Reflections on wave reflections in chronic thromboembolic pulmonary hypertension

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This editorial refers to 'A novel echocardiographic predictor of in-hospital mortality and mid-term haemodynamic improvement after pulmonary endarterectomy for chronic thrombo-embolic pulmonary hypertension'† by M. Hardziyenka et al., on page 842.

Chronic thromboembolic pulmonary hypertension (CTEPH) is a surgically curable form of severe pulmonary hypertension. However, in ~10% of the patients, the procedure may not be successful, because of persistent pulmonary hypertension after removal of proximally located thromboembolic material. This complication is of particularly poor prognosis. In the present issue of the European Heart Journal, Hardziyenka et al. report on its prediction by the analysis of Doppler pulmonary arterial waves. The authors defined a time to notching expressed as a notch ratio (NR), or the ratio of time from onset of flow to maximum flow deceleration to time from maximum flow deceleration to end of flow. This NR was found to be associated with in-hospital mortality and increased systolic pulmonary artery pressure at 3 months. The authors explain these results by the effects of proximal as opposed to distal wave reflection. Thus an increased NR would allow for the identification of peripheral small vessel disease that is not amenable to surgery. The report of Hardziyenka is remarkable, because it introduces a simple measurement that is easily integrated into routine echocardiography, for great clinical relevance and a lot of physiological sense.

The pulmonary circulation is a low resistance and high compliance circuit with little wave reflection. Therefore, normal pulmonary arterial flow and pressure waves present with rounded contours and are superposable, in contrast to aortic pressure and flow waves where wave reflection determines a phase lag and early systolic peaking of flow with late systolic peaking of pressure. Patients with pulmonary hypertension present with a right ventricular pressure wave with a sharp initial upstroke, followed by a short plateau, and a late systolic peaking, a pulmonary wave with a huge pulse pressure, and a flow wave with a shortened time to peak velocity and a late or mid systolic deceleration. All these changes are largely determined by wave reflections.

The effects of wave reflection on pulmonary artery pressure and flow waves can be shown experimentally by the comparison of the effects of proximal and distal obstruction, respectively by pulmonary arterial banding and injected of small glass beads, to produce the same increase in mean pulmonary artery pressure. As illustrated in Figure 1, proximal obstruction causes a mid-systolic deceleration of flow even when mean pulmonary artery pressure is only moderately increased. This can be further analysed in the frequency domain to decompose waves into their forward and backward components. In the case of the example shown in Figure 1, mid-systolic notching is clearly caused by the subtraction of an early returned reflected wave on the forward wave. Wave reflection explains previously reported shorter time to notching on pulmonary arterial flow waves in embolic pulmonary hypertension when compared with pulmonary arterial hypertension (PAH), in spite of lower mean pulmonary artery pressures. This result would not be affected by the adjustment of time to notching to heart rate, which is inherent to the NR as calculated by Hardziyenka et al.

While a proximal site of reflection on thromboembolic material is an obvious cause for an earlier return of a reflected wave, this can also be caused by an increased wave speed, or, as shown in the initial report of pulmonary artery flow patterns to evaluate pulmonary hypertension, by a longer preceding R–R interval in an arrhythmic patient. Pulmonary arterial wall distension with decreased compliance as a consequence of high pressures increases wave speed. This is why mid-systolic deceleration of pulmonary flow is also seen in patients with severe PAH, in spite of a site of resistance and wave reflection that is at the periphery of the pulmonary arterial tree.

These physiological notions were recently challenged by the report of a close correlation between systolic, mean, and diastolic pulmonary artery pressures in pulmonary hypertension of various severities. The implication of this observation is that any pulmonary artery pressure can be predicted from any other, with for example mean pulmonary artery pressure reliably estimated from 0.6 times systolic pulmonary artery pressure plus ~2 mmHg. While it is
likely that the functional adaptation of the pulmonary circulation to disease processes is generally monotonous, with any change in pulmonary vascular resistance associated with proportional changes in compliance and wave reflections, embolic pulmonary hypertension is particular by more predominant wave reflection as a cause of a disproportionate increase of systolic pulmonary artery pressure. Nakayama et al. showed indeed that pulmonary artery pressure waveform analysis by the difference between systolic and diastolic pressures divided by mean pressure or the augmentation index defined by the change in pressure from the short plateau seen on the upstroke of the pulmonary artery pressure curve to peak pressure divided by pulse pressure as an index of wave reflection were both useful for the differential diagnosis between CTEPH and idiopathic PAH. However, these findings could not be reproduced by Castelain et al., who nevertheless confirmed a tendency to increased and anticipated wave reflection in CTEPH patients. Thus, pressure wave morphology analysis alone may be disappointing, which is probably explained by a predominant effect of increased wave speed when pulmonary artery pressures are very high. Pressure waveform analysis has not been used yet to identify a peripheral component to increased pulmonary vascular resistance in CTEPH. Perhaps that it could be combined to flow wave analysis to further improve the prediction of the NR proposed by Hardziyenka et al.

Pressure and flow wave analysis can also be performed in the frequency domain, with results expressed as impedance spectra or pressure on flow ratios as a function of frequency. Huez et al. recently showed that this is feasible, with a posteriori synchronization of pressure and flow signals measured respectively with standard fluid-filled pulmonary artery catheters Doppler echocardiography. However, this semi-non-invasive approach has not yet been applied at the bedside, probably because of the conceptual difficulty physicians often have with frequency domain reasoning.

Another approach for the identification of distal vasculopathy in CTEPH is the analysis of pressure decay curves after pulmonary arterial occlusion. Such curves are made of a first fast component, which corresponds to the stop of flow through arterial resistance, and a slower component, which corresponds to the emptying of compliant capillaries through a venous resistance. There is therefore an inflection point, from which one calculates an upstream resistance, essentially determined by the resistive properties of the large pulmonary arteries, and a downstream resistance determined by the cumulated resistances of small arterioles, venules and capillaries. A study on a small series of CTEPH patients referred for thrombo-endarterectomy showed the excellent predictive values of residual pulmonary hypertension and associated mortality by a relative increase in downstream resistance. This result awaits multicentre confirmation on larger patient populations.

The introduction of right heart catheterization with balloon-tipped thermodilution catheters by Swan et al. in 1970 heralded a considerable progress in the understanding of unstable haemodynamic states, heart failure, and pulmonary hypertension. But also disappointments, related to insufficient integration of the measurements into clinical context, and probably also exclusive reliance on automated mean flow and pressure measurements. Haemodynamic studies are currently refocusing on original pressure and flow signals. For the evaluation of pulmonary hypertension, it is fascinating to see Doppler echocardiography gaining more prognostic importance than a standard right heart catheterization. In this respect, the report of Hardziyenka et al. is a major stride in the good direction that should not be hindered by backward waves.

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References


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Inadvertent grafting of the posterior cardiac vein during coronary artery bypass graft surgery

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A 74-year-old male patient who had a coronary artery bypass graft (CABG) surgery 14 years ago underwent coronary angiography for pre-operative evaluation for non-cardiac surgery. He was reported to have a left internal thoracic artery (LITA) graft to the left anterior descending coronary artery and a saphenous vein graft sequentially to the first and second obtuse marginal arteries. The coronary angiogram revealed a patent LITA graft. As the saphenous vein graft is engaged, the injection revealed simultaneous opacification of the obtuse marginal artery and the coronary sinus, which indicates inadvertent grafting of the posterior cardiac vein instead of the first obtuse marginal artery (Panel A, white arrow). The vein graft was then anastomosed sequentially to the second obtuse marginal. The left anterior oblique projection revealed retrograde opacification of the middle cardiac vein (Panel B, black arrow). As the patient was asymptomatic and no objective signs of ischaemia were found by non-invasive testing, we did not attempt any further intervention.