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Two-Dimensional Echocardiographic Identification of Papillary Muscles

To the Editor:

The recent committee report on two-dimensional echocardiographic nomenclature refers to the two left ventricular papillary muscles as medial and lateral when viewed in the standard short-axis plane. Considerable confusion exists as to which of these represents the papillary muscle seen in the standard long-axis view. The excellent article by Tajik et al. which focuses on the anatomic correlates of two-dimensional echocardiographic planes, identifies the papillary muscle seen in the long-axis view as the one positioned medially in the short-axis section (their figures 6 and 9).

Figure 1 depicts single frames obtained from the two-dimensional examination of a patient with hypertrophic obstructive cardiomyopathy whose papillary muscles were markedly unequal in size. In the long-axis view, the papillary muscle arising from the left ventricular "posterior" wall appears enlarged. When the transducer was slowly rotated 90° to obtain the short-axis view, keeping this hypertrophied papillary muscle constantly in view, it was evident that this muscle was positioned in the short-axis section laterally, and not medially, as Tajik et al. concluded. The smaller medial papillary muscle corresponds to the papillary muscle arising from the inferior (diaphragmatic) wall of the left ventricle in the apical two-chamber view.

Our echo-autopsy correlations in other patients with papillary muscles of unequal size or markedly differing echo brightness due to the presence of fibrotic or calcific areas further support our findings. The same conclusion is reached if one inspects normal heart specimens and correlates them with transducer positions. Therefore, it appears that the identification of the papillary muscles as suggested by the Mayo Clinic group is erroneous.

Laura von Doenhoff, M.D.
Navin C. Nanda, M.D.
University of Rochester Medical Center
Rochester, New York

Figure 1. (upper panel) The long-axis view from a patient with hypertrophic obstructive cardiomyopathy. The papillary muscle (PM) arising from the left ventricular posterior wall (PW) is hypertrophied. (lower panel) The short-axis view from the same patient shows the hypertrophied papillary muscle positioned laterally. This was confirmed repeatedly during slow scans from the long-axis plane to the short-axis position, keeping the enlarged papillary muscle constantly in view. AV = aortic valve; VS = ventricular septum; LA = left atrium; AO = descending aorta; MV = mitral valve; RV = right ventricle; LV = left ventricle; LPM = lateral papillary muscle; MPM = medial papillary muscle.
Diet and its Relation to Coronary Heart Disease

To the Editor:

In an editorial on our paper dealing with diet and its relation to coronary heart disease (CHD) and death,1 Scott et al.2 addressed some general statistical issues. The initial tables in our paper compared the means of various nutrients for CHD cases and noncases. The editorial describes circumstances in which such a comparison will mislead us. We would go further than the editorial. It is, in fact, conceptually imprecise to consider cases and noncases arising from a set of prospective observations as constituting two separate populations. They arise from one population and should be so treated.3,4 Thus, we are not persuaded that the statistical issue warrants the concerns expressed in the extended consideration in the editorial of the two-population model. On the other hand, at the practical level, the t test for mean differences usually yields conclusions consistent with those from a more exact analysis.5 In the analysis of large bodies of data it has the advantage of being cheaper and less time-consuming than a logistic analysis. This is particularly attractive when a large number of variables must be considered. However, when an examination of the mean differences suggested a potentially significant association, we confirmed it with a logistic analysis, as the editorial noted. Uniformly, the conclusions were the same by both methods.

It is true that the logistic function assumes monotonicity of risk over the independent variables.6 As was noted in the editorial, sample data for the key diet variables were presented in our paper. The trends were “not easily discernible,” but nothing about the data would suggest we reject the assumption of monotonicity. However, the possibility does exist that some diet variables found to have insignificant relations when examined by our methods are, in fact, related to the incidence of coronary heart disease or death. Beyond these statistical issues, however, it must be conceded that in some instances the biologic facts may be more complicated. Thus, it is possible that persons consuming large quantities of alcohol on a regular basis may be at greater risk of developing coronary heart disease than moderate drinkers, as one study suggests.6 Our data do not indicate that to be the case for our populations; hence we had no reason to allow for that in our model.

Multiple comparisons can affect the probability estimates. However, we emphasized not individual test results, but the replication of results; that is, the concordance of results from three separate studies. And what we are concerned with now is in finding out whether other studies confirm or help to explain our findings.

T. GORDON
A. KAGAN, M.D.
M. GARCIA-PALMIERI, M.D.
W.B. KANNEL, M.D.
W.J. ZUKEL, M.D.
J. TILLOTSON, M.A.
P. SORLIE, M.S.
M. HJORTLAND, Ph.D.
Division of Heart and Vascular Diseases, NHLBI; and the Department of Medicine, School of Medicine, University of Puerto Rico, San Juan, Puerto Rico

References


Naloxone Administration Before Sleep

To the Editor:

Rubin et al.1 demonstrated that naloxone administration before sleep prevented the decrease in systolic pressure that is normally seen during the second cycle of deep sleep. This effect of naloxone was delayed, becoming manifest more than 140 minutes after drug infusion. The mechanism of naloxone's action on the blood pressure may be mediated through naloxone-induced release of endogenous vasopressin.

Both apomorphine and naloxone block the morphine-induced increases in serum prolactin,2 and the time course of effect of naloxone on preventing the blood pressure decrease during sleep coincides with its effect on prolactin release, maximal at 180 minutes.3 Apomorphine is a potent dopamine-receptor stimulant, and some of the morphine withdrawal signs have been linked to an increased stimulation of dopamine receptors.4 Rowe et al. showed that apomorphine administration (16 mg/kg subcutaneously) resulted in marked increases in plasma vasopressin level, associated with nausea and/or vomiting.4 Blockade of opioid withdrawal symptoms can be accomplished by haloperidol, a dopamine-receptor blocking agent.5 Haloperidol also blocks the apomorphine-induced vasopressin release.4 Although naloxone is said to antagonize some of apomorphine's actions,4 it potentiates apomorphine's effect on rotations in rats with unilateral 6-hydroxydopamine lesions of the substantia nigra.6 Naloxone was recently demonstrated to enhance apomorphine's induction of vasopressin release as well.6

The finding that naloxone's hypertensive effect is delayed by more than 2 hours suggests that the control it exerts on blood pressure is indirect. Plasma vasopressin has been demonstrated to be responsible for long-term blood pressure maintenance in rat and dog.8 Apomorphine induces vasopressin release and its action can be blocked by haloperidol, which also blocks naloxone-induced withdrawal reaction, and so, possibly, naloxone's delayed hypertensive effect may be mediated through vasopressin release. Although Lightman et al. proposed that naloxone may block or exert no effect on vasopressin release,9,10 it is noteworthy that in these two studies, naloxone was used at very low doses (0.08–0.12 mg/min) administered by slow infusion over a protracted period (40–120 minutes), and was not associated with a delayed hypertensive effect. In contrast, the studies demonstrating a delayed effect on blood pressure and elevation in plasma vasopressin used much larger doses (14–20 mg), administered in bolus form.11 Because some signs of opioid withdrawal have been attributed to an increase, and others a decrease, in dopaminergic activity,6 naloxone's effect
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L von Doehoff and N C Nanda

Circulation. 1981;64:651-652
doi: 10.1161/01.CIR.64.3.651

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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