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CORRESPONDENCE
Coronary artery disease

Isn't coronary dicrotic wave analogous to reflected arterial pulse wave?

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The imaginative use of rest distal coronary pressure dicrotic wave to diagnose coronary microvascular dysfunction (CMD) by Fujimori *et al.*[1] is thought provoking. It is synonymous to arterial pulse wave reflection, which could augment systemic blood pressure giving rise to isolated systolic hypertension in the elderly, subject to stiff aorta, and elevated total peripheral resistance. Its waveform also varies along the course of the aorta. Coronary dicrotic wave similarly is a composite phenomenon due mostly to coronary artery elasticity with a resistance component. On the other hand, CMD is defined by impaired vasodilatory capacity, or the inability of flow to increase as needed, and not by resistance, neither vascular compliance, nor endothelial function. With catheter-based bolus thermodilution, which currently defines CMD, coronary flow reserve (CFR) and index of microvascular resistance (IMR) have a tenuous inverse curvilinear relationship.[2] Fujimori *et al.*'s index is akin to using IMR to prognosticate CFR. But even at the extreme end, very high IMR does not correlate with a CFR of < 2.0,

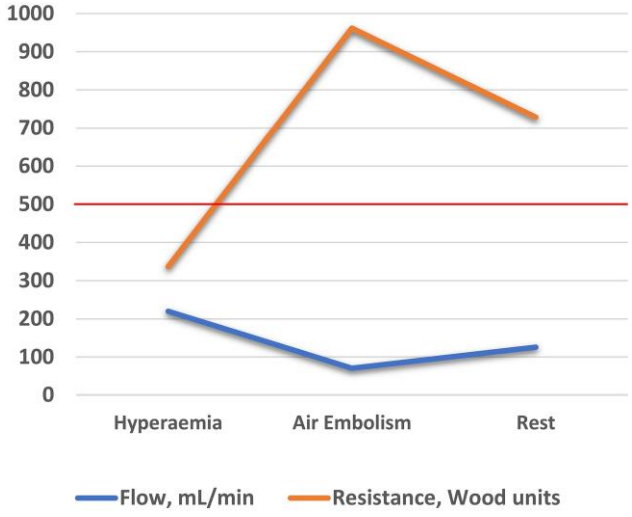

Figure 1 Transient rise in coronary resistance due to no-reflow from air embolism in the circumflex artery of a patient with convalescent Takotsubo syndrome on continuous thermodilution. The hyperaemic absolute microvascular resistance is normal at 337 (< 500) Wood units, and the rest resistance is 729 Wood units; hence the microvascular resistance reserve, MRR is 2.2 (< 2.1, for 'definite' CMD). The low CFR of 1.75 (220/126) is due to baseline vasodilatation with high flow and low resistance in an unobstructed coronary artery. Since resistance rises nearly three-fold in response to microvascular spasm, which means that the 'vasodilatory ⇔ rest ⇔ vasoconstrictive' range, or the 'true' MRR is a magnitude higher at 2.9, and a potential CFR of 3.1 (220/71). This case illustrates the pitfall of relying solely on CFR to define CMD, since coronary haemodynamics are everchanging, labile and circumstantial.

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the numerical standard for CMD. In other words, any surrogate for resistance cannot predict how much the flow will alter.

Further, true rest state rarely exists in the cardiac catheter laboratory. The coronary resistance dynamically and proportionally changes in response to blood pressure as per Ohm's law; pressure ∝ resistance × flow. Hence, an anxious patient with a hypertensive response will have a reactively increased coronary resistance. As an illustration, *Figure 1* shows a patient previously described[3] with an intact microvasculature and a paradoxically low CFR on continuous thermodilution. When air is inadvertently introduced down the circumflex artery, transient no-reflow occurs due to microvascular spasm, resistance triples with a corresponding fall in flow, implying an untapped 'vasoconstrictive reserve'. Likewise, caffeine to a lesser extent raises resistance as an adenosine antagonist, and it is not clear how this, or nicorandil pre-treatment could relate to CMD, i.e. by undermining hyperaemia, and then facilitating it.[1] It would be helpful if Fujimori *et al.*[1] could further elaborate their key assumption that a high ratio of distal coronary pressure to aortic pressure (Pd/Pa) beyond a coronary lesion with a low fractional flow reserve, FFR < 0.75 signifies CMD; that failure of microvascular dilatation in response to ischaemia causes this apparent divergence of Pd/Pa and FFR. However, a non-compliant vessel via an augmented pulse pressure would transmit an amplified mean pressure distally to account for a high Pd/Pa, an adaptive mechanism to accentuate forward flow. But when Pd/Pa falls precipitously with adenosine hyperaemia, this suggests preserved microvascular dilatory function. After all, the cardinal feature of CMD is a blunted hyperaemic response, giving a falsely high FFR for an otherwise significant lesion. Thus, syllogistically, a high Pd/Pa coupled with a higher than expected FFR across an anatomically flow-limiting lesion indicates both endothelial dysfunction and CMD.

Finally, even with bolus thermodilution, high rest flow of whatever cause spuriously reduces CFR since this is a ratio between hyperaemic and baseline flow. Therefore, the interpretation of low CFR should be more nuanced than comfirmatory of CMD. Absolute coronary resistance obtained from continuous thermodilution conceivably could overcome this uncertainty. Although flow down a coronary artery depends on the muscle mass subtended, counterintuitively resistance has no such limitation, that is, it has a reasonable concordance between different vessels in the same patient. The latter technique, which allows coronary flow and resistance to be accurately measured, gives confidence in diagnosing, or excluding CMD. Taking into account the above theoretical considerations, the shortcut method as proposed by Fujimori *et al.*[1] is unlikely to be verifiable.

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