Correspondence

Blood flow patterns in the human aorta studied by magnetic resonance

Sir,

It was fascinating to see the measurements of aortic blood flow velocity obtained with magnetic resonance by Klipstein et al (1987;58:316–23). In the early 1970s a number of us struggled at the limits of the then available technology (mainly hot-film anemometry) to make measurements of this kind, and it is satisfying to see those results so elegantly confirmed and extended.

In only one respect are these new results at odds with previous work, and that is in the authors’ conclusion that turbulence was absent. Turbulence was seen in several studies in the ascending aorta of man and several other mammalian species1–3 and this is to be expected on the basis of fluid mechanics. I wonder whether this discrepancy is a matter of measurement technique?

Turbulence consists essentially of a disorganised collection of small eddies moving with the bulk flow. These cause high frequency small amplitude fluctuations in velocity that are very easily missed unless the sensing technique samples local flow and is sensitive to high frequencies. The energy content of the turbulence is small (typically less than 0.5% of the total kinetic energy in the flow) and the frequencies are high (up to several hundred Hz). Hot-film anemometer probes are ideally suited to detecting such turbulence because they are tiny and have a very high frequency response. Klipstein et al’s suggestion that the presence of such probes actually caused the turbulence seen in the earlier work is not, I think, plausible—flow turbulence caused in that way would only be detectable downstream, in the wake of the probe. Could an alternative explanation be that the magnetic resonance technique requires a degree of spatial or time averaging that makes it insensitive to low level turbulence?

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References


This letter was shown to one of the authors, who replies as follows:

Sir,

I note with interest the comments of Dr Seed, and agree with them. Despite the many advantages that we discuss in the paper, magnetic resonance velocity mapping does require both spatial and temporal averaging.

The spatial averaging is based on a typical voxel size in the region of 50 mm³ (the precise value varying both with the field of view and slice thickness). The temporal averaging arises because acquisition of the data for a series of frames takes several minutes, the images being reconstructed afterwards. The only detectable turbulence (seen as signal loss) is that which occurs repeatedly in the same place with every heart beat. High frequencies as such are not a problem because they increase the likelihood that the resultant of all velocities in the voxel over the 12 ms of data acquisition will be zero, and therefore that signal loss will occur.

The even echo rephasing that we use to retain the signal from moving blood (even though there may be a wide range of velocities passing through a single voxel) would be enough to eliminate the expected signal loss from small degrees of turbulence (as we mention at the end of the section on the principles of
magnetic resonance velocity mapping). Only gross turbulence can thus be detected by the technique. It is therefore very probable that turbulence accounting for less than 0.5% of the total kinetic energy would not be detected unless it was of large magnitude and consistently localised to a few adjacent voxels.

Within these small limitations, we feel that the convenience of magnetic resonance velocity mapping, particularly its non-invasiveness, opens the way to making practical a more complete understanding of the patterns of human blood flow, both normal and abnormal.

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Prediction of outcome in dilated cardiomyopathy

Sir,

A recent publication has prompted me to write concerning the diagnosis of myocarditis on endomyocardial biopsy. Diaz et al stated that they were “perplexed by the great increase in biopsy diagnosis of myocarditis since 1982” quoting that 21 of 39 cardiac biopsy specimens since March 1983 demonstrated “active myocarditis” or “healing” myocarditis. The Dallas criteria as quoted in Dr Billingham’s editorial on acute myocarditis clearly state that the diagnosis of ongoing (persistent) myocarditis, resolving myocarditis, and resolved myocarditis can only be made after a previous biopsy specimen has shown active myocarditis. Thus it seems evident that an initial, diagnostic biopsy specimen cannot be classified as “healing” myocarditis in the absence of earlier information. The changes may have causes other than myocarditis and these could account for the apparent increase in the diagnosis of myocarditis.

When considering myocarditis and dilated cardiomyopathy and the question of whether the former may cause the latter, it is vital that statements about biopsy specimens are accurate and that precise labels such as “healing myocarditis” are not attached to imprecise data.

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References


Notice

British Cardiac Society

The Autumn Meeting will be held at the Wembley Conference Centre, London, on 22 to 24 November 1988. The closing date for receipt of abstracts was 24 June 1988.

The Annual General Meeting for 1989 will take place in Oxford on 6 and 7 April 1989, and the closing date for receipt of abstracts will be 6 January 1989.