To the Editor:

A few thousand patients die of asthma each year in the United States, and Dr. Sears fears that an explanation may be found in his discovery that some patients with relatively mild asthma appear to suffer deterioration when they use β-adrenergic aerosols routinely. Nevertheless, it is evident that many millions of patients use routine β-adrenergic aerosol therapy, and there is good reason to believe that as a result they enjoy a comfortable life. I feel that there is a logical inconsistency in the extension of Sears's claims: he implies that patients with chronic, variable asthma are harmed by using standard daily doses of β-adrenergic therapy, but their resultant lapse into acute asthma can be safely managed by giving far greater doses of these same drugs. It is possible that the apparent harm of routine therapy is only of significance in a subgroup of patients such as those who use partially use meterol.

The established dogma of using routine β-agonist aerosol therapy is supported by the bulk of the literature and by a vast experience. Before aerosol steroids were introduced in the 1960s, children with asthma relied principally upon symptomatic treatment. With few exceptions, it appears that these children not only did well, but they "grew out" of their asthma. In the era of aerosol steroids, there is still enough documented experience to suggest that long-term β-agonist aerosol therapy given alone is beneficial in children and younger adults. The vision of deaths that Dr. Sears relates to the continuing daily use of β-agonists appears to be a mirage; most deaths occur in poorly compliant, psychologically disturbed, or economically disadvantaged patients with brittle asthma who fail to use their medications appropriately.

Fortunately, there is an important point that both sides can agree upon: without doubt, all drugs can be dangerous if used in excess. Thus, if a patient uses "excessive" doses of a β-adrenergic aerosol, there is a risk of an adverse outcome. The critical issue lies in the definition of "excessive": all parties might agree that this means "more than is symptomatically necessary." Two problems face us, however: (1) Do all symptoms (eg, wheezing on forced expiration as well as wheezing on effort) deserve treatment? (2) How should we interpret the term "p.r.n." If I hear an asthmatics wheeze, but he does not report dysnea on effort (perhaps because he makes no effort), I tend to advise treatment with β-adrenergic aerosol several times a day. Dr. Sears presumably would tend not to treat such a patient, unless the individual started exercising and became aware of dysnea. Thus, Dr. Sears advocates β-adrenergic aerosols only for airway obstruction that becomes symptomatic under special circumstances, whereas I favor treatment for all patients who present any evidence of reversible asthmatic bronchostenosis.

The division between the Sears camp and traditionalists such as myself may depend on inappropriately loose terminology, and our differences may be partially resolved if we could correlate the need to treat clinical disease with the presence, as Sears suggests, of objective quantified measures of disordered physiology. If we could establish a rational set of criteria for defining "p.r.n.," we might find that our different approaches are really very similar. Thus, it is probable that those asthmatic patients for whom I choose to not prescribe routine β-agonists are similar to those of Dr. Sears, whereas those for whom he does end up having to treat with routine daily "p.r.n." β-agonist therapy are similar to those whom I treat with two- to four-times-a-day therapy. There is an intermediate group for whom the establishment of an appropriate therapeutic regimem is a challenge for each of us. However, it is now important for the two apparently opposed views on asthma therapy to define a mutually acceptable approach to β-agonist dosing that covers the majority of patients with asthma—leaving room for disagreement on the remaining few.

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References


Prominent Elevation of the ST Segment by Convulsion

To the Editor:

Elevation of the ST segment on electrocardiography reflects the electrophysiologic state of injured myocardium, as in myocardial infarction, variant angina, and pericarditis. In acute myocardial infarction, the ST segment is elevated with a curve that is upwardly convex in the leads facing the infarction. An increase in serum creatine phosphokinase MB isoenzyme (CPK-MB) confirms the diagnosis. I report the case of a patient who showed a prominent elevation of the ST segment on ECG and an increase in serum CPK-MB, but had no evidence of myocardial infarction at autopsy.

A 66-year-old man with a history of symptomatic epilepsy due to brain infarction and chronic renal failure caused by bilateral renal stones was admitted to our hospital for acute pyelonephritis and exacerbation of renal function. The pyelonephritis did not respond to therapy and produced a high-grade fever, which was considered to be a trigger of general convulsion. The convulsion appeared repeatedly in spite of anticonvulsive drug infusion. Elevation of the ST segment was noticed on a monitored ECG 4 days after admission. A 12-lead ECG was then recorded immediately after an episode of general convulsion. The ECG showed a remarkably elevated ST segment, which was prominent in lead V1 (Fig 1). The shape of the ST segment and the lack of a Q wave were not identical with the appearance in acute myocardial infarction. Echocardiography during a convulsion did not show dyskinesis of the left ventricular wall.
There was also no associated chest pain. Serum chemistry study revealed increased total CPK (1,952 U/L), CPK-MB (221 U/L), glutamic oxaloacetic transaminase (51 IU/L), and lactate dehydrogenase (781 IU/L) and normal electrolyte levels, suggesting the occurrence of acute myocardial infarction. The patient died 5 days after admission, and the elevation of the ST segment persisted until immediately before his death.

An autopsy showed that bilateral acute pyelonephritis and perirenal abscess were the cause of death. No evidence of acute myocardial infarction, sclerosis, or obstruction of coronary arteries or recent occurrence of cerebrovascular diseases was observed at postmortem investigations.

Although elevation of the ST segment, except in ischemic heart disease, appears in acute pericarditis, with high serum potassium level, and after DC cardioversion, these conditions were clearly excluded by the clinical and autopsy findings. I assume that mechanical damage of the heart that occurred during a general convulsion may have been the primary cause of the remarkable and vast elevation of the ST segment on the ECG in this patient.

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Complications of Central Venous Catheterization

To the Editor:

I read with interest the article by Duntley et al., which appeared in the June 1992 issue of Chest. The authors presented clinical data on eight patients with complications caused by central venous catheterization. The authors attributed the respiratory and cardiovascular symptoms observed to vascular erosion and secondary perforation caused by these catheters and offered radiographs to support their hypothesis. I would like to discuss whether another mechanism might be more probable.

In at least five of the eight patients, multilumen catheters were inserted. In such patients, extrusion of central lines can be caused by inadequate fixation of the catheters and/or movements of the patients resulting in extravascular positioning of at least one lumen. Pleural effusions and mediastinal widening can also occur by this mechanism. I reviewed 469 of our ICU patients with central lines (218 double- or triple-lumen catheters) and detected 3 secondary dislocations of the multilumen catheters with similar radiographic signs. In our intensive care patients, diagnosis of outward displacement was confirmed by extravasation of radiographic contrast media infused through proximal ports. Contrast infusion through the distal port demonstrated an adequate intravascular position of the catheter tip in these three patients.

The authors did not give detailed information concerning which port was used to infuse the contrast media in their patients. Our findings suggest that the migration of at least one catheter lumen into an extravascular position, not vascular erosion and perforation, could be the cause of the symptoms described in the patients reviewed by Duntley et al.

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To the Editor:

We appreciate Dr. Bach's suggestion of an additional mechanism for the formation of hydrothoraces in patients with central venous catheters. We do not believe, however, that catheter withdrawal