Communications to the Editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Severity of Aortic Stenosis

To the Editor:

The report on noninvasive estimation of the severity of aortic stenosis by Voelkel and colleagues (Chest 77: 135, 1980) is an important contribution to the conceptual aspects of testing. The authors handled their data with great sophistication and confirm the common experience that prediction from one or more noninvasive descriptors is risky. One wonders, however, whether it is appropriate to use the Bazett formula for rate correction. That was determined for the QT interval some 60 years ago in a patient population of uncertain relevance to other studies. In any case, validity of extrapolating from a QT interval relationship to mechanical time-based measurements is uncertain. It is likely that some of the measurements are not truly rate-related and others have their own regressions on heart rate.

(The authors accept this for LVET, although it, too, is sometimes “corrected” à la Bazett. I am not proposing that ascertaining individual rate relationships would necessarily improve the value of the measurements. I am only questioning whether the Bazett formula is valid and, if so, whether it is optimal, ie would the usefulness of some or all of the authors’ measurements be improved by more appropriate rate-correction? It was of considerable interest that the “corrected” Q wave to peak murmur measurement (Q-MP) was by far the best single indicator of aortic valve area. The authors note, in passing, “Phonocardiograms can be technically difficult.” This needs greater emphasis, particularly with relation to ejection murmurs. The highly selected examples—often a single cycle—illus- trating PCC in papers and textbooks do not accurately mirror day-to-day experience. Experienced phonocardiographers are painfully aware that murmur configuration frequently varies both with the respiratory cycle and the cardiac cycle so that it is common to have wide beat-to-beat variability of the largest or “peak” vibrations; frequently there is only one peak vibration in a single cycle. Here, this in itself might not have been an absolute obstacle (although it is a formidable one) if the study protocol had included methods for minimizing biases. As in valid therapeutic trials, valid diagnostic trials should have equal safeguards against bias. The method, of course, should be blind to observers to data other than those which they are observing and measuring. Thus, we should have a statement of assurance that those doing the measurements—and in particular the measurement of the Q wave to peak murmur—knew nothing of the patients’ other results. (This must have been feasible given the large number of authors of this report.) Yet, the readers must always take “Material and Methods” sections at face value as “telling all” and here a comparable assurance is lacking.

Clinical experience and the broad relationship between the time-courses of murmurs and gradients indicate that, in general, a late-peak murmur is indeed more likely than an early-peak murmur to accompany greater aortic obstruction. (It should be noted that this was first reported by Aldo Luisada.) Yet, the reliability of the authors’ determination of the relative value of Q-MP and the numerical measurements they report rests on their methodology.

These comments are aimed at soliciting clarification and amplification rather than in criticism of a valuable report which is otherwise well done.

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Effect of Angiotensin on Left Ventricular Function in Man

To the Editor:

I have read with great interest the paper by Bianco et al1 in the February issue. I agree with the observations; however, their statement “there is no information on the LV ejection fraction response to afterload augmentation produced by angiotensin infusion” is incorrect. We not only reported the effect of angiotensin on LV ejection fraction in both man and dog, but our report also described the effects of angiotensin infusion on left ventricular contractility in the normal and diseased heart of the intact dog and man.2 Whereas the indices of left ventricular contractility decline in both man and dog whether normal or diseased (cardiomyopathy and ischemic myocardial scarring respectively) LV ejection fraction was decreased in cardiomyopathic patients only. The LV ejection fraction remained unchanged in normal man and mongrel dog.

The discrepancy in the two studies is obviously a reflection of experimental protocol. In contrast to the magnitude of pressure increment in the Bianco study (60-70 percent of control values), the mean rise in arterial pressure in each of the four groups in our study ranged between 15 to 25 percent above the control levels. Variations of angiotensin infusion are reported to produce variable effects on the left ventricular function.3 Angiotensin infusion at any of those levels, nevertheless, appears to depress cardiac function in man whether normal or diseased.

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References

2 Ahmed SS, Levinson GE, Weisse AB, Regan TJ. The

To the Editor:

It's amazing to me how in this age of computers that an article as specific as yours was somehow missed in our personal review and in our automated computer search. However, I have had the occasion recently to review it and agree with you that your report on the effects of angiotensin on left ventricular ejection fraction in both man and dog was well described. Although your methods of determining left ventricular ejection fraction and your reduced levels of infused angiotensin with their resultant lower increments of pressure elevation differed from our current study, I would agree that your conclusions were appropriate. In both studies, angiotensin infusion had an effect on depressing left ventricular contractility in both the normal and diseased heart. The discrepancy in the depressed left ventricular ejection fraction found in both normal and diseased hearts of man may be explained, as you suggest, by methods of ejection fraction measurement and by levels of angiotensin infused.

We both conclude that angiotensin infusion at either level used appears to depress cardiac function in man, whether normal or diseased. I appreciate your having brought this to our attention.

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Diffusing Capacity in Alcoholics

To the Editor:

The editorial by Dr. Banner in the April, 1980 issue of Chest (77:460-461) addressing the interaction of "Alcohol and the Lung," and the accompanying article by Peavy et al in the same issue (77:488-492), both make reference to reports of reduction in the single breath diffusing capacity for carbon monoxide (DLco) in alcoholics. They correctly note that this has not been accounted for by cigarette consumption alone and that it seems to be reversible since the diffusing capacity is normal in former alcoholics. We wish to point out, though, that when the DLco is also corrected for anemia in these individuals, the DLco is not significantly reduced. This information comes from a study1 in which the DLco was measured in 20 consecutive patients with a physician diagnosis of excessive alcohol consumption. The mean observed DLco was significantly reduced to 70 percent of predicted (± SD 15.8) and was less than 70 percent of predicted in 12 of the 20 patients. However, when the DLco in anemic individuals was corrected2 to a hemoglobin concentration of 14.7 g/dl the mean DLco was then 82.3 percent of predicted (± SD 12.9) and was abnormal in only 4 of the 20 patients. In anemic subjects, the mean DLco adjusted for hemoglobin was in fact no different from the DLco in subjects with a normal hemoglobin. Furthermore, when the DLco prediction is corrected for the smoking history3 there is no difference (P > 0.2) between predicted and observed DLco adjusted for hemoglobin.

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REFERENCES

To the Editor:

The data presented by Fisher is at variance with my own work1 and that of Emirgil et al2 which found that diffusing capacity is impaired in alcoholic patients. Fisher attributes this difference to our failure to correct for anemia. I believe the different results can be attributed to other factors. I did not correct for anemia in my patients since none was significantly anemic (all had a hematocrit > 40 percent). The hematocrits of the patients in the study by Emirgil et al2 ranged from 36 to 47 percent. I believe Dr. Fisher's conclusion can be attributed to the use of a regression equation with large scatter and to the matter of the definition of alcoholism. The mean diffusing capacity of our patients was less than his, 62 percent vs 70 percent of predicted. All of our patients were drawn from a group of individuals admitted for alcoholic detoxification, confirming the severity of their addiction. The patients of Fisher were judged alcoholic if their physicians felt they consumed excessive quantities of alcohol. This is a subjective judgment and may relate more to the individual physician's concept of alcoholism rather than to the amount of alcohol actually consumed.

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REFERENCES

Diagnosis of Sarcoidosis by Transbronchial Lung Biopsy

To the Editor:

The article by Roethe et al (Chest 1980; 77:400-02) deserves some comment. While the authors have attempted to determine the optimal number and sites of transbronchial lung biopsies necessary to diagnose sarcoidosis, their data fall short of providing conclusive answers.

COMUNICATIONS TO THE EDITOR 123