The use of isotopes such as thallium-201 would also be a relatively noninvasive method to further subdivide the false positives from the true positives.

It is heartening to see the statement, “only 11 cases were picked up by the other criteria which emphasizes the importance of the exercise ECG in the early detection of coronary heart disease.” At the same time, however, the title, “False Positive Diagnostic Tests” may decrease the importance of the exercise ECG.

Because of the pandemic of coronary heart disease and the sometimes critical allegations of the limitations of the electrocardiographic response to exercise in predicting coronary artery disease, it behooves all of us who are interested in this particular aspect to eliminate as many of the known entities that produce false exercise tests by using all the noninvasive methods at our disposal.

I would find it of great interest if the authors could review their data in light of the above comments to see if the incidence of false positives would be less than quoted.

JOSEPH P. LISS, JR., M.D.
Saint Francis Hospital
University of Connecticut
Farmington, Connecticut

The author replies:

A person who is erroneously labeled with a diagnosis of latent coronary heart disease (CHD) may suffer serious psychological and economic consequences, and any noninvasive procedure which can minimize such errors should seriously be considered. In our study we have made an attempt to define the error-ratio of various criteria suggestive of CHD. One always later regrets procedures which for various reasons were not done (or thought of) in a study such as ours. Concerning the remarks of Dr. Liss we have the following additional information:

1) Standing ECGs were performed in all individuals in whom a suspicion of vasoregulatory abnormalities was present. (As stated all such individuals were excluded, including a number of individuals who presented with significant, early ST-depressions during the exercise test, provided these changes became less pronounced — or disappeared — as the test progressed into more strenuous stages.) Hyperventilation-ECGs have not been performed in our subjects.

2) In the individuals on whom we obtained angiograms we had no reason to suspect valvular heart disease. In none was a systolic murmur of more than I–II degree present, and in none was this pansystolic or late systolic. Systolic clicks were not present clinically or phonocardiographically. Thus hypertrophic subaortic stenosis and/or mitral valve prolapse was not suspected but since we did not perform left ventricular angiography we are unable to make any definite statement on this possible pitfall. The main reason for not including routine left ventricular angiography in our protocol was that our men represented presumably healthy individuals, and ethical considerations suggested that all invasive procedures should be limited to those recommended under medical advice for a cardiac complaint, and they were only hospitalized after the survey findings were thoroughly explained to them.

3) Echocardiography has not been performed, but since all angio-negative individuals are still alive we may sometime in the future have echocraphic findings on this group to correlate with the clinical data presented in the present article.

4) A detailed history of all medications was obtained in all 2014 survey-individuals. In the very few who used tranquilizers the primary ECG was disregarded. All such individuals were retested after 1–2 weeks off the drug(s), keeping in mind possible vasoregulatory abnormalities in these men after withdrawal of their prescriptions. In particular none of our men used saluretics, antiarrhythmics, antihypertensives, anticholinergics, sympathomimetic/sympatholytic drugs (or digitalis). Thus we probably have — with a reasonable degree of accuracy — excluded possible influence of drugs on the ECG. As stated in the paper individuals with electrolyte aberrations were retested after correction of such aberrations (or otherwise excluded).

During follow-up we have retested several times all men who had angiography and findings have been essentially similar. In particular the individuals whose angiograms were negative reflected this pattern, suggesting that the reasons for their ST-depressions — whatever they may be — were not acute, concurrent illness at the time of the survey. Only 2 of these individuals were clinically neurotic.

As we have suggested, our study might be considered a feasibility study. Any study repeating our procedures should carefully consider possible improvements in the study design with due consideration to the added risk if further invasive measures should be added.

JAN ERIKSEN
Rikshospitalet
Oslo, Norway

Paradoxical Hypertension in Postop Coarctation Patients

To the Editor:

The recent paper by Rocchini et al.1 provides convincing evidence for the involvement of the sympathetic nervous system and the renin-angiotensin system in the pathogenesis of paradoxical hypertension after coarctation resection, but the authors offer no explanation of the mechanism underlying this change. When he first described this phenomenon,2 Sealy suggested that the postoperative rise of pressure might be initiated by the arterial baroreceptors. Since there is elevation of both mean pressure and particularly of pulse pressure in coarctation, the baroreceptors are likely to be reset to a raised level of pressure, and will therefore operate to maintain the pressure at this level. When the coarctation is removed, the sudden reduction of afterload should lower both the mean and pulse pressure, thereby reducing the afferent input to the baroreceptors. Such a reduction would have effects similar to carotid occlusion, and induce a reflex increase in sympathetic drive, hence raising the pressure again.

The major objection to this theory is illustrated by figures 1 and 2 of the paper of Rocchini et al. There is no detectable immediate reduction of arterial pressure, and the first change (as noted by others) is in fact a large increase of systolic pressure. At this early stage, therefore, the arterial baroreceptors should be subjected to an even greater degree of stimulation than preoperatively, which should induce a reflex reduction of sympathetic tone, which manifestly does not occur. Rocchini et al. did not measure catecholamine levels, but the earlier observations of Goodall and Sealy3 indicated that urinary levels of epinephrine and norepinephrine are elevated from the first postoperative day. The evidence would thus suggest that sympathetic tone is raised at a very early stage. It would seem logical to suppose that this increase is reflexly induced. Similar circulatory changes have been described after surgical denervation of the carotid sinus in man,4 which may also result in transient postoperative hypertension. Denervation of the aortic arch baroreceptors might be expected to produce a similar result, although to my knowledge this has not been deliberately carried out in man. This is an unlikely cause for the hypertension following coarctation repair, however, since the aortic receptors are proximal to the coarctation, and unlikely to be damaged.

The most dramatic hemodynamic consequence of coarctation repair is the sudden increase of arterial pressure distal to the coarctation. Could this in some way be responsible for the paradoxical rise of pressure? One possible explanation that has not been suggested before would be via spinal sympathetic reflexes. In 1974, Lioy et al.5 described the effects of sudden distension of the thoracic aorta in cats, which is a situation hemodynamically analogous to the repair of a coarctation in man. This resulted in a
Paradoxical hypertension in postop coarctation patients.
T Pickering

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