

Correspondence

Acceleration time in the aorta and pulmonary artery measured by Doppler echocardiography in the midtrimester normal human fetus

Sir,

I wish to take issue with Machado *et al* (1987;58:15-8) who measured aortic and pulmonary artery acceleration times in a series of human fetuses and inferred that pulmonary vascular resistance was increased. They based this conclusion on studies in adults that showed that acceleration time is shortened in the pulmonary trunk when pulmonary mean pressure is increased.¹ Machado *et al* inferred that the fetal pulmonary vascular resistance is higher because acceleration times were slightly shorter in the fetal pulmonary trunk than the aorta.

The observation that flow profiles in the aortic and pulmonary circuits are different is not new; it was reported in the fetuses of sheep by Rudolph.² It is well known that aortic and pulmonary pressures are equal in the fetus and that fetal pulmonary vascular resistance is high. The precise relation of the vascular resistances of the aortic and pulmonary circuits has, to my knowledge, not been investigated in any animal model. Because the ductus is widely patent in utero the vascular resistance reflected by events in the pulmonary trunk, where Machado *et al* made their measurement, must have reflected the vascular resistance of the placenta (which is low) and that of the lower body of the fetus as well.

The original observation by Kitabatake *et al* assumes that acceleration time relates to mean pulmonary pressure and consequently vascular resistance.¹ There are other more complex factors which alter this measurement, including the vascular impedance, capacitance of the vascular bed in the distal circuit, the intrinsic contractile properties of the left and right ventricles³ and the preload conditions of the two ventricles in the fetus. These factors may be quite different in the fetus and adult, and Machado *et al*'s inference that the acceleration time reflects higher pulmonary vascular resistance seems to be too simple.

It is well known that pulmonary vascular resistance in the fetus is high although pulmonary and aortic pressures are similar. Machado *et al* provide evidence only that there is a slight difference in acceleration times, with that of the pulmonary trunk measurement being shorter, without examining the factors affecting acceleration time which appear to

be complex and affected by many physiological variables.

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References

- 1 Kitabatake A, Inoue M, Asao M, *et al*. Non-invasive evaluation of pulmonary hypertension by pulsed Doppler technique. *Circulation* 1983;68:302-9.
- 2 Rudolph AM. Organization and control of the fetal circulation. In: Jones CT, Nathanielsz PW, eds. *The physiological development of the fetus and newborn*. London: Academic Press, 1985:343-53.
- 3 Reller MD, Morton MJ, Reid DL, Thornburg KL. Fetal lamb ventricles respond differently to filling and arterial pressures and to in utero ventilation. *Pediatr Res* 1987;22:621-6.

This letter was shown to the authors, who reply as follows:

Sir,

Dr Silverman seems to have misunderstood the intention of our paper. Apparently he believes that we are saying something about the lung fields. But in the fetus the pressure in the main pulmonary artery reflects the cumulative resistance of the lung fields, the lower body of the fetus, and the placenta.

We found that the acceleration time in the pulmonary artery in the early human fetus was different from that in the aorta. Based on evidence from adults *and children* we felt this indicated a difference between the pressures in the two great arteries. We agree with Dr Silverman that alternative indices of cardiovascular function may account for our findings. Those he quotes are not measurable in the intact human fetus. Our interpretation is supported by further work by us that shows that later in pregnancy the acceleration times in both great arteries become equal; this is consistent with the fall in placental resistance as gestation advances. Conversely, when placental resistance is abnormally high the acceleration time in the pulmonary artery is abnormally short in late pregnancy.

He also makes the classic error of thinking that indices derived from the fetal animal model can in every respect be extrapolated to the intact human fetus. Recent research from many workers who looked at different aspects of the fetal circulation shows that this is not so.¹⁻⁶ His statement that "it is well known that aortic and pulmonary pressures are equal in the fetus" was reported only in the fetal lamb. The gestational age at which this relation was found is not clear nor is it known how gestation in sheep relates to human gestation in terms of fetal development.

Much work needs to be done in the study of the haemodynamic function of the human fetus. But we must keep an open mind on the findings and not jettison results that do not fit with preconceived ideas that are derived from work in a different species with quite evident developmental differences.

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References

- 1 Kenny JF, Plappert T, Doubilet P, *et al.* Changes in intracardiac blood flow velocities and right and left ventricular stroke volumes with gestational age in the normal human fetus: a prospective Doppler echocardiographic study. *Circulation* 1986;74:1208-16.
- 2 De Smedt MCH, Visser GHA, Meijboek EJ. Fetal cardiac output estimated by Doppler echocardiography during mid- and late gestation. *Am J Cardiol* 1987;60:338-42.
- 3 Allan LD, Chita SK, Al-Ghazali W, Crawford DC, Tynan M. Doppler echocardiographic evaluation of the normal human fetal heart. *Br Heart J* 1987; 57:528-33.
- 4 Kenny JF, Plappert T, Doubilet P, Saltzman DH, St. John Sutton M. Effects of heart rate on ventricular size, stroke volume and output in the normal human fetus: a prospective echocardiographic study. *Circulation* 1987;76:52-8.
- 5 Lingman G, Marsal K. Circulatory effects of fetal cardiac arrhythmias. *Pediatr Cardiol* 1986;7:67-74.
- 6 Tonge HM, Wladimiroff JW, Noordam MJ, Stewart PA. Fetal cardiac arrhythmias and their effect on volume blood flow in descending aorta of the human fetus. *JCU* 1986;14:607-12.

Notices

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 has been changed to 24 June 1988 and is not as indicated in an earlier notice.

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.

European-American cardiology in London

A combined course organised by the American College of Cardiology, British Cardiac Society, and European Society of Cardiology will be held in

London on 1 to 3 June 1988. Inquiries from those residing in Europe to European Cardiology Congress Organization, European Society of Cardiology, Rue Juste Olivier 22, CH-1260 Nyon, Switzerland; from those residing in North America to Registration Secretary, Extramural Programs Department, American College of Cardiology, 9111 Old Georgetown Road, Bethesda, MD 20814, USA.

International Union of Angiology

The 15th World Congress of the International Union of Angiology will take place in Rome from 17 to 22 September 1989. Inquiries to Professor Antonio Strano, Via di Vigna Stelluti 40, 00191 Rome, Italy.