Pulmonary Diffusing Capacity

To the Editor:

The recent article on 'Pulmonary diffusing capacity in disorders of the pulmonary circulation' by Dr. J. H. Burgess published in the March issue of this Journal prompts us to make a few observations, since our findings on the measurements of Dm and Vc in patients with mitral valve disease are somewhat conflicting with their report. First of all, it is unfortunate that Dm and Vc measurements were not made on all of their patients. It is difficult to accept the statement that 'the values of Vc are regularly normal when Dco is normal or increased when Dco is high.' As a matter of fact, Dr. Burgess himself points out that patients with mitral valve disease are well known to have high Vc in spite of a normal Dco because of a concomitant reduction in Dm. The actual values of Dm and Vc in 10 of their patients in group 2 (table 2) are rather surprising. Although the results of Dm and Vc estimations in mitral valve disease have been extremely conflicting, almost all the studies reported so far show a decrease in Dm as a consistent abnormality. The only exception is the study by Yuba. The Vc has been variously reported as being decreased, normal or increased. Unfortunately, Dr. Burgess does not give the normal values of Dm and Vc in his laboratory, but to us the Dm in 10 patients in group 2 appears to be normal or increased. This is a group of patients with severe mitral stenosis and high PVR where one would have expected the Dm to be considerably decreased.

Almost all the studies reported in literature show that the Vc/Dm ratio is considerably increased in patients with mitral valve disease as compared to normal subjects. It has not been less than 1 in any of the reported studies. An increase in this ratio may occur as a result of increased resistance to diffusion by the alveolar-capillary membrane. Vc/Dm ratio would also be directly proportional to the radius of the capillaries and independent of their number or length. In presence of capillaries of different radii, the Vc/Dm ratio would vary as the geometrical mean of the radius of the capillaries. In all the patients in group 3 and most of the patients in group 2 one would expect the radius of the capillaries to be considerably increased. This would be reflected in an increased Vc/Dm ratio as is borne out by the studies reported earlier. Therefore, the observation of an extremely low Vc/Dm ratio (0.3 approximately) in group 2 patients of Dr. Burgess is rather difficult to explain. Such a low Vc/Dm ratio has never before been reported even in normal subjects.

References

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Figure 1

Figure 2. Graph of A1-A2 (X axis) versus H1-H2 (Y axis) during initiation of type I SVT (open circles) and type II SVT (open triangles) at basic cycle lengths of 480 msec and 490 msec. The scatter of closed circles may be due to the fact that the plotted data were obtained at different times during the study and at slightly different basic cycle lengths.

CASE 2

- NO SVT
- TYPE I SVT
- TYPE II SVT

A1-A2 (msec)

H1-H2 (msec)

280 290 300 310 320 330 340 350 360 370 380 390 400

0 200 220 240 260 280 300 320 340 360 380 400
LETTERS TO THE EDITOR

Our own findings indicate an increase in Vc/Dm ratio as the most characteristic abnormality in patients with mitral valve disease. In the accompanying figure Vc has been plotted against Dm in different clinical situations including 19 patients with mitral valve disease. Isopleths of DLco which follow a rectangular hyperbola have been constructed assuming \( \frac{1}{\beta} = 1.263 \). Eighteen of the 19 patients with mitral valve disease have a ratio > 3 which is more than the ratio in all the 18 normal subjects. These estimations were performed by a steady state technique, the details of which have been described earlier.4

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References

The author replies:

To the Editor:

The observations of Drs. Pande, Gupta and Guleria are limited to measurements of the subdivisions of the diffusing capacity in patients with mitral valve disease. This is a minor point of my article. Estimations of Dm and Vc are based on the measurement of Deco at different inspired oxygen tensions and the graphical solution of Roughton and Forster's equation. As a result, values obtained for these subdivisions are considerably less reliable than the total Deco. One should not draw conclusions from variations in Dm and Vc when the Deco is normal. An increase in inspired oxygen tension in patients with mitral valve disease probably produces physiological changes in the pulmonary capillary bed. This casts further doubt on the interpretation of Dm and Vc.

The major point of the above letter is their finding of a low Dm and therefore increased Dm/Vc ratio in patients with mitral valve disease. However, Dm is a less reliable measurement than Vc, especially when the pulmonary capillary blood volume is small.1 In the latter case the slope of the line for the graphical solution for Dm and Vc is steep. The intercept on the y axis is then less certain and Dm varies widely. It may approach infinity when Vc is very low indicating that the capillary resistance to diffusion predominates. This explains the rather high values for Dm that I reported.

Deco measurements are clinically useful in patients with mitral valve disease and other pulmonary circulatory disorders. Measurements of the subdivisions do not provide further information of clinical value. For the reasons stated above, I do not believe that values obtained for Dm in these patients are very meaningful physiologically. Conclusions regarding capillary geometry based on changes in Dm and a Vc/Dm ratio in patients are unreliable. The subdivisions of the Deco are conceptually useful, but interpretation of the total Deco is on much firmer ground.

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Sleep and Ventricular Premature Beats

To the Editor:

We were pleased by the results reported by Lown and his colleagues (Circulation 43: 691, 1973) regarding the importance of neural mechanisms to the ventricular ectopic activity seen in some patients. Their suggestion that neural events might trigger ventricular extrasystoles in patients is substantiated by experimental studies in animals. We as well as others1 have shown that electrical stimulation of brain centers will produce a variety of cardiac arrhythmias. The same is true when peripheral autonomic nerves are stimulated.2 Furthermore, circumstances that give rise to arrhythmias such as digitalis administration and experimental myocardial infarction produce generalized excitation of brain stem nuclei.1,3 Depression of these brain sites by neurodepressant drugs or surgical removal delays and even corrects these arrhythmias.1,3

The animal data suggest that enhanced sympathetic tone is the causative factor in disrupting cardiac rhythm. But as pointed out by Lown and colleagues, "it is hard to account for the failure of large doses of propranolol to reduce VPBs in three patients, though such a result was observed with sleep alone." This seeming paradox becomes clear when one considers the results of Randall and colleagues.2 They have shown that electrical stimulation of cardiac sympathetic nerves produces arrhythmias which cannot be prevented by pretreating animals with propranolol. Additionally, it was shown that the arrhythmias could not be blocked by atropine. Thus, arrhythmogenic stimuli from the CNS may travel to the heart through nerves whose neuroeffector junctions are not amenable to blockade by conventionally employed antagonists.

Lown and colleagues put forward the view that "the pharmacologic focus should be in restraining the neurophysiologic trigger rather than in attempting to protect the cardiac target." This is an approach that we have been advocating for several years. For example, we have found that several of the drugs currently in use for treating arrhythmias possess neurodepressant actions.1 Furthermore,
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