarct angina, there might be time for a gradual collateral development leading to prevention of transmural infarction. However, the collateral may not be sufficient to uphold full nutritional supply, hence the myocardium may remain viable but non-functional—that is, hibernating. One of the major causes for the wide variability and lack of correlation between collateral function, and myocardial viability and ventricular function, besides the time course of the occlusion, seems to be an individual predisposition, with the presence of immediately recruitable collateral connections in about 20% of patients.[12 13]

THE PROBLEM OF COLLATERAL ASSESSMENT

The aforementioned relationships and interactions between collaterals and myocardial function could be assessed only recently after improvements of collateral assessment in man. The in vivo assessment of collaterals became possible with the introduction of coronary angiography, and initial studies meticulously analysed collateral radiographic anatomy, and these findings are still valid to some extent even today.[14] The most widely used index of collateral function introduced by Rentrop *et al*[15] more than 20 years ago was easy to apply, but only semiquantitative. However, these angiographic methods were not sufficient to provide more detailed insights into collateral physiology, and needed to be refined or replaced by a quantitative assessment of collateral function.[16–18] The main limitation of the quantitative assessment by intracoronary Doppler flow and pressure probes is the need for a balloon occlusion to study recruitable collaterals, or the passage of a chronic occlusion with microcatheters, which makes it difficult or impossible to be used for longitudinal studies of collateral function.

The method presented by Muehling *et al*[19] (***see page 842***) could provide a non-invasive approach with the additional advantage of not requiring exposure to radiation in contrast to angiography or radionuclid scintigraphy. MRI is already the gold standard for detecting myocardial viability and predicting functional recovery after revascularisation of chronic coronary occlusions,[20] and the functional study of collateral supply would add a further aspect to this multidimensional diagnostic tool. The paper in this issue only assessed the predictive value of the perfusion delay for the presence of a chronic coronary occlusion. Together with the measurement of absolute myocardial perfusion, which is not possible with the present invasive methods of collateral assessment, this might provide a tool for non-invasive assessment of collateral function.

Some issues certainly remain to be addressed in the validation of this non-invasive method of perfusion delay and perfusion quantification with collateralised chronic coronary occlusions. One issue well known from scintigraphic studies is the influence of coronary steal on the perfusion distribution during pharmacological stress.[21] The authors had tried to account for such influences by excluding patients with a lesion of >50%

Correspondence to:
Professor G S Werner,
Medizinische Klinik I,
Klinikum Darmstadt,
Grafenstrasse 9, D-64283
Darmstadt, Germany;
gerald.werner@
klinikum-darmstadt.de

angiographic stenosis in the potential collateral donor artery, but this may not be sufficient to eliminate the influence of coronary steal.[22] A future validation of this method should include a comparison with the invasive assessment of collateral function during pharmacological stress.

The investigation of collateral function would be advanced considerably by a reliable, repeatable, quantitative non-invasive assessment of collateral function. All interventions to promote collateral development, such as the influence of pharmacological interventions, the often discussed but never proven effect of physical exercise training, and, above all, molecular genetic approaches, would benefit from such a measurement tool to replace less valuable surrogate parameters, such as exercise capacity or clinical symptoms.

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IMAGES IN CARDIOLOGY

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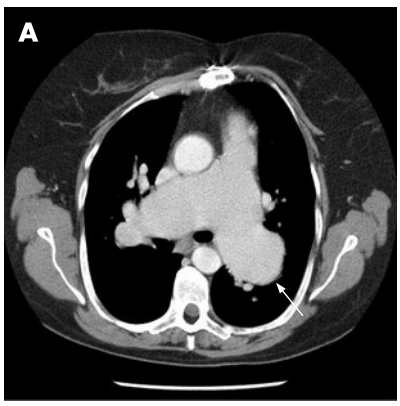
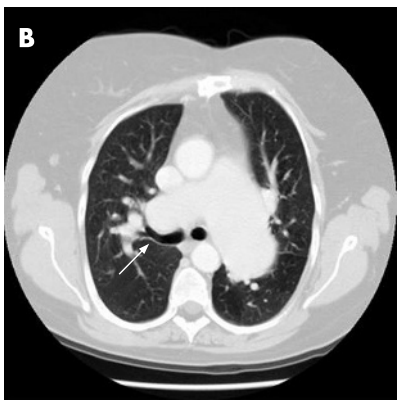
Congenital absence of the pulmonary valve





The CT scans show dilated pulmonary arteries (PAs) (48 mm) in a 59-year-old woman. Absent pulmonary valve syndrome (APVS) was considered at tetralogy of Fallot (ToF) repair in 1961, when no pulmonary leaflets were seen. Moderate dilatation of PAs (34 mm) was noted when a pulmonary homograft replacement for pulmonary regurgitation was inserted in 1997. Recurrent chest infections, breathlessness with good LV and RV function (PA pressure 28/14) and hoarseness secondary to a recurrent laryngeal nerve palsy led to CT scanning, confirming large PAs (panel A) compressing the right middle lobe bronchus (panel B).

APVS is a rare congenital malformation of unknown frequency. It may occur in 3–6% of patients with ToF, but can be associated with ventricular septal defect, atrial septal defect, coarctation of the aorta and tricuspid atresia. Characteristic features include aneurysmal dilatation of PAs, which often leads to major bronchial and intrapulmonary compression, obligate pulmonary regurgitation and degrees of pulmonary branch stenoses.

Long-term follow-up of APVS without early plication of PAs is limited. Management in this case is difficult but may include right middle lobe resection or a Lecompte procedure (translocation of the PA anterior to the aorta away from the airways to eliminate bronchial compression), with PA plication.

S Howard, S Wan, L J Freeman
leisa.freeman@nnuh.nhs.uk