bifascicular disease relative to the search for improved criteria for the prediction of impending trifascicular block, prior to the occurrence of prolonged episodes of ventricular asystole with attendant life-threatening Stokes-Adams attacks. In our large study population of 50 bifascicular disease patients with Mobitz II or transient complete heart block (CHB), it was possible to determine from conducted sinus beats a critical degree of H-Q widening or breakpoint (> 65 msec; fig. 1) serving as an important harbinger for probable imminent symptomatic CHB. We agree that this new knowledge must be judged with appropriate caution concerning its extension to revised indications for prophylactic permanent pacemaker implantation, since present understanding of the development of CHB in bifascicular disease is still incomplete and particularly because Drs. Gupta, Lichstein and Chadda have seriously misinterpreted our findings and recommendations.

A careful reading of our paper clearly states that we neither recommend pacemaker insertion merely for the presence of “bifascicular block accompanied by first degree heart block” nor simply for “bifascicular block of three or more years’ duration.” Instead, we have suggested that asymptomatic patients with these conditions undergo ambulatory ECG monitoring and His bundle electromyography (HBE). Only if Mobitz II or transient CHB, or an H-Q > 65 msec is thereby documented are such individuals considered for pacemakers. In the rare circumstance of the unavailability of ambulatory ECG monitoring and HBE as well as inadequate serial follow-up, due to the high likelihood of H-Q > 65 msec in bifascicular block with the combination of both first degree heart block and the bifascicular block present for more than three years, would a pacemaker be considered. Obviously, we have not implemented a pacemaker in this theoretical circumstance since these patients are evaluated by ambulatory ECG monitoring and HBE at our institution.

To clarify our recommended indications for pacemakers in bifascicular block, one of five major criteria is now considered sufficient: 1) otherwise unexplained syncpe, 2) intermittent CHB, 3) Mobitz II block, 4) alternating bundle branch block, and 5) prolonged H-Q > 65 msec. Concerning the two minor criteria: 1) associated first degree heart block and 2) bifascicular block greater than three years’ duration; neither is sufficient evidence alone for pacemaker implantation. However, in bifascicular block, since a prolonged P-R interval substantially increases the probability of prolonged H-Q interval from 42 to 72% of patients’ and the risk of occurrence of complete heart block is 6% per year, it is suggested that the presence of either minor criterion requires further evaluation with HBE and ambulatory ECG monitoring. If this evaluation procedure uncovers a major criterion, only then is a pacemaker considered in, as yet, an asymptomatic individual with bifascicular block.

Our rationale for selecting an H-Q prolongation of 65 msec, above which in bifascicular disease there is a high incidence of developing CHB in the near future, is explained by our finding of such critical H-Q widening in conducted sinus beats in 48 of 50 bifascicular block patients with concomitant intermittent trifascicular disease (fig. 1). From these data, we infer that this degree of H-Q prolongation in bifascicular disease is the usual prerequisite for the onset of CHB. On the other hand, we have emphasized that the extent of H-Q breakpoint leading to CHB is disparate among patients with bifascicular disease, as well as the variability of time prior to the development of CHB in such individuals with critical H-Q prolongation. We agree that further prospective analysis of a large number of bifascicular disease patients may provide more precise information regarding these crucial questions. In this regard, three of our nine asymptomatic bifascicular disease patients with an initial H-Q of 55 to 67 msec developed symptomatic CHB with H-Q prolongation of 67 to 80 msec within an average of 12 months. Therefore, in view of our adverse experience with prolonged cardiac asystole after the occurrence of trifascicular block and the accompanying risk of sudden death, accidents with physical disability, and compromised cerebral function from such ventricular inactivity, we feel obligated to consider H-Q > 65 msec as auguring imminent symptomatic CHB and thus indication for pacemaker insertion.

Concerning the previous report of 16 bifascicular disease patients with intermittent trifascicular disease by Gupta and associates, we agree their work is consonant with our observations in 50 such patients that the majority of these individuals have prolonged H-Q intervals during periods of antegrade conduction. Although these workers did not extend their descriptive observations to precise clinical recommendations for pacemakers, it is interesting that they are in general concurrence with our postulation that bifascicular disease patients with prolonged H-V intervals possess a higher risk of experiencing CHB. Finally, our manuscript was not intended to present an exhaustive review of pharmacologic effects on the conduction system in bifascicular block and we acknowledge that the additional references presented by Dr. Gupta and colleagues are indeed pertinent and worthy of consideration.

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References

Swan-Ganz Catheter Repair
To the Editor:

Hemodynamic monitoring of critically ill patients with the Swan-Ganz catheter has made an important contribution to patient management and clinical research in the past few years. The use of this catheter is now widespread in many institutions and in our own Coronary Care Unit we commonly employ the triple lumen thermal dilution catheter. On rare occasions while changing dressings near its proximal end one of the lumens has been inadvertently cut.
A method has been devised to repair the catheter in such circumstances rather than replace it. The severed catheter is cleaned with appropriate sterilizing agents and a #19 B-D needle inserted into either the right atrial or the pulmonary arterial line. The lumen supplying the balloon tip requires a #20 B-D needle.

Grinding of the bevel of the needle and tapering of its stump facilitates passage into the lumen of the catheter and reduces the possibility of embolizing plastic chips created during insertion. This adaptation is stable, simple to perform, and withstands substantial pressure without leakage. A supply of needles prepared as indicated may be gas sterilized and kept available for use when needed.

**Echo Technique in Open-Chested Dogs**

To the Editor:

In their paper, "Correlation between echocardiographically demonstrated segmental dyskinesis and regional myocardial perfusion," which appeared in Circulation 52: 1097, 1975, Kerber et al. refer to a paper by Kraunz and Kennedy for a description of the characteristic normal posterior wall motion in human subjects. These authors describe the major movement of the posterior wall as anterior during ejection. They also point out that there is a small initial movement posteriorly (B-C), during isovolumic contraction. In figure 1A of Kerber's paper, the B-C excursion cannot be considered small. In actual fact it represents 53% of the C-D excursion. In addition, if one looks at the relationship between the onset of septal and posterior wall movement, it is apparent that the septal C point corresponds to the posterior wall B point. Furthermore, the septal posterior excursion is greater than the posterior wall anterior excursion, and in addition at what is labeled the D point, the posterior wall has not completed its anterior excursion, even though the septal posterior movement has ceased. The point that is marked E appears well into diastole and coincides with the P wave of the electrocardiogram. This incongruity of noninfarcted septal and posterior wall movement is at variance with the reported normal wall movement in humans. Myerowitz et al. and Stefan and Bing describe two techniques for studying posterior wall movement in dogs. The posterior wall movement pattern they describe correlates with that outlined by Kraunz and Kennedy.

We have looked at the suitability of open-chested dogs for assessing posterior wall movement. We found that it was impossible to "fix" the transducer to the anterior heart wall. Even though several clamps were used, there was always some movement of the transducer. Figure 1A shows the transducer (T) on the myocardium. Using the R wave of the electrocardiogram as a reference point, the major movement of the posterior wall is away from transducer, that is posteriorly, and it sustains this position throughout systole. In IC, the transducer does not make direct contact with the myocardium, and ultrasonic coupling is established by means of a saline bath, which lies on the myocardium. The major movement of the posterior wall is anterior. Thus, the method used to couple the transducer to the open-chested canine heart can result in very different patterns of wall motion. Figure 1A is an example of transducer movement-induced artificial posterior wall dyskinesis. It is also interesting to note that the septal motion varies with the position of the transducer. In A its major movement is posterior following the R of the QRS, and in C its major movement is anterior.

In reviewing the description of the model given in a previous paper by these authors, no attention has been paid to the problem of transducer artifact-induced posterior wall movement. It is important to address oneself to this problem, particularly as one of the conclusions in their present paper suggests that segmental dyskinesis has occurred in normally perfused myocardium immediately adjacent to areas of ischemia. Is it possible that the segmental dyskinesis seen in this normal area is due to anterior motion of the transducer induced by contraction of the subjacent...
Letter: Swan-Ganz catheter repair.
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