1965 Report of Idiopathic Hypertrophic Subaortic Stenosis with Ostium Secundum Atrial Septal Defect

To the Editor:

In an article entitled “Idiopathic Hypertrophic Subaortic Stenosis with Ostium Secundum Atrial Septal Defect: Successful Surgical Correction” (Chest 68:246-248, 1975), Smith and associates reported the surgical correction of coincident idiopathic hypertrophic subaortic stenosis with ostium secundum atrial septal defect. These investigators stated that in 1971 their patient was reported by Forker and Morgan1 as the “first” patient with coincident idiopathic hypertrophic subaortic stenosis and ostium secundum atrial septal defect; however, it would appear that the first case may have been reported prior to that in the American Journal of Cardiology in 1965.2 Although we are fully aware that, in fact, our case might not have been the first, a thorough review of the literature at that time suggested that it was. Surgical repair was attempted in that four-year-old patient, with initial correction being directed towards relief of the outflow obstruction; however, the patient died in surgery. That surgical correction of both lesions is possible is clearly shown by the report of Smith and associates.

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REFERENCES

Ventricular Unloading Agents

To the Editor:

The editorial by Kattus entitled “Antiangina or Antimyocardial Ischemic Drugs” (Chest 67:832-833, 1975) correctly emphasizes the need to expand the concept of antianginal drugs to the broader designation of “antimyocardial ischemic drugs.” But why stop here? For example, the organic nitrates have been shown to be useful in the management of congestive heart failure.1-3 Their beneficial effects appear to be mediated by systemic (ie, ventricular unloading) action rather than through their classic “coronary-vasodilator” action. How should we best classify these nitrates (and other vasodilators)? Certainly, the term, “antianginal” nitrates, is too narrow. How about “ventricular unloading agents”? New applications of old drugs always pose these questions. (Should dipyridamole, aspirin, and papaverine, which inhibit platelet aggregation, be reclassified? Probably not. Their main pharmacologic actions require myocardial, analgesic, and peripheral vasodilator classification. Yet many physicians and investigators use them solely as antplatelet aggregation agents to modify thrombus formation.)

I agree that new designations, such as “antimyocardial ischemia drugs,” are probably more precise, at least as they apply therapeutically. Hopefully, such phrases as “ventricular unloading agents” may also be more widely employed.

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Intermittent Mandatory Ventilation

To the Editor:

The editorial of Dr. Petty entitled “IMV vs IMC” (Chest 67:630-631, 1975), while full of witticisms, is not an accurate reflection of the real uses of intermittent mandatory ventilation (IMV), and I submit that intermittent mandatory cerebration (IMC) as advocated by Dr. Petty would allow one to reach a more favorable decision about the utility of IMV.

The advantages of IMV, which has been developed at Gainesville, Fla, and modified at other centers, are the following: (1) In selected patients,
IMV allows for a smoother transition from mechanical to spontaneous ventilation. (2) The mean intrathoracic pressure on IMV vs mechanical ventilation should be lower, and the incidence of pneumothorax has been found in some centers to be lowered by use of IMV vs standard mechanical ventilation. (3) The use of IMV allows one to have an alert responsive patient while on end-expiratory pressure; and, thus, the dangers of curare and other sedatives are avoided. What is needed are controlled studies to determine the uses and limitations of IMV. Witticisms regarding IMV are merely that; they do not answer the question of its real uses.

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To the Editor:

My main criticisms of intermittent mandatory ventilation (IMV) were presented in the editorial (Chest 67:630-631, 1975) and included the fact that no controlled clinical trials of acceptable design in support of IMV were available, although this ventilatory technique is widely heralded as a superior means of weaning patients from mechanical ventilation. In the interval, no studies have appeared. The problem of the resistance of the artificial airway may or may not be clinically significant, depending upon the size of the tube and the resources of the patient.

I was fascinated to find out from Dr. John Downs' group, whom I visited in San Antonio, Tex, that IMV is usually begun at a rate equaling the patient's own spontaneous respiratory rate. This is tantamount to controlled ventilation; but if the combined assisted and controlled mode were used along with sufficient cycling sensitivity, this would approach a true state of assisted ventilation, which is the preferred technique in my opinion. Only in the recovery phase is the mandatory rate reduced. It was pointed out to me that with this technique, patients rarely need to be paralyzed. Since I abhor paralyzing patients and know that resorting to this technique is an admission of defeat in terms of ability to interface a ventilator to suit the patient's need, I became somewhat more interested in IMV at a rate equal to the patient's own respiratory rate. I see the possibility of convincing those who still feel they must paralyze patients by using this ploy to get them to interface the ventilator to meet the patient's needs and thus assist the patient in his mechanical work of breathing. If IMV accomplishes this, a great victory will be won. As a matter of fact, I am considering writing an editorial entitled "In Defense of Intermittent Mandatory Ventilation."

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Electrocardiographic Terminology

To the Editor:

We highly agree with the comments of Dr. Flowers in the Communication to the Editor, "Aerosolized Isoproterenol and Myocardial Infarction Analog" (Chest 68:271, 1975), which is related to our paper entitled "An Electrocardiographic Pattern of Acute Myocardial Infarction Associated with Excessive Use of Aerosolized Isoproterenol" (Chest 68:107-110, 1975). In fact, the original title of the paper submitted was "Myocardial Necrosis Produced by Aerosolized Catecholamines." It was the referee of the journal who recommended the present title and the change because he was concerned that a causation between aerosolized catecholamines and necrosis of the myocardium is suggestive but not necessarily proven. We certainly did not wish to imply a causative relationship between catecholamine-induced myocardial necrosis and myocardial infarction resulting from the atherosclerotic process and blood flow deprivation, although this is certainly a provocative model. Unfortunately, little or no clinical data are available on this topic.

Of importance, Dr. Flowers' comments provoke an important question on electrocardiographic terminology. In the daily reading of electrocardiograms, most electrocardiographers do use the term, myocardial infarction, in cases in which the patient does not have an infarction but rather has a disorientation of myocardial fibers, and/or myocardial necrosis, fibrosis, conduction block, etc. Should we change our terms and use a more electrophysiologic word (for example, "nonconductance of depolarization is present in a particular area") and then add in the report the clinical deductive commentary, "probably myocardial infarction." With the advent of computerized electrocardiology, we are now being forced into the use of a common terminology. Fortunately, with a computerized system, we can list multiple "clinical" possibilities and thereby separate the electrocardiographic wave-form analysis from the clinical implications. Of interest, a group has been formulated, encouraged by the Engineering Foundation meeting in New Hampshire in 1975, under the auspices of the Public Health Service to