However, many features in parasystole except the delay in the next discharge of the pacemaker can be explained from the observations in experiments by Jalife et al. Therefore, we think that there may be some cases showing the delay in the next discharge of the pacemaker, though we have not yet seen such cases. We also think that in our cases, too, "electrotonic modulation" may occur in the reentrant pathway containing the parasystolic focus; i.e., markedly delayed "conduction" in the reentrant pathway may be caused by "electrotonic modulation" in the late phase of the ectopic cycle.

We suggest a mechanism that might reconcile Dr. Jalife's theory with ours. Their observations in experiments showed that when the condition in the tissue surrounding the pacemaker was changed, the magnitude of electrotonic influence varied, and the transition point shifted earlier or later in the ectopic cycle. We think that there may be many small regions having different degrees of injury in the pathway in which parasystole originates; i.e., the pathway is inhomogeneous. "Electrotonic modulation" in such regions may cause "unidirectional block," "concealed conduction" and "longitudinal dissociation" in the pathway. Dual pathways due to "longitudinal dissociation" in such an inhomogeneous pathway consist of a "fast" and a "slow" pathway, in which "electrotonic modulation" occurs. When a sinus impulse falls in the early period of the ectopic cycle, it delays the next discharge of the pacemaker, whereas in the late period, it "captures" the pacemaker electrotonically. The early period of the fast pathway is longer than that of the slow pathway. When a sinus impulse falls in the terminal part of the early period of the fast pathway, it is "blocked" at the entrance of the pathway, and it will markedly delay the next automatic discharge in the region distal to the site of "entrance block." On the other hand, the same sinus impulse will fall in the initial part of the late period of the slow pathway, and "captures" the pathway after considerable delay. After several delayed "captures" of regions in the slow pathway, the impulse "reaches" the distal common pathway. Thereafter, the impulse retrogradely "captures" the fast pathway before the next markedly delayed discharge of the pacemaker can occur in the pathway. If this impulse "reenters" the slow pathway, a markedly slow ectopic rhythm will be caused by "microreentry." Such a slow "reentrant" rhythm may be "parasystole" in clinical cases. In such a case of "parasystole," when a sinus impulse falls in the early period of the fast pathway, the next "reentrant parasystolic" impulse will "capture" the pathway before the next delayed discharge of the pacemaker can occur in the pathway. Thus, in clinical cases of "parasystole," the delay of the next discharge of the pacemaker cannot be seen even though "electrotonic modulation" occurs.

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References

Alcohol and Hypertension

To the Editor:

We were interested in the recent paper on alcohol and hypertension by Wallace et al. in which they discuss the possibility that blood pressure elevation is related to the severity of alcohol withdrawal. We have shown in a group of alcoholic patients with a mean daily alcohol intake in excess of 80 g that this is indeed the case. Blood pressure exceeded 140/90 mm Hg in more than half and was closely related to the presence and severity of alcohol-withdrawal symptoms, graded on a seven-point scale using estimates of tremor, sweating and eating disturbances (fig. 1).

Of 36 patients who had no withdrawal symptoms, only three (8%) had a blood pressure > 140/90 mm Hg, and the mean systolic (SBP) and diastolic (DBP) blood pressures of this group (118.4 ± 2.3 mm Hg [± SEM] and 73.1 ± 1.6 mm Hg, respectively) were significantly lower than in patients who had withdrawal symptoms (mean SBP 147.3 ± 1.8 mm Hg, p < 0.001; mean DBP 90.9 ± 1.5 mm Hg, p < 0.001). Thirty of the 35 patients (86%) who had withdrawal symptoms of grade 3 or more were hypertensive.

The degree of blood pressure elevation is not merely a reflection of the patient's habitual alcohol intake, for, as Wallace and colleagues suggest, the period of abstinence before measurement of blood pressure is important. Patients in our study who had been drinking up to the time of admission showed a rise in both SBP and DBP over the subsequent 24 hours, which was related to the development of the withdrawal syndrome. It seems likely that heavy drinkers attending hospital clinics and participating in community health surveys abstain from alcohol for several hours beforehand and may therefore be in a state of relative alcohol withdrawal at the time of examination.

The alcohol-withdrawal syndrome implies physical dependence on alcohol, and one might think that this is an unlikely explanation for the excess of hypertension in patients drinking relatively moderate amounts of alcohol (40-100 g/day). We wonder whether a proportion of these patients underestimate their alcohol consumption. One cannot always be sure that patients reveal their true intake when self-administered questionnaires are used or

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Alcohol withdrawal symptoms.
when patients are asked merely to check the number of “drinks” they take in an average day. In a British study, the presence of abnormal liver function tests alerted the investigator to the possibility of excessive alcohol consumption in hypertensive patients. Raised serum transaminase levels are not found in moderate drinkers or after the occasional drinking session, but indicate that alcohol consumption has been sufficient (i.e., 80–300 g/day for at least 1 year) to produce some degree of liver cell necrosis.

The importance of the link between alcohol consumption and hypertension is that blood pressure falls in the majority of patients, usually to “normal” levels. Strict abstinence may not be necessary, but it is advisable to limit alcohol intake to not more than 20–30 g/day. In our experience and that of others, treatment with antihypertensive drugs is usually of no avail while patients continue to drink heavily.

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Coarctation Resection vs Patch Angioplasty
To the Editor:
Connor and Baker, in their recent paper comparing coarctation resection with patch angioplasty, stated that “the long-term results of patch angioplasty are not available, so the long-term complications of this procedure are not known.” This may have been true at the time the manuscript was submitted, but by the time their revision was accepted, Bergdahl and Ljungqvist had already described a disturbing high incidence of aneurysm of the aorta, unrelated to infection on the opposite side from the patch. They postulated that because the Orion or Dacron patches used are less elastic than the aorta, they could therefore increase the forces acting on the aortic wall opposite, and therefore cause aneurysm formation. All these patients had been followed for at least 17 years after operation.

I feel I must draw attention to this important paper lest any reader conclude that patch aortoplasty is necessarily the treatment of choice for any variety of coarctation.

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Benefits of Exercise for CAD Patients
To the Editor:
The recent article by Elsani and colleagues was encouraging to those of us who believe in the health benefits of an exercise program for patients with coronary heart disease. However, I have several questions for the authors.
(1) You report a highly selected group of 10 patients with coronary heart disease, all with asymptomatic exercise-induced ST-segment depression. This is a rather unusual group. How many patients did you screen to get these 10?
(2) In addition, they all sustained high levels of exercise, levels that younger, healthy men frequently cannot tolerate. Of those admitted to your intensive exercise program, how many adhere? How many have orthopedic or cardiovascular complications?
(3) The specific lead with ST-segment depression was not given. Was ST-segment depression in lead Z included? In lead Z, ST-segment depression is really ST elevation anteriorly and is not due to ischemia when it occurs over Q waves.
(4) How many of these asymptomatic men with coronary disease actually had false-positive ST abnormalities (i.e., depression not due to ischemia)? Even though they had coronary disease, they might not have had ischemia. Did you perform radionuclide tests on them?
(5) In postinfarction patients, how could you measure left ventricular mass when the M-mode beam can cut through areas of scar or compensatory hypertrophy? Even using a cross-sectional area approach, we have been unable to find changes. Though these results differ from those of most others, they are exciting, but their generalization to clinical practice is uncertain. The 10 men reported are a highly selected group with asymptomatic ST-segment depression and able to exercise at levels often difficult for younger men. If applied to most patients with ischemic heart dis-
Alcohol and hypertension.
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