should be substituted for the inconstant and more subjective criterion of asbestosis in evaluating persons at risk for lung cancer.

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**REFERENCES**


**Left Ventricular Diastolic Overload in Young Africans**

*To the Editor:*

The article by Schamroth and colleagues (Chest 1986; 89:95-99) on etiologic differentiation by ECG of left ventricular diastolic overload is interesting and potentially valuable to the extent that it expands the work of the distinguished Mexican cardiologists1 who first introduced the concept. Before one can evaluate this material, I hope that the authors can correct two conspicuous omissions from the Methods section to provide confidence in the results and their applicability. First, were the ECGs measured blindly, ie, without knowledge of the diagnoses? Second, the reader does not know the nature of the population from which these data are drawn. Thus, we would need to know ages and, given its source—the Baragwanath Hospital—the racial composition of these individuals. Age would particularly affect the prevalence of complications, particularly heart failure, additional chamber enlargements accruing over the years and, of course, the prevalence of coronary artery disease (which would be a significant factor in older and Western populations). The latter could introduce other alterations, particularly in repolarization. Congestive failure, or any fluid retaining condition, could markedly affect voltage. These remarks are not meant to criticize an interesting study, but rather to request complete data to indicate, for example, whether the results may be strictly applicable to a young (and presumably) black population.

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**REFERENCES**

1. Cabrera GE, Monroy JR. Systolic and diastolic loading of the heart. Am Heart J 1962; 43:661-70
2. Spodick DH. Low voltage ECG and pericardial effusion. Chest 1979; 75:113-14

*To the Editor:*

We would like to thank Dr. Spodick for his interest in our paper and his pertinent comments.

The purpose of our paper was to document a new electrocardiographic sign in the further differentiation of the four main causes of left ventricular diastolic overload. The presentation was thus, in effect, a pilot study, and the cases were selected consecutively on the basis of diagnosis. The electrocardiographic examinations were consequently interpreted with foreknowledge of the diagnosis.

All patients came from the black population of Soweto, which is served by Baragwanath Hospital. Coronary artery disease is virtually non-existent in this population. For example, approximately 50 cases of myocardial infarction are seen annually from an admission of almost 30,000 patients to the Department of Medicine. Furthermore, most of the patients in the study were children or young adults (see below). Coronary artery disease could therefore be excluded as a factor.

Since rheumatic heart disease is rampant in this population, most cases of mitral and aortic incompetence occur in the first three decades, as do the cases of congenital heart disease.

Thus, the ages in years of the various groups were as follows:

- Mitral incompetence: 10 to 30, median 15
- Aortic incompetence: 12 to 35, median 19
- Patent ductus arteriosus: 1 to 13, median 4
- Ventricular septal defect: 1 to 26, median 10

None of the patients were in severe congestive cardiac failure, and all had left ventricular functions within normal limits.

We appreciate the opportunity Dr Spodick has given us to clarify these points.

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**Inotropes, Inotropics, and Cardiotonic Agents**

*To the Editor:*

I am concerned about the introduction into the cardiologic lexicon of the terms "inotrope" or "inotropic agent" to designate the action of drugs that produce a positive inotropic effect. The word inotropic (ino = fiber, tropic = turning toward; having an affinity for), when used by itself, does not connote the direction of the effect produced. The modifying adjectives "negative" or "positive" must be used with the word inotropic, in order to properly describe the action produced on the myocardium. The introduction of the word "inotrope", both in peer-reviewed papers and in drug company advertisements, suggests that there has been tacit agreement by editors, journal referees and authors (and the FDA, also?) to accept the word "inotrope" as a short-hand term to describe a positive inotropic agent. Is this new jargon superior to the more complete and accurate phrase?

Similarly, designating old and new agents as "inotropics", or capable of producing potent "inotropic effects", defies good scientific word usage. Would we tolerate the phrase "potent inotropic agent" to describe the negative inotropic effect of a high dose of versapamil? Not likely. These errors are compounded by the use of the phrase "inotrope-vasodilator" to describe agents that possess both a positive inotropic effect and peripheral vasodilator action. Perhaps authors and editors become lazy as terms are used casually in conversations, eg, "beta-blockers" when we mean to say "beta-adrenergic receptor blocker".

Have the editors of cardiovascular journals officially accepted the words "inotrope" or "inotropic agent" to mean an agent that produces a positive inotropic effect on the myocardium? Otherwise, we should expunge these short-hand terms from our written and spoken vocabulary. Authors who have been using the new terminology might rediscover old terms such as "cardiotonic" (with its shortcomings) if they insist on a single word description for an agent that increases myocardial contractility by a direct positive inotropic effect.

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