LETTERS TO THE EDITOR

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Extent of Anoxic Brain Damage

To the Editor:

The estimation of the extent of anoxic brain damage to accurately predict neurologic outcome of individual patients after resuscitation from cardiac arrest has continued to pose problems to clinicians caring for these patients. Various methods, including electroencephalography and systematic neurologic examinations have been evaluated, but with varying degrees of reliability. It is for these reasons that we believe the report of Vaagenes et al.,1 is so important.

We found a similar relationship between cerebrospinal fluid creatine kinase BB isoenzyme activity (CKBB) and neurologic outcome in 20 patients resuscitated from out-of-hospital cardiac arrest; higher values were associated with more severe neurologic damage and worse outcomes.2 In contrast to their observations, however, we found that the serum CKBB was present in a high percentage of cases upon admission, but that it persisted or reappeared more than 6 hours after the arrest only in those with poor clinical outcomes: vegetative states and no neurologic recovery before death.

However, because of the many known extra-cerebral sources of serum CKBB, we believe that cerebrospinal fluid, rather than serum, CKBB to be more reliable in predicting neurologic outcomes after anoxic-ischemic brain insults.

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References


Echocardiographic Documentation of Vegetative Lesions

To the Editor:

I read with interest the paper by Stewart et al.,1 on the echocardiographic documentation of vegetative lesions in infective endocarditis. However, quoting Buchbinder and Roberts,2 the authors maintain, “Previous autopsy studies have reported vegetative valvular lesions in 53% of patients who died of active infective endocarditis.” Later the authors state, “Only 54% of patients in the present series had echocardiographically detectable vegetations despite firm clinical and laboratory evidence of infective endocarditis. . . . The fact that autopsy series reveal a similar occurrence of vegetations suggests that either two forms of endocarditis exist (one with and one without vegetations). . . .”

Buchbinder and Roberts3 only write that “in the present study of forty-five patients, 53 per cent had anatomically normal valves before the infection.” Furthermore, from the analysis of table I of the same work, we can deduce that all 45 patients of the series presented valvular vegetations.

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References


LV Function after Repair of TOF

To the Editor:

We read with great interest the article by Borow and colleagues.1 The authors have demonstrated left ventricular dysfunction in older patients after repair of the tetralogy of Fallot (TOF) and have raised the possibility that early definitive repair of the lesion may help preserve postoperative left ventricular function. A recent study in older patients several years after repair of TOF showed abnormalities in right ventricular performance during exercise, but none in the left ventricle.2 We wish to add that our histological studies3,4 also emphasize the possible importance of early repair in TOF. We performed a quantitative microscopic study by quantitating the right and left ventricular interstitial tissue space vs myocardial fiber space in four groups of autopsy hearts: five normal hearts, five hearts from infants less than 2 years of age with TOF, 10 hearts from children 2½–18 years old with TOF and five hearts from adult patients (more than 20 years of age) with TOF. We found that up to 2 years of age, both right and left ventricles of patients with TOF are similar to normals, as far as the ratio of interstitial tissue space vs myocardial fiber space is concerned. After 2 years of age, however, the proportion of interstitial tissue space increased dramatically, and, after 13 years, the left ventricle also showed marked increase of interstitial tissue space. Although the importance of such findings and their correlations to the two studies1,2 are not clear, the increase of interstitial tissue space in right ventricle and, subsequently in left ventricle of patients with TOF beyond 2 years of age can explain the postoperative myocardial function abnormalities that are most probably secondary to persistent hypoxia. As proposed by Borow et al.,1 we also raise the question of early (before 2 years of age) definitive repair of TOF to preserve myocardial function. Perhaps further studies in this field would finally show us that the slightly increased risk of definitive repair of TOF in infancy is worth having a better myocardium for the rest of the patient’s life.

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