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).

Management of patients with Björk-Shiley prosthetic valves

SIR,—In his editorial¹ Mr Tom Treasure correctly describes the development of the Björk-Shiley prosthesis—from the Delrin disc valve to the spherical disc valve, to the convexo-concave valve with an opening angle of 60° (CC60), to the convexo-concave valve with an opening angle of 70° (CC70), and finally to the Monostrut valve that is manufactured without any welding procedures. He also mentions that the risk for outflow strut fracture seems limited to the CC60 and CC70 models, with the highest risk in some CC70 valves. He claims that the operative risk for elective replacement of these valves might be as high as 16% for mitral valves and 9% for aortic valves and argues that prophylactic replacement of these valves (making no distinction between CC60 and CC70 valves) “for fear alone is unjustifiable”.

Certainly, replacement of these valves should not be done “for fear alone”, but should be done after a careful assessment of the risk for strut fracture and the risk of a prophylactic reoperation. Unfortunately, I cannot agree with Mr Treasure's conclusions, but believe that some patients with CC70 valves would benefit from elective replacement of their valves on the following grounds.

In 1986 we published the long-term results of valve replacement with the Björk-Shiley prosthesis² and in 1989 we presented an updated follow-up on those with CC60 and CC70 valves.³ Our original report included 3334 Björk-Shiley valves (of which 884 were CC60 and 577 were CC70 valves) followed for 17 511 years (mean follow up 6.3 years). Our updated follow-up included 8299 patient-years for CC60 and CC70 valves. The follow up was 99% and the necropsy rate was 75%. These are the largest series of Björk-Shiley valves followed for an extended time, and unfortunately also the largest single-institution experience with Björk-Shiley valve fractures. Neither of these reports were referenced by Mr Treasure.¹ In our original report we found a constant, but similarly low risk of strut fracture in the CC60 valves (constant hazard function). The risk in CC70 valves was considerably higher, although it seemed that this high risk decreased over the years (decreasing hazard function). However, in our more recent report it was clear that this apparent decrease in the risk of strut fracture was an artefact and that the risk among the

large (that is, 29–33 mm), early CC70 valves (so-called group I valves⁴) remained almost constant over a seven year follow up. Furthermore, and contrary to Mr Treasure's comments, this was not limited to mitral valves. The seven year actuarial incidence of strut fracture among large aortic CC70 valves was 15.3% and that for large mitral CC70 valves was 11.3%. This corresponds to a yearly incidence of strut fracture of 1.8–2.0%. These findings have recently been corroborated in an international multicentre study including 20% of all CC70 valves implanted worldwide.⁵ In this study it was found that a 29–33 mm diameter was the only risk factor for strut fracture in CC70 valves. There was no difference between aortic and mitral valves.

Also we do not believe that prophylactic valve replacement entails such a high operative risk as proposed by Mr Treasure. As he correctly points out, the re-replacements in the United Kingdom's Cardiac Surgical Register includes all patients undergoing valve re-replacement, irrespective of the cause, and most of these patients probably had severe symptoms. Prophylactic re-replacement in highly selected patients would carry a much lower risk. In our own, albeit small, experience of prophylactic or incidental replacement 30 day mortality was one in 27 patients (3.7%). This was a 72 year old woman who underwent incidental replacement of her large CC70 mitral prosthesis together with aortic valve replacement for symptomatic aortic stenosis. Also two fairly large series of prophylactic replacements of aortic Braunwald-Cutter prostheses have been reported, with early mortality less than 3%. These procedures were performed when methods for intraoperative myocardial protection were much less developed than they are today.^{6,7}

I believe that there is strong statistical support for prophylactic replacement of some of the large (29–33 mm) CC70 valves, whether in the aortic or the mitral position.

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- 1 Treasure T. Management of patients with Björk-Shiley prosthetic valves. *Br Heart J* 1991;66:333–4.
- 2 Lindblom D, Björk VO, Semb BKH. Mechanical failure of the Björk-Shiley valve. Incidence, clinical presentation, and management. *J Thorac Cardiovasc Surg* 1986;92:894–907.
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- 7 Wideman FE, Blackstone EH, Kirklin JW, Karp RB, Kouchoukos NT, Pacifico AD. Hospital mortality of re-replacement of the aortic valve incremental risk factors. *J Thorac Cardiovasc Surg* 1981;82:692–8.

This letter was shown to the author, who replies as follows:

SIR,—I am grateful to Dr Lindblom for his response which may help us towards a

rational approach to the question of elective reoperation to replace Björk-Shiley heart valves that are known to be at risk of strut fracture.

He draws to our attention his own publications that I did not cite directly in the editorial. The data to which he refers, from Björk and colleagues, were included amongst the six clinical series that comprised the data base of the multi-institutional advisory panel.¹ I apologise for any offence this has caused but the other five series in the panel's meta-analysis were not individually cited either. What is perhaps more important is to draw readers' attention to the new data contained in the retrospective cohort study of 2303 patients with convexo-concave (CC) valves in The Netherlands.² For large mitral (≥ 29 mm) 70°CC valves the risk of strut fracture is over 2% per annum. Valves of 27 mm or less in the 60°CC pattern were not liable to fracture. There were some discrepancies between the Dutch and Swedish results with the mitral prostheses being much more at risk than the aortic ones in Holland, but the authors warned that with a 25% necropsy rate there must be an overall underestimate of strut fracture. In my article I seemed to emphasise the mitral valve, but only because fewer patients have aortic prostheses ≥ 29 mm. In fact they represent 5.3% of all Björk-Shiley aortic valve replacements in the UK Heart Valve Registry.³

I quoted data from the United Kingdom Cardiac Surgery Register for mortality with reoperation and Dr Lindblom questions this, reiterating my own caveat, that emergency reoperation and endocarditis were included and this may exaggerate the true risk for selected, elective re-replacement. However, in challenging the use of these data he illustrates the two reasons why I believe we should quote registry data whenever possible for discussions of this type, rather than quoting selected series. First he tells us that his group have lost only one out of 27 cases with a risk of under 4%. The 95% confidence interval is 0.1% to 19% for mortality and even if we follow Kirklin and take the more lenient 70% confidence interval the upper limit is 13%. We cannot be sure that his excellent results represent a true low risk rather than a lucky run. He then quotes the University of Alabama experience in replacement of Braunwald-Cutter aortic valves in which the mortality was 1/65 in a series of operations performed by Kirklin, Karp, Kouchoukos, and Pacifico.⁴ It is possible that not all groups will be able to emulate these results.

The operative risk that patients face is that in the centre to which they are referred and if that is estimated, on the basis of registry or audit data, to be less than the risk of death from strut fracture then prophylactic replacement may be considered. Some patients may find intolerable the knowledge that they have within them a valve that may fracture at any time, and in such patients an operation would be justified. That is a very real issue but must be distinguished from a cold analysis of numerical risk.

As far as numerical risk is concerned it seems justifiable to offer elective operation to selected groups as both Lindblom and van der Graaf⁵ have suggested. Those most at risk are patients with large (≥ 29) 70°CC valves. Those who have most to gain are the younger patients with good cardiac status for whom median survival after valve replacement is in excess of 15 years. For them risk of an elective re-replacement in experienced hands is sig-

nificantly less than the cumulative risk of strut fracture during their expected lifetime.

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- 1 Hiratzka LF, Kouchoukos NT, Grunkemeier GL, Miller DC, Scully HE, Wechsler AS. Outlet strut fracture of the Björk-Shiley 60° convexo-concave valve: current information and recommendations for patient care. *J Am Coll Cardiol* 1988;11:1130-7.
- 2 van der Graaf Y, de Waard F, van Herwerden LA, Defauw J. Risk of strut fracture of Björk-Shiley valves. *Lancet* 1992;339:257-61.
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Abnormal right heart filling after cardiac surgery

SIR,—Dr Wranne and colleagues demonstrated in figure 5 of their interesting study that the lateral aspects of the tricuspid annulus showed a more pronounced motion loss after cardiac surgery than those of the mitral annulus.¹ As one of the possible explanations they suggested that the left ventricle was better preserved during surgery than the right ventricle. This theory is confirmed by an experimental study of the tissue electrolyte content in the right and left ventricular myocardium after normothermic open heart surgery in dogs.² Cardiac arrest had been induced (a) by clamping the ascending aorta, (b) by aortic clamping with additional injection of a cardioplegic solution, (c) and by electrically induced fibrillation (with preservation of the coronary circulation). Tissue electrolyte content was determined before extracorporeal circulation was started, as well as after an hour of recovery from a cardiac arrest of 30 or 45 minutes. In all these forms of cardiac arrest, tissue water had increased and potassium and magnesium decreased. These changes were more pronounced in the myocardium of the right ventricle in all experimental groups. A decrease in potassium and magnesium content in tissue is an indicator of cellular injury.³⁻⁵ In the study in dogs the loss of these electrolytes was more pronounced in the myocardium of dogs with low cardiac output than in animals with adequate circulation after cardiac arrest. Because the dogs did not have genuine cardiac surgery cardiac arrest was relatively short and hypothermy was not used. Hence we do not believe that the observed differences between the ventricles were predominantly caused by a mechanical impediment, more pronounced exposure of the right ventricle to room temperature, or heat radiated from the operating room lights, as suggested by Wranne *et al.* We attribute this phenomenon to a proposed difference in the susceptibility of the right and left ventricular myocardium to systemic disturbances, as it has been described for various diseases, such as hyperosmolar coma or liver failure.^{3,6} Histologically, ultrastructurally, and biochemically, the right ventricular myocardium differs from that of the left ventricle.^{4,6,7} According to Doerr the different susceptibility of the ventricles to disease can be explained by phylogenesis: the right ventricle belongs mainly to the priscomyocardium and is phylogenetically older than the left ventricular neomyocardium.⁶ Perhaps this so-called "theory of pathocliosis" also explains the differences between the right and

left ventricular function seen after cardiac surgery and described by Wranne *et al.*

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Working party report on cardiac rehabilitation

SIR,—I was most interested to read the report from the working party on coronary rehabilitation and would like to congratulate them on their obvious hard work.¹

Because coronary rehabilitation has not been a high technology subject it has lapsed into a cinderella type of service with a few enthusiastic doctors and many more enthusiastic nurses, physiotherapists, and occupational therapists soldiering on in isolation. It is for this reason that research has not been very forthcoming because individual units find that they do not have enough patients to produce meaningful controlled trials: for example, the trial by a Glasgow team in 1991 in the end had 12 patients in the treatment group and 10 in the control.²

Up to now it is the paper from O'Connor *et al* in 1989, which provided an overview of 22 small trials, that has allowed us to believe that coronary rehabilitation had a part to play in the treatment of acute myocardial infarction.³

Your readers may be interested to know that here in the North-West we have had the Coronary Rehabilitation Development Organisation (CREDO) for about two years now. It is a loose knit confederation of just such enthusiasts as I have described who meet quarterly to exchange ideas and support others who are in the process of setting up a rehabilitation programme.

Three things have become apparent. Firstly it requires a dedicated nurse/physiotherapist/occupational therapist around whom the whole programme pivots; secondly, it does not need much money to set up a simple scheme; and thirdly, and probably the most important, any scheme to be successful needs the wholehearted support of the consultant in charge of cardiology for that district—whether cardiologist or general physician with an interest in cardiology.

A meeting is to be held on 26 September 1992 in Oxford to try to organise a national association that may well then allow us to produce some proper scientific evidence to persuade those who control the budgets that rehabilitation is a worthwhile and cost-

effective part of the treatment of myocardial infarction.

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BOOK REVIEWS

Heart disease: A textbook of cardiovascular medicine. 4th ed. Edited by Eugene Braunwald. (Pp 1874; \$119.) Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W B Saunders Company, 1992. ISBN 0-7216-3097-9.

When the original edition of this comprehensive textbook appeared, I was given the opportunity to review it in 1980. It was the first of a new generation of such works, and set the standards by which others might be judged. Not only did other important similar works respond by improving their quality but a number of other textbooks have appeared, mainly in the United States though with one British based offering.

As with previous editions, Braunwald is an author or co-author of many of the chapters as well as having orchestrated the whole. He has taken considerable trouble to ensure that what appears is up to date even at the cost of shedding some earlier references from some chapters that may be of historical relevance. But as his purpose is to present contemporary evidence, this has enabled him to limit the size to some extent. Nevertheless, there are still 1874 pages of text and illustrations, with ample and appropriate references.

The whole of the subject is covered extensively and if some miss their favourite topics, that will be highly exceptional. For a description of pathological processes generally one will have to turn elsewhere and this is probably appropriate in a book aimed at providing clinically relevant information, yet readers should not forget the potential importance of such knowledge.

There have been modest changes in authorship once again between the third and fourth editions, and Braunwald's system of having all the chapters externally reviewed ensures a high standard throughout.

The most striking feature of the present edition is the lavish use of colour. Generally this is helpful and indeed to have the chapter numbers coloured red discriminates them from the pages above and makes for the easier finding of what one needs from an index that did not fail several random tests. Those interested in electrocardiography will, however, question the use of red for the tracings and a dark but absolutely crisp background for the grids (the latter is an excellent feature); surely it would have been easier for most if the conventions had been observed and the tracings had been dark and the background coloured as you see in clinical tracings. In other respects I accept, and on the whole welcome, the use of colour, which