Heart Block in the Aged. Is the Patient Too Old to Be Permanently Paced?

If arteriosclerotic heart disease were the most common cause of conduction defects requiring long-term management with pacemakers, one would expect this complication to be manifested with greater frequency in the fourth through sixth decades when other manifestations of this disease are so frequently seen. The average age of 73 years in our own series of patients and similar age groups in other reports indicates on the contrary that acquired heart block is generally a disease of older people.\textsuperscript{1-5} Additionally, those dealing in any volume with conduction system diseases have been impressed with the infrequency with which patients developing heart block give a history of angina, previous myocardial infarction, congestive heart failure and other hallmarks of coronary artery disease. This correlates with the observations of Lev and Lenegre\textsuperscript{6-8} who have shown that idiopathic focal and diffuse fibrosis is a more common cause of anatomic disturbance and impaired function of the conduction system.

Since one can therefore expect rhythm and conduction disturbances with increasing frequency in the aged, the question arises: When is a person too old to be paced? This question is likely to be asked by those who, impressed with the patient's infirmity, are unaware of its frequent association with an underlying conduction defect particularly when symptoms are less spectacular than those characteristic of Stokes-Adams attacks. Such a patient's candidacy for permanent pacing is further doubted by those who, less familiar with current techniques of establishing this mode of therapy, continue to feel that it represents "heroic treatment."

Our own experience in over 80 patients, 63 of whom have been followed from 4.3 to 37 months (mean 15 months), is one of the oldest reported in the literature. These patients, whose average age was 73 years, presented with varying degrees of heart block usually complicated by Stokes-Adams attacks and less frequently by congestive failure, azotemia or cerebral ischemia. Permanent pacing was established utilizing a transvenous endocardial system. Detailed analysis of 68 of these cases, 63 of whom were discharged from the hospital and 57 of whom were observed to have remained significantly improved at the end of the follow-up period, does not support the view that these elderly patients are at best only marginal candidates for permanent pacing. On the contrary, they are prime candidates if one recognizes, as he must in all patients so treated, that they may still succumb to the underlying cardiopathy if not to other extracardiac infirmities which must be anticipated in the aged. Even in the absence of any grossly demonstrable disease, their longevity is abbreviated.

In these older people who are often surprisingly productive and occupy an important position in the family structure, the rigors of any surgical procedure must be weighed against the fragility of age. With the advent of the transvenous endocardial pacing system, this has ceased to be a significant problem since the procedure of Furman et al\textsuperscript{9} obviates the necessity of thoracotomy and can be performed under local anesthesia without even preoperative sedation or analgesia. This procedure utilizes a subclavicular incision through which the cephalic vein is isolated for transvenous passage of catheter electrodes to the right ventricle and for formation of a pocket in which the pulse generator is secured. Surgical complications are infrequent and minor. The major problem with this form of therapy is loss of catheter position which occurs in approximately 20 percent of cases. While this is high, it is easily remedied and very infrequently recurs after the first three weeks. Complications involving electrode stability, position and integrity are infrequent with the use of the epicardial pacing system. Because it requires thoracotomy, however, it is essentially obsolete in this age group.

While an individual case may be taxing and
marginal, one cannot help, over the course of a larger experience, be impressed with the generally gratifying results with this wonderful group of people, 62 percent of whom were more than 71 years of age in our own series.

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REFERENCES

1 Unpublished data

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Mitral Ballooning—A Possible Mechanism of Mitral Insufficiency in Diseases Associated With Reduced End-Systolic Volume of the Left Ventricle

Angiographically proved mitral ballooning is a common entity as evidenced by the 11 cases included in our report in Chest and by 17 other cases observed since preparation of this manuscript.

In reviewing our series of 22 cases of idiopathic hypertrophic subaortic stenosis (IHSS) and/or nonobstructive left ventricular hypertrophy (LVH) with obliteration of the apex, we were impressed by the frequency of ballooning in these entities (15 of our 22 cases, Fig 1). Though not commented upon by the authors, several reports, including a recent article in this journal, contain angiograms illustrating the presence of ballooning in IHSS.

To explain mitral regurgitation (MR) in IHSS, attention has focused on anomalies of the anterior mitral leaflet. We believe that ballooning of the posterior leaflet past the anterior leaflet is an additional mechanism of MR and is due to the well-documented reduction of left ventricular end systolic volume. We postulate that the reduced end-systolic volume by narrowing the distance between the papillary muscle and the mitral leaflet permit the latter to billow into the left atrium before tensing of the chordae could check its advance.

Grossman et al have ascribed both ballooning and MR to "an abnormal convex deformity during systole along the inferior aspect of the left ventricle.” Their cases probably represent prominent posterior and anterior papillary muscles in patients with nonobstructive LVH (three of their five cases had LVH by electrocardiography) or in patients with primary ballooning and increased contractility (see below).

Myxomatous degeneration of the mitral cusps has been well documented in ballooning and was proved in two of our cases. This type of ballooning should be termed primary as opposed to secondary “myocardial” ballooning resulting from excessive LV contraction. The presence of a click would favor a primary origin, whereas LVH by electrocardiogram would favor a secondary ballooning. In the absence of obvious obstruction, the angiographic distinction between these two forms of ballooning can be difficult because primary ballooning alone can be associated with reduction of the volume encompassed by the left ventricular muscular elements. The easily distensible ballooned cusps act as

Figure 1. Lateral left ventriculogram. IHSS—ballooning of the posterior mitral leaflet (PL) with regurgitation across this leaflet into the left atrium (LA). Arrow points to hypertrophied and elevated posterior papillary muscle.