

The Management of Patients with Premature Ventricular Contractions

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Premature ventricular contractions (PVCs) occur frequently in patients with virtually all types of cardiac disease and in persons without evidence of cardiac disease. Since PVCs and advanced grades of PVCs are known to be predictors for ventricular fibrillation, increased mortality, and sudden death in certain clinical settings, the management of patients with PVCs is frequently based on the clinical setting in which they occur. For the purpose of this talk we will discuss the treatment of PVCs in the following three instances: acute myocardial infarction in the coronary care unit, post-myocardial infarction patients, and persons without any evidence of organic heart disease.

Acute Myocardial Infarction

Almost all patients with acute myocardial infarction have PVCs during the first few days. The frequency of PVCs is greatest in the first hours after infarction and decreases gradually and progressively during the next several hours and days. In acute myocardial infarction, Lown and co-workers¹ described certain types of PVCs that commonly preceded ventricular fibrillation. They applied the term "serious ventricular arrhythmias" to the following types of PVCs: (1) occurrence at a rate of five or more per minute, (2) "R on T" phenomenon, (3) multi-form PVCs, (4) paired PVCs, and (5) ventricular tachycardia. When observed on routine monitoring, these premonitory ventricular arrhyth-

mias were treated with lidocaine, and Lown and associates reported less than a 2% incidence of primary cardiac arrest in 300 patients with acute myocardial infarction.² The detection of serious ventricular arrhythmias from the cardiac monitor followed by suppression with anti-arrhythmic drugs is the approach in many coronary care units today.

There are two problems with this approach. First, the serious ventricular arrhythmias appear to be neither very sensitive nor very specific for the subsequent development of ventricular fibrillation. El-Sherif and associates³ found that only 11 (58%) of 19 patients with an acute myocardial infarction who developed ventricular fibrillation had premonitory arrhythmias. In the 17 patients where ventricular fibrillation was preceded by a single PVC, 10 had early PVCs or "R on T" phenomenon. Of the 430 patients without ventricular fibrillation in this series, 236 (55%) had premonitory ventricular arrhythmias and 194 (45%) did not. A second study⁴ reported similar findings.

The second problem is the adequacy of the detection system for arrhythmias as it is routinely employed in the coronary care unit. We compared the recognition of arrhythmias in 31 patients with acute myocardial infarction by the nurses and by rate-alarm signals in the coronary care unit with a continuous five-day tape recording that was subsequently processed through an automated Hewlett-Packard arrhythmia detection system. The Hewlett-Packard system printed out all arrhythmias for verification and quantification by the investigators.⁵ The observation of conventional monitors by coronary

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care unit personnel in conjunction with rate-alarm systems resulted in an inadequate detection rate of PVCs and serious ventricular arrhythmias compared to the subsequent analysis of the tape recording (Table). In addition, there was a considerable time delay between the initial occurrence of PVCs and serious ventricular arrhythmias on the tape and their recognition by coronary care unit personnel. Arrhythmia recognition in coronary care units has improved and computer systems are available which can be used on line to improve the detection rate for PVCs; however, most coronary care units continue to rely on trained personnel for arrhythmia recognition.

Since there is such a high incidence of PVCs in patients with acute myocardial infarction, the prophylactic administration of antiarrhythmic drugs has been evaluated extensively. Initially quinidine,⁶ procainamide,⁷ lidocaine,⁸ and phenytoin⁹ were reported to reduce significantly PVCs and serious ventricular arrhythmias, without reducing mortality. The patients in these studies were always a good-risk population with a low mortality in the control groups. In addition, when arrhythmias were recognized in the control groups, they were treated. More recently, Lie and co-workers¹⁰ in a double-blind randomized study demonstrated that lidocaine given prophylactically as an initial 100 mg bolus followed by an infusion rate of three mg/min significantly decreased the incidence of primary ventricular fibrillation, but mortality was not effected since all but one patient with ventricular fibrillation was resuscitated. The frequency of side effects at this infusion rate was 15% and was higher in the 60- to 70-year age group than in patients below the age of 60.

Since the PVC predictors for ventricular fibrillation are less reliable than indicated in initial reports, and PVC detection by conventional

monitoring in the coronary care unit is not optimal, there is evidence available to recommend the prophylactic administration of lidocaine to patients during the first 48 to 72 hours of an acute myocardial infarction for the prevention of primary ventricular fibrillation. Patients with evidence of heart block, cardiogenic shock, severe congestive heart failure, and sinus bradycardia (below the rate of 50) probably should be excluded from this recommendation. Since patients above the age of 70 apparently have a low incidence of primary ventricular fibrillation in the coronary care unit and a higher incidence of adverse reactions to lidocaine, they also should probably be excluded from this recommendation.

The dosage of intravenous lidocaine used for prophylaxis is important. When a loading dose of 50 to 100 mg followed by a 2 mg/min infusion rate is used, there is an initial period of six to seven hours when plasma levels are suboptimal.¹¹ In an effort to avoid this dip in plasma levels it is necessary to give about 200 mg of lidocaine in the first 20 minutes.¹² There are several ways to give this loading dose: (1) 100 mg given twice at ten-minute intervals; (2) 50 mg given four times at five-minute intervals; (3) infusion of 20 mg/min for ten minutes; and (4) 100 mg bolus followed by infusion of 6 to 7 mg/min for 15 minutes. I prefer the last method followed by the continuous administration at an infusion rate of 2 to 4 mg/min, preferably 3 mg/min. In patients with shock, congestive heart failure, or hepatocellular disease, the loading dose and infusion rates should be decreased by about 50%. This may also apply to patients over the age of 70. If PVCs occur, the plasma concentration of lidocaine can be raised rapidly with a 25 to 50 mg bolus and an increase in the infusion rate. There is some evidence that the plasma half-life of lidocaine increases by a factor of about two after 24 hours

TABLE
Arrhythmia Recognition in Acute Infarction
(31 Patients)

	CONVENTIONAL MONITOR	AUTOMATED DETECTION SYSTEM	TIME DELAY IN RECOGNITION (HOURS)
Premature Ventricular Contractions	64.5%	100%	18
Serious Ventricular Arrhythmias	16%	93.5%	10
Premature Atrial Contractions	45.2%	96.8%	23
Ventricular Fibrillation, Atrial Fibrillation, Asystole, Complete Heart Block, Junctional Tachycardia	Identical	Identical	—

in patients with acute myocardial infarction, thus the infusion rate may have to be decreased at this time.¹³

Post-Myocardial Infarction

In patients with a previous myocardial infarction many studies have reported an increased mortality and/or an increased frequency of sudden death in patients with PVCs or advanced grades of PVCs.¹⁴⁻¹⁸ Although there is some variability in the reported studies, the increased mortality appears to be true for the presence of PVCs on a routine electrocardiogram as in the Coronary Drug Project,¹⁴ and for an increased frequency of PVCs (>10 per hour or >20 per hour) or advanced grades of PVCs or both during monitoring studies. In contrast to these studies, the work of Moss and co-workers,^{18,19} has indicated that PVCs and advanced grades of PVCs have limited use in predicting subsequent cardiac events and a similar study has been reported by de Soyza and associates.²⁰ It is likely that PVCs and advanced grades of PVCs will be neither sensitive nor specific for the prediction of subsequent mortality in post-myocardial infarction patients similar to the findings in patients with acute myocardial infarction. There may be two exceptions to this. The first exception may be those patients with a low-ejection fraction (less than 40%) and advanced grades of PVCs who have an increased incidence of sudden death compared to patients with a low-ejection fraction without advanced grades of PVCs.²¹ The second exception may be those patients who develop ventricular arrhythmias with low-level exercise tests to a heart rate of 130 beats per minute or symptoms at three weeks post-infarction.²²

There are three controlled clinical trials that have been carried out in Europe, evaluating the prophylactic administration of beta adrenergic-blocking drugs in postinfarction patients. The first trial, in Sweden, using alprenolol in 230 patients demonstrated a significant reduction in sudden death, but not in total mortality or recurrent infarction.²³ The second study, also in Sweden and also using alprenolol, reported a significant reduction in sudden death and recurrent infarction but not in total mortality.²⁴ A large multicenter trial in England with practolol demonstrated a significant reduction in cardiac death and sudden death, particularly after ante-

rior infarctions.²⁵ In this trial the rate of recurrent infarction was reduced but was not statistically significant. The trial had to be terminated because of the ocular complications associated with long-term practolol administration. Since the incidence of recurrent infarction was shown to be reduced, the mechanism of the beneficial effect of beta adrenergic-blocking drugs may not be entirely related to their antiarrhythmic properties. In June 1978 a large multicenter trial was started in the United States, using propranolol in post-infarction patients, and the Medical College of Virginia is one of the clinical centers.

It is possible that oral antiarrhythmic drugs (quinidine, procainamide, disopyramide, phenytoin, or propranolol) on a long-term basis may be beneficial in postinfarction patients, but the results of additional clinical trials with drugs available in the United States are needed. At the present time postinfarction patients with frequent or advanced grades of PVCs may benefit from antiarrhythmic drugs, particularly when the PVCs are associated with a low-ejection fraction, angina pectoris, or a positive exercise test. It is essential that patients have a baseline monitoring or exercise study (whichever demonstrated the PVCs) prior to therapy and again after institution of therapy in conjunction with blood levels of the drug, when available, to evaluate the effectiveness of the antiarrhythmic therapy. It also should be remembered that we do not really have a good oral antiarrhythmic drug approved for use in the United States.

Without Organic Heart Disease

The significance of PVCs in persons without evidence of organic heart disease is less clear and can be a difficult problem with respect to treatment. Hinkle and co-workers reported that 62% of middle-aged American men had PVCs during a six-hour recording.²⁶ The presence of PVCs or more than 10 PVCs per 1000 beats correlated with an increased risk of sudden death. In the Tecumseh Study there was a significant increase in mortality in persons over the age of 30 with PVCs on an electrocardiogram compared to those without PVCs.²⁷ However, in both of these studies there was also a correlation between PVCs and the presence of coronary artery disease. Thus, PVCs appear to be only a marker for coronary artery disease in these studies, but are not spe-

cific enough for coronary artery disease to be useful in individual patients. Fifty percent of male medical students had PVCs during a 24-hour period, but only 2% had more than 50 PVCs during the 24 hours.²⁸ In persons without organic heart disease PVCs are frequently associated with fatigue, anxiety, or use of coffee, tobacco, tea or alcohol.²⁹ Removal of these factors will often decrease or eliminate the PVCs. In general the use of antiarrhythmic drugs should be avoided in individuals who have PVCs without evidence of organic heart disease.

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