measurements to the nearest millisecond may well imply unjustified precision when using a recording paper speed of 100 mm/sec and time lines of 10 msec, but it is unlikely that the precision of our data would be enhanced by measuring to the nearest 5 msec on paper with 20 msec time lines, as recommended by Weissler and co-workers,¹ or to the nearest 5 msec on paper with 40 msec time lines, as reported by Aronow et al.²

The STIs obtained immediately following exercise were corrected for heart rate using regression equations developed in resting supine subjects.¹,³ Such an approach has been used by other workers in this field,⁴ and it is our opinion that such corrections would at least partially neutralize the chronotropic effects of exercise and better expose other exercise-related hemodynamic alterations. Although we concur with Dr. Weissler that it would have been preferable to apply regression equations developed in supine exercising subjects, to our knowledge such data are not presently available. Furthermore, the ratio of PEP/LVET did not require heart rate correction.

From the conceptual standpoint, we agree that the STIs deviate from normal in parallel with other hemodynamic parameters as quantitated by correlation coefficients. In our study, these noninvasive measurements permitted the separation of a group of patients with chronic coronary artery disease (CAD) from an age- and sex-matched group of normal subjects, the former group having a significantly longer PEP, shorter LVET, and larger PEP/LVET at rest and exercise. The abnormal STIs occurred predominantly in a subgroup of patients in the CAD group in whom significant abnormalities of stroke-volume index, cardiac index, LV dP/dt, or ejection fraction existed. However, we must take issue with Dr. Weissler if he contends that the STIs reliably detect minor abnormalities of ventricular function in the individual patient with chronic CAD. Despite significant correlations in our report between the STIs and several directly measured hemodynamic parameters, the overlap in the data prevents any confident prediction of left ventricular dysfunction as conventionally defined unless the functional abnormalities are severe. For example, in our figure 2 which correlates the left ventricular ejection fraction as measured angiographically with PEP/LVET obtained immediately after exercise a PEP/LVET of 0.30 was associated with ejection fractions varying from 0.40 to 0.90. This occurred despite the significant correlation (r = -0.67; P < 0.0005) between these two variables.

On the basis of our study, we are in agreement with Dr. Weissler that one should not attempt to predict from measurements of STIs such hemodynamic variables as cardiac index, stroke-volume index, left ventricular end-diastolic pressure, pulmonary artery and wedge pressures, exercise factor, or LV dP/dt, or attempt to predict the presence and extent of coronary artery disease or left ventricular asynergy as demonstrated on cineangiography. Furthermore, the available data suggest that the STIs cannot reliably separate the individual subject with normal cardiac function or normal coronary arteries from the patient with chronic CAD with mild-to-moderate left ventricular dysfunction. Yet it is in this latter group of patients rather than those with clinically more apparent abnormalities of ventricular performance that STIs might have offered the most useful contribution as a noninvasive bedside technic. If such indirectly obtained time intervals provide an imprecise potentially misleading measure of the underlying interdependent parameters of left ventricular function, then caution should be exercised in applying them in the clinical evaluation and follow-up of the individual patient with CAD.

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References

The Conotruncus: I. Its Normal Inversion and Conus Absorption

To the Editor:

I have read the interesting paper by Drs. Goor, Dische, and Lillehei (Circulation 46:375, 1972). May I congratulate the authors on their embryologic work.
However, I found in this paper what I consider to be a misunderstanding of the difference between the anatomic term "conus" and the embryologic term "conus." The anatomic term conus colloquially is used synonymously with the term "outflow tract" of the right ventricle and infundibulum. The embryologic term conus is used synonymously with the term bulbus.

If we follow the concepts of Pernkopf, Wirtinger, and Asami as to the development of the heart, then the definitive right and left ventricles both have components of proa ampulla, meta ampulla, and bulbus (conus-embryologic) in their composition. However, the left ventricle is mostly proampulla with lesser metaampulla and bulbus, but the right ventricle contains more metaampulla and bulbus (conus-embryologic). This is because in the movements of the auricular canal to the right, and in the absorption of the bulbus during the second phase of development, there is expansion of the tricuspid orifice and dorsal wall of the metaampulla, shrinkage of the right side of the proampulla, and shrinkage of the mesocardial wall of the proximal bulbus, as it lies in the groove between the mitral and tricuspid orifices. Thus, the inflow tract of the right ventricle contains both proampulla and metaampulla, while the outflow tract contains both metaampulla and bulbus (conus-embryologic). The outflow tract of the right ventricle is only partially bulbus (conus-embryologic).

The outflow tract of the right ventricle anatomically is lined by the septal and parietal band groups of muscles. According to Pernkopf and Wirtinger and many others these groups of muscles are bulbar and metaampullar in origin.

Thus, the embryologic term conus and the anatomic term conus are not the same since the anatomic term conus includes in it embryologically both bulbus (conus-embryologic) and metaampulla. One can get around this semantic confusion by calling the outflow tract of the right ventricle infundibulum, and not conus.

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The authors reply:

To the Editor:

Needless to say, Dr. Lev's summarization of the development of the base and outflow portions of the heart is as accurate as it can be. In fact, it conforms with our present and previous reports dealing with the development of the base of the heart and the interventricular septum.1

Dr. Lev, however, does not define what part of his definitive outflow tract of the right ventricle is derived from the embryologic conus and what part is metaampular. At what stage of human development should we substitute the embryologic conus for the anatomic conus and why? Moreover, Dr. Lev declines to say why he objects to the use of the same term (conus) for the same part of the heart regardless of whether it is embryonic or definitive. In other words, why should we leave the embryologic conus as a segment without a name in the definitive heart?

Our efforts to define the true anatomic conus are not merely academic. In recent years Van Praagh2 identified the embryologic and true anatomic conus in the various types of transposition of the great arteries. (Paradoxically, however, Lev's is the one who initiated the interest in the anatomic conus in the American literature.) Proper recognition of this structure is priceless for a surgeon at work as well as during diagnostic procedures in complex cardiac malformations. Our own studies4 of transposition complexes not only endorse Van Praagh's conus definition, but have shown that each of the four basic conotruncal developments described in the present report, namely, inversion of conus, inversion of truncus, leftward shift of conoventricular junction, and conus absorption may individually fail. The conus, hence, (embryologic and true anatomic) is the factory where transposition complexes are manufactured. It deserves a name.

We cannot, therefore, agree with Dr. Lev's approach—that Pernkopf and Wirtinger's dictum5 (although monumental) is a holy book that cannot be changed.

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The Conotruncus: I. Its Normal Inversion and Conus Absorption
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