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 wildly 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 electrocardiography, 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
Aneurysm was recorded as having entirely normal findings. On a progression of great-vessel connective-tissue abnormalities in some patients with gonadal dysgenesis.

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Turner's Syndrome and Aortic Aneurysm

To the Editor:

It is well known that about one-third of the patients with Turner's syndrome have coarctation of the aorta; however, only a relatively small number of patients with Turner's syndrome and coarctation of the aorta have been found to also have an aortic aneurysm. Patients with Turner's syndrome and aortic aneurysm but without coarctation of the aorta have been documented even less frequently.1,2 We would like to report another case of the latter type.

CASE REPORT

An 18-year-old white woman with an XO sex-chromosome constitution is being followed in the pediatric endocrinology clinic following the initiation of exogenous estrogen therapy. No cardiac murmur was noted prior to beginning the hormone regimen, and a cardiac evaluation seven years earlier was recorded as having entirely normal findings. On a return visit ten months after starting hormone supplementation (conjugated estrogens), the patient was noted to have a diastolic murmur consistent with aortic insufficiency. Her blood pressure was 115/75 mm Hg. The chest x-ray film was normal except for a dilated ascending aorta. The electrocardiogram was normal. An echocardiogram showed marked dilatation of the aortic root. Cardiac catheterization one month after the murmur was heard demonstrated marked dilatation of the ascending aorta, with only minimal aortic regurgitation. There was no evidence of aortic coarctation or a bicuspid aortic valve.

DISCUSSION

While it seems certain that hormones contribute to the regulation of connective-tissue metabolism,3,4 the precise influence of estrogen on connective tissue remains to be defined. The experience with our patient would lead us to ask whether exogenous estrogen therapy might trigger the development and progression of great-vessel connective-tissue abnormalities in some patients with gonadal dysgenesis.

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More Female Smokers: More Female Lung Cancer

To the Editor:

During the last year, there have been many discussions in Chest about smoking women and lung cancer. Recently, Wynder et al1 presented a retrospective study of 108 female lung-cancer patients during 1970 to 1972 and showed that cigarette smoking was closely associated with epidermoid and anaplastic lung cancer and less strongly with glandular types of lung cancer. As women adopt cigarette-smoking habits similar to those of men, these investigators propose that the death rates from lung cancer in women will continue to increase.

We recently have seen a heavy-smoking 19-year-old woman dying because of a small-cell anaplastic lung cancer.

CASE REPORT

Except for cystitis, this 19-year-old woman had always been healthy. She started to smoke at the age of 13 years and continued to smoke 20 to 30 cigarettes or more daily. During her last two years, the patient had a cough; and after a common cold in July 1974, her cough increased. In January 1974 the patient became pregnant, and in September she bore a healthy child. A chest x-ray film at that time showed a large tumor in the patient's left lung. At that time, she already had metastatic destruction in the spine. A chest x-ray film one year earlier was normal. Histopathologic and cytologic examination of material obtained by fiberoptic bronchoscopic techniques and fine-needle aspiration biopsy showed a small-cell anaplastic lung cancer. In spite of radiotherapeutic and cytostatic treatment, the patient died two months after the delivery of her child. Autopsy confirmed the diagnosis.