HEART

Editorial

Is pressure recovery an important cause of "Doppler aortic stenosis" with no gradient at cardiac catheterisation?

The derivation of pressure data from Doppler ultrasound was first described by Holen *et al* in 1976.[1] Some 20 years later we remain obsessed by comparisons between pull-back "gradient" at catheterisation and peak or mean pressure difference derived from Doppler echocardiography. It should now be well-known that the two are related, but different. The pull-back gradient does not exist physiologically because the peaks of the left ventricular and aortic pressure waves do not occur simultaneously. The Doppler-derived peak instantaneous gradient is usually higher than peak to peak gradient at catheterisation, often by about 20 mm Hg but with a range from 1 to 53 mm Hg.[2] Pressure recovery is an underappreciated cause of this discrepancy.

What is pressure recovery?

As blood loses momentum beyond a stenotic aortic valve, the pressure within the aorta rises (fig 1). This is pressure recovery. More formally, Bernoulli's theorem states that in a closed system the sum of pressure head, potential energy, and kinetic energy must be the same in all parts of

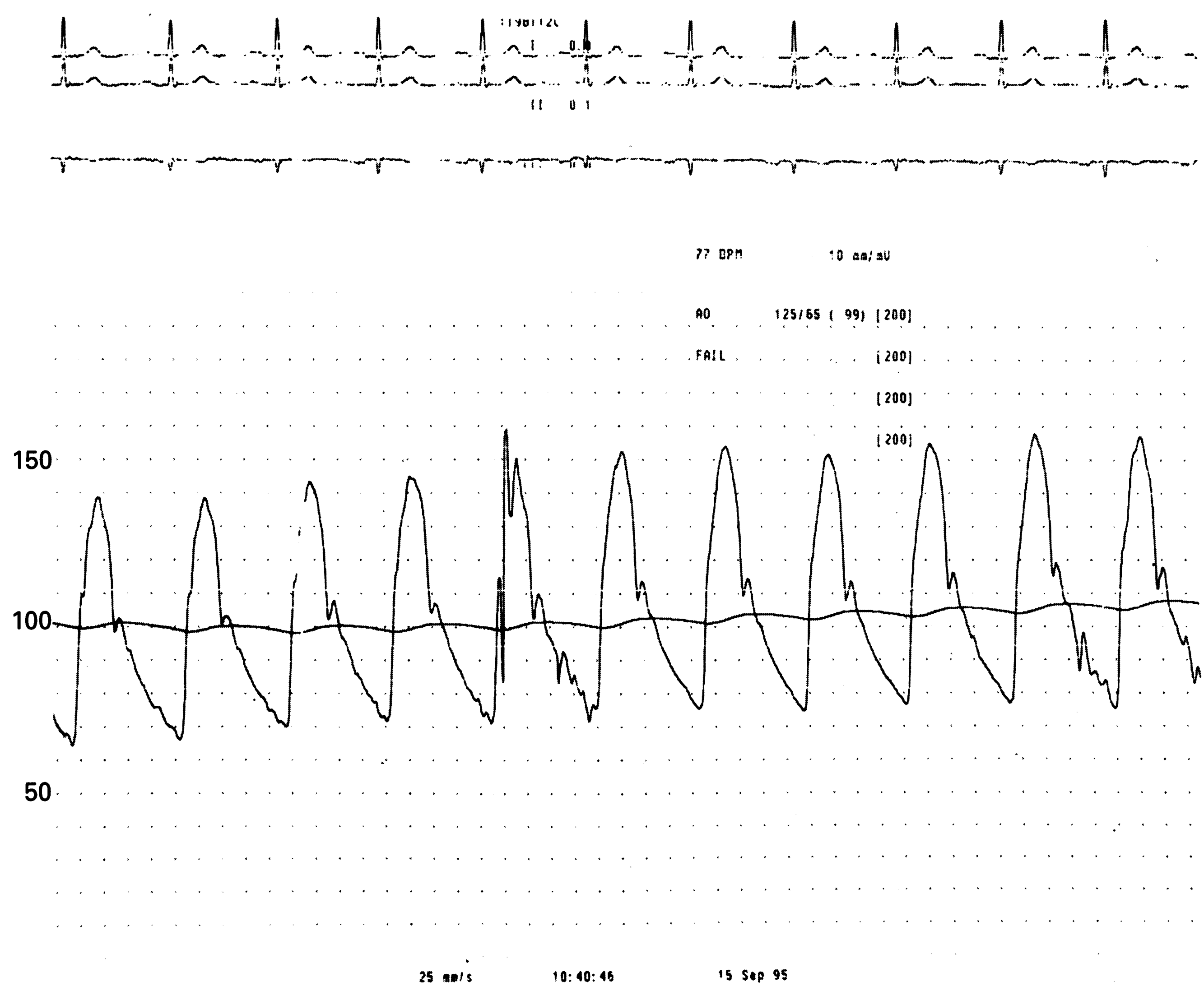

Figure 1 Pressure recovery. A pigtail catheter has been pulled from a point just beyond the aortic valve to the aortic arch. The pressure rises progressively by 15–20 mm Hg.

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the system. Thus assuming a constant pressure head, zero acceleration, and no viscous losses, a fall in static pressure across a valve must be accompanied by a rise in kinetic energy. This is the foundation for our estimation of pressure difference (ΔP) from velocity (v), which at its most simple uses the formula $\Delta P = 4v^2$. The reverse happens downstream from a stenosis where, as flow lines diverge and blood loses momentum, there is an ensuing rise in static pressure. However, not all the static pressure drop across the aortic valve is available for recovery.

The total pressure recovered is determined by the velocity of blood at the orifice, by the geometry of the aorta, and by the quantity of pressure energy dissipated as heat because of turbulence.[3 4] In severe diaphragmatic stenoses, the jet expands rapidly producing significant turbulence which reduces the total pressure available for recovery. In more streamlined stenoses, proportionately more pressure is recovered. Examples of such situations are subaortic tunnels, some types of mechanical replacement valve such as the St Jude,[5 6] and relatively mild native valvar stenosis where the cusps form a funnel rather than a diaphragm.[3 4] In an in vitro study 47% of the transvalve pressure drop was recovered (from 68 mm Hg to 36 mm Hg) in a 61% area stenosis compared with only 10% recovery (from 119 mm Hg to 107 mm Hg) in a 94% area stenosis.[7] Similar results have been found in replacement valves[8] (fig 2).

How important is pressure recovery?

A pigtail catheter cannot be held too close to the aortic valve because of whip artefact and in practice it is pulled across the valve towards the upper ascending aorta. At this point, pressure recovery is well advanced. It is usually complete at a distance 10 to 15 orifice diameters downstream from the aortic valve. Thus a pull-back gradient at catheterisation is really the pressure difference between the left ventricle and the ascending aorta. By contrast,

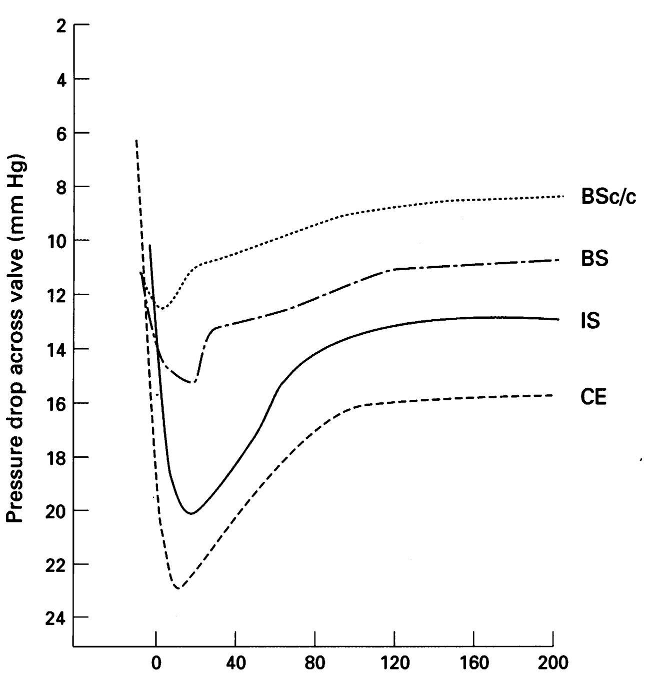

Figure 2 Pressure recovery in vitro. The rise in static pressure (y axis) is plotted against distance from the valve ring (x axis) in four different replacement valves: BSc/c, Björk-Shiley convexo-concave 23 mm; BS, Björk-Shiley ABP 23 mm; IS, Ionescu Shiley 21 mm; CE, Carpentier-Edwards model 2625 23 mm. Reproduced from reference 8 with the permission of the authors.

Doppler ultrasound estimates the instantaneous pressure difference between the left ventricle and the vena contracta, which is the point of lowest pressure where flow lines start diverging just beyond the aortic valve. Pressure recovery might therefore be expected to be an important determinant of differences between these two different types of pressure measurement.

However, in the clinical situation, there are many other confounding factors. Firstly, Doppler and catheter assessments are not made simultaneously so that the aortic disease or left ventricular function may have deteriorated between examinations or flow may be different as a result of posture, sedation, or loading conditions. Secondly, fluid-filled catheters may give erroneous pressure readings if there is damping or shatter. A pigtail catheter placed in the centre of the aorta close to the aortic valve where the velocity of blood is high may partially register the effect of kinetic energy as well as static pressure, with consequent underestimation of the true transvalvar drop in potential energy. Thirdly the velocity profile across the aorta may not be uniform and there may be large differences in pressure estimation depending on the placement of the catheter tip.[6] If the aorta is dilated, flow effectively goes from one reservoir to another and pressure recovery is limited. Finally, Doppler may occasionally underestimate velocity as a result of viscous losses related to the shape of the orifice [4 9] or occasionally the true maximum transaortic velocity may be missed, particularly if the echocardiographer fails to use all available windows.[10] In mild aortic stenosis the simplified Bernoulli equation is inaccurate.[2 3]

There are virtually no clinical studies of the effect of pressure recovery. Vandervoort *et al*[11] recently assessed the effect of pressure recovery in St Jude Medical valves in both the aortic and mitral positions and they showed that pressure drop and subsequent pressure recovery were greater in the central than the two sided orifices. They showed that the pressure difference on transoesophageal examination across the central orifice in the mitral position gave a higher result than the catheter gradient. However, in studies of the native aortic valve using simultaneous left ventricular and aortic catheters, Doppler tends to underestimate rather than overestimate relative to catheter-derived gradients, usually by about 10 mm Hg.[10 12] This suggests that pressure recovery is not often of major importance. Nonetheless, within these study populations are individual patients in whom the Doppler-derived peak instantaneous pressure difference is sometimes as high as 40 mm Hg, but in whom no gradient is found at catheterisation.[2] Pressure recovery as well as the inappropriate use of the shortened Bernoulli formula are likely to be the main explanatory factors in these cases.

Conclusion

Leaving aside the errors and uncertainties of the techniques, we can say that continuous wave ultrasound measures velocity at about the level of the aortic valve, whereas cardiac catheterisation measures the difference between left ventricular and the fully recovered static pressure in the aorta. These are different haemodynamic entities. Their comparison may reveal information about haemodynamic events downstream from the valve, but this comparison will be meaningless if it is attempted as a slavish auditing exercise.[13]

JOHN CHAMBERS

Adult Echocardiography,
Department of Cardiology,
Guy's Hospital,
Guy's and St Thomas' NHS Trust,
London SE1 9RT

Heart: first published as 10.1136/hrt.76.5.381 on 1 November 1996. Downloaded from http://heart.bmj.com/ on May 21, 2022 by guest. Protected by copyright.

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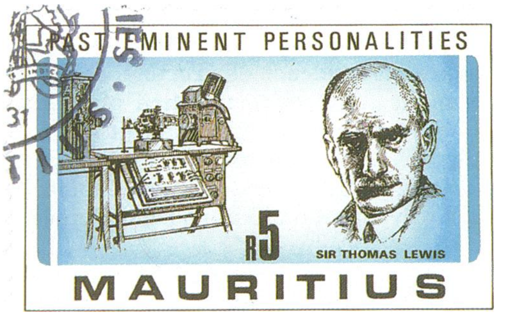
STAMPS IN CARDIOLOGY

Sir Thomas Lewis (1881–1945)



The only stamp depicting Sir Thomas Lewis was issued by Mauritius on the centenary of his birth. At five rupees it was the highest value stamp in a set featuring past eminent personalities, and the only one devoted to a non-Mauritian. The credit for it goes to the then Prime Minister of Mauritius, the late Dr Sir Seewoosagur Ramgoolam who had been a pupil of Lewis at University College Hospital London in the 1930s. The suggestion for the stamp came from Dr Arthur Hollman. The design shows the 1911 Cambridge string galvanometer electrocardiograph used by Lewis and his photograph at age 33.

Thomas Lewis was born in Cardiff of Welsh parents. Having written his first scientific paper at the age of 19, he graduated in 1905 from University College Hospital London where he worked for the rest of his life. Einthoven's invention of the string galvanometer in 1901 made clinical electrocardiography a practical proposition and Lewis did more than anyone to establish its value. His 1911 book *The Mechanism of the Heart Beat* was hailed as the bible of electrocardiography, and his electrophysiological work gained him the FRS. He identified atrial fibrillation and proposed that its mechanism was a "circus movement". In 1909 he founded, with James Mackenzie, the influential journal *Heart* and was its only editor. Although he was a founder member of the Cardiac Club and wrote a widely acclaimed book, *Diseases of the Heart*, Lewis was more than a heart specialist (he disliked the term cardiologist). His guiding star was experimental medicine, and he eagerly promoted the discipline of clinical science and founded the Medical Research Society. He gave up electrocardiography in 1925 and then had two equally successful periods of research, firstly on the blood vessels of the skin and the triple response to injury, and secondly on pain. Lewis drove himself at high pressure and nothing was allowed to interfere with the work in hand. He was a tough chief, but his co-workers, many of them American, were inspired by his search for the truth and greatly admired him. Away from work his chief love, ever since boyhood, had been in natural history and he was a fine bird photographer and a skilful fisherman. He died after a third myocardial infarction, having had the first at the age of 43.

M K DAVIES
A HOLLMAN

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