

## LETTERS TO THE EDITOR

- *The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.*
- *All letters must be typed with double spacing and signed by all authors.*
- *No letter should be more than 600 words.*
- *In general, no letter should contain more than six references (also typed with double spacing).*

SIR,—While it is encouraging to note the advances that are being made in the non-invasive definition of the heart and great vessels, it is a cause of anxiety if this leads to surgical correction of trivial lesions that are less life-threatening and morbid than the treatment.

In their article Houston *et al* did not advocate closure of the silent ductus but they did raise the question.<sup>1</sup> To support this attitude they referred to papers published before antibiotics were available and when sepsis generally, and dental sepsis in particular, was rife. In 1991 the incidence of infective endocarditis on a ductus must have been low and similarly the morbidity of the condition—yet all clinically detectable ducts are closed.

From 1978 to 1987 about 500 cases per year were treated by “closed” surgery, and now others are treated percutaneously. Some will have other cardiac defects, some will be haemodynamically compromised but many will be symptom free with small shunts. For a long time, I have believed that treatment of patients in the latter group is unwarranted but the response has always been “Well if you don’t do it, someone else will!”

Perhaps the time has come for a national survey to discover the risk to life and health of the untreated small ductus, if only, initially, from the history and presentation of those who survive the neonatal period.

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<sup>1</sup> Houston AB, Granapagasam JP, Lim MK, Daig WB, Coleman EN. Doppler ultrasound and the silent ductus arteriosus. *Br Heart J* 1991;65:97–99.

### Doppler ultrasound and the silent ductus arteriosus

SIR,—Houston *et al* described 21 cases of “silent” patent ductus arteriosus (PDA) that were apparent only on echocardiogram by colour flow mapping and pulsed Doppler.<sup>1</sup> Two of the twenty one patients in their group had previously undergone surgical ligation of a PDA and residual ductal

flow was seen on colour flow mapping and pulsed Doppler studies. Houston *et al* raised the question, “do patients with a silent ductus arteriosus require prophylaxis against infective endocarditis?” They did not recommend endocarditis prophylaxis for the silent ductus. We offer further echocardiographic evidence against using endocarditis prophylaxis for the silent ductus.

We evaluated nine patients aged 3–21 years who had an echocardiogram for various unrelated indications, and were found incidentally to have clinically unsuspected PDAs. Six of the nine patients did not even have a murmur. We used echocardiography to determine whether or not there was a haemodynamic difference between the clinically unsuspected or “silent” PDA and the small, clinically appreciable PDA. We used eight age matched comparison patients with small PDAs. Our criteria for the “silent” PDA group were the same as the criteria used by Houston *et al*.

All patients with clinically unsuspected PDAs had normal M mode dimensions, with the PDA only being visualised by colour flow mapping and confirmed by pulsed Doppler, which demonstrated the characteristic Doppler signal showing continuous pulmonary flow with a relatively high velocity in diastole. No patient with a “silent” PDA had diastolic reversal in either the thoracic or descending abdominal aorta, unlike the patients with known clinically audible, small PDAs who also had normal M mode dimensions on the echocardiogram. In these patients, however, the PDA was visualized on cross sectional imaging, as well as by colour flow mapping and pulsed Doppler. All eight patients had diastolic flow reversal in the thoracic aorta, and 4/8 had diastolic flow reversal in the descending abdominal aorta. Our initial clinical experience indicates that diastolic flow reversal in the thoracic aorta distinguished a “silent” PDA from a clinically appreciable, small PDA.

The management of the “silent” PDA is a dilemma for the paediatric cardiologist. There is currently no published evidence that haemodynamically trivial, “silent” PDAs present a risk for endocarditis. In fact, our echocardiographic evidence supports the fact that there is a haemodynamic difference between the “silent” PDAs and clinically appreciable PDAs. We suspect that the amount of flow through the ductus is so small that often it not only does not give rise to a murmur but it also probably does not produce enough turbulence to form a jet lesion and thus does not place the patient at a risk for endocarditis.

We support the recommendation made by Houston *et al* that these patients with “silent” PDAs be placed in a registry and followed prospectively for the development of endocarditis.

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<sup>1</sup> Houston AB, Granapagasam JP, Lim MK, Daig WB, Coleman EN. Doppler ultrasound and the silent ductus arteriosus. *Br Heart J* 1991;65:97–99.

## BRITISH CARDIAC SOCIETY NEWSLETTER

### More on Senior Registrars and Registrars

The January newsletter gave stop press news that we have secured approval from the Joint Planning Advisory Committee (JPAC) for an increase in the number of senior registrars in adult cardiology and paediatric cardiology from 60 to 80.5 whole time equivalents. Paediatric cardiology is likely to require only one or two of these posts at the present time, but we must retain a degree of flexibility that will permit some adjustments between the two branches of the specialty. This is particularly important for paediatric cardiology because the overall numbers are small, and needs for trainees vary markedly from time to time depending on the number of retirements that are expected. In large specialties the variations from year to year are minor in relative terms.

We welcome this possibility for expansion of the senior registrar grade. We are entering a period when consultant posts would be difficult to fill if an expansion did not occur. But we must also recognise that we are also entering a period of uncertainty: sadly we may see the need for redeployment, and the Trusts may adopt policies that will confound the planners. We would be unwise if we were to implement at once all of the potential new expansion without continued careful monitoring and fine tuning. In any case it would be impossible to do so. Many difficult steps have to be taken between JPAC approval for an increase in numbers and filling new posts. First cardiac centres have to make a decision that new posts are desirable, then local approval and local funding must be obtained. In these difficult times funding may be a formidable hurdle. JPAC does not carry new money with its approval for expansion. Moreover, the specialty does not have full freedom to decide where new posts will be created. Except for small specialties, guidelines exist for the distribution between Regions of senior registrar posts: these must be followed unless good reasons can be shown for not adhering to them. Cardiologists from centres that could appropriately train an additional senior registrar—and would like to do so—should contact the Specialist Advisory Committee directly or through ourselves so that approaches can be planned and coordinated.

We are already facing major reductions in the number of career registrars. One welcome knock-on effect of an increase in senior registrar numbers will be a commensurate increase in the number of registrars. At present we have a serious bottle neck at this level because numbers have not been controlled effectively. We learn for example that a recent advertisement for a senior registrar appointment in Cambridge attracted applications from 39 registrars. Almost all seemed fully qualified for the post. Most of the applicants had considerable catheter experience; six already had an MD thesis;