Vigorous Cleaning of Inspirease Reservoir Devices

To the Editor:

The Inspirease Reservoir Device (Figure 1a), developed to maximize benefit from the metered dose inhaler, has become a very popular and useful adjunct for aerosol therapy. The design of this reservoir device 1) does not require coordination on the patient's part between firing the metered dose inhaler and inhaling, 2) offers the patient visual assurance that he is inhaling the aerosol as the bag collapses, 3) does not allow aerosol leakage to occur during rebreathing since the system has no opening to the outside, and 4) provides an audio cue when individuals inhale at inspiratory flow rates too fast for maximal deposition of aerosol in the lower respiratory tract. The plastic mouthpiece (Figure 1b) is equipped with two delicate horizontal plastic reeds that provide the audio cue.

Reservoir bag shows plastic in bag with two intact horizontal reeds. Mouthpiece valve with one broken and one missing reed. Medication Canister.

We would like to bring to the attention of the readers of Chest potential problems associated with the use of this device. During a one-month period of time, five patients came into our outpatient Pulmonary Clinic complaining that their Inspirease devices no longer provided an audio cue for rapid inhalation. Upon examining these devices, we noted the plastic reeds were either bent and/or broken (Fig 1c). Several of the reeds were completely missing. One of the reeds fell off in clinic after being barely touched with the point of a pencil. The patients who used these devices all related a history of vigorous, aggressive cleaning and drying of their devices with hot, strong running water or paper towels. While none of our patients, to our knowledge, sustained any adverse occurrence from the missing or broken reeds, the breakage of the reeds in the mouthpiece could place other patients at risk for two potential problems. First, the thin piece of plastic that breaks off could be inhaled; second, patients may be unaware that the audio cue is no longer functioning and may be improperly using the device.

Since the Inspirease Reservoir Device is an important adjunct in the treatment of patients requiring inhaled bronchodilator therapy, it is important to impress upon patients the importance of gently cleaning and air drying the device and to remind patients to visually inspect their devices for breakage on a regular basis.

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REFERENCES


Severe Reactive Airways Disease Induced by Propafenone

To the Editor:

Since the advent of propafenone as an antiarrhythmic drug, severe reactive airways disease has been a complication sporadically recognized as a side effect. After recent experience in a patient with severe spastic airways reaction in acute intoxication with propafenone, we read with great interest the article of Hill et al evaluating the asthmogenicity of propafenone.

The effect of propafenone may be attributed to a structural and functional resemblance to propanolol, a beta-adrenergic receptor antagonist. In acute intoxication, bronchodilator agents can be used to prevent airways reactivity. In subjects with mild intermittent asthma or chronic obstruction of airways, this drug should be used with caution at doses always less than to 450 mg/day.

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High Output Congestive Heart Failure in Septic Shock

To the Editor:

In their article entitled "Depressed Left Ventricular Performance: Response to Volume Infusion in Patients with Sepsis and Septic Shock", Ognibene et al present additional evidence that the human heart in septic shock is often dilated and near the end of its preload reserve despite normal filling pressures.\(^1\)\(^2\) Using data from their article, I have penned two hypothetical left ventricular compliance curves, one for their patients without infection and one for their patients in septic shock (Fig 1). As indicated by the arrow, the curve in septic shock is shifted upward and downward. On average, volume loading of patients in septic shock increased preload by 5 percent (5 ml/m\(^2\)) while pulmonary capillary wedge pressure (PCWP) increased 64 percent (6 mm Hg). Furthermore, none of the 21 patients in septic shock responded paradoxically to the increase in PCWP. Their fall in left ventricular end-diastolic volume suggests that volume loading produced an acute change in left ventricular compliance, shifting the curve upward and to the left. This shift is characteristic of tamponade physiology and could be explained by an increase in the size of the right ventricle inside a constraining pericardium (ventricular linkage). Previously, I postulated that ballooning of the right ventricle might explain the failure of the left ventricle to dilate in nonsurvivors of septic shock.\(^3\) Ognibene et al do not report right-sided parameters such as central venous pressure. Possibly there was a trend toward equalization of right and left end-diastolic pressures in the patients in septic shock.

The pathology of septic shock includes high output congestive heart failure. The cardiac output is elevated and filling pressures are normal because the acutely depressed heart is unloaded. The heart is indeed failing since, despite an adequate venous return (its ventricles over-filled under normal pressures), it is unable to increase its output sufficiently to restore adequate blood pressure. If peripheral vasodepression recovers before myocardial depression, the cardiac output falls and filling pressures rise since the performance of a failing ventricle is very sensitive to changes in afterload. Similarly, raising PCWP by volume infusion increases the afterload on the right ventricle, reduces its ejection fraction, increases its size and—through ventricular linkage—cancels the expected increase in left ventricular preload. Thus PCWP around 10 mm Hg may be optimal in septic shock, providing most of the preload available to the left ventricle without taxing the pulmonary bailing system. If at a venous pressure of 10 mm Hg pulmonary lymphatic flow is nearing the end of its reserve capacity because of capillary damage, a 60 percent increase in transudation might precipitate alveolar flooding, a dear price for a bit more preload. This view of the heart in septic shock blurs the distinction between cardiac and noncardiac pulmonary edema.

Both experimentally and clinically, a striking feature of septic shock is its failure to respond to fluid administration. In the past, hemorrhage and dehydration were said to cause "surgical shock" (for which volume expansion was crucial), whereas heart failure and infection were said to cause "medical shock" (in which volume expansion was detrimental). Some patients in septic shock do respond partially to volume loading, probably because they suffer from both medical and surgical shock (a status which can not be determined solely on the basis of cardiac filling pressures).

Assuming that they are dealing with a left ventricle whose compliance curve is not shifted downward, many physicians now

![Graph](image_url)

**Figure 1.** In septic shock, preload reserve is exhausted at normal filling pressures.