COMMUNICATIONS TO THE EDITOR

Communications for this section will be published as space and priorities permit. The comments should not exceed 500 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Right Atrial Mass Simulated Echocardiographically by a Swan-Ganz Catheter

To the Editor:

We would like to compliment Kendrick et al\(^1\) on their concise report of abnormal tricuspid valvular echoes associated with a redundant wire of a right ventricular pacemaker. Silverman et al\(^2\) described similar findings in patients with ventricular septal rupture after infarction. Correspondence\(^3\) following that article suggested that the echoes actually originated from an indwelling Swan-Ganz catheter. Charuzi et al\(^4\) also described “catheter echoes” that could be confused with several cardiac structures, including the anterior leaflet of the tricuspid valve.

Both Levisman\(^3\) and Charuzi et al\(^4\) emphasized the catheter as the sole source of the echoes. The report by Kendrick et al\(^1\) and our Figure 1 would support the theory that the abnormal echoes are the result of the catheter and the distorted motion of the anterior leaflet of the tricuspid valve caused by the catheter. These findings, which suggest an atrial mass or myomata, are not seen in all patients with a Swan-Ganz catheter because the motion of the anterior leaflet of the tricuspid valve is not distorted (perhaps by redundancy of the catheter) in all patients.

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REFERENCES


To the Editor:

We would like to thank Yarnal and Smiley for their interest in our article and the echocardiogram that they

Figure 1. Echocardiogram showing anterior leaflet of tricuspid valve (*thin arrow*) with increased D-E excursion, decreased E-F slope, and dense echoes posterior to it. Catheter was abruptly withdrawn (*thick arrow*) from pulmonary artery to right atrium while position of transducer and gain settings were unchanged. Motion of anterior leaflet of tricuspid valve is normal after withdrawal of catheter.
have submitted. It is a striking graphic confirmation of
the observation reported by us and others, ie, that cathe-
ters in the right side of the heart can appear as masses
behind the anterior tricuspid leaflet.

Marvin H. Kendrick, Jr., M.D., Concord, Mass
and Alfred F. Parisi, M.D., West Roxbury, Mass

Problems with Prolonged
Endotracheal Intubations

To the Editor:

We will attempt to answer Cash's¹ question concerning
long-term endotracheal intubations. His question
asked what damage is done to the vocal cords (during
long-term intubations of up to ten days).

We² have recently looked at this problem in a large
number of patients in intensive care units and have
compared the degree of laryngeal pathologic abnor-
malities in both oral and nasal endotracheal intubations.
Damage to the vocal cords occurs routinely following
both oral and nasal intubations. As alterations in the
microscopic surface are found to occur after two hours of
intubation and macroscopic changes after six hours, it is
apparent that laryngeal damage increases in severity as
the duration of intubation increases. The injuries to the
vocal cords that we observed during the five-day to nine-
day intubations (Fig 1 and 2) included posterior ulcer-
ations and craters of the vocal cords, maceration of the
vocal cords, ulceration of the arytenoid cartilage and cor-
cinate tubercle, ulceration of the glottic surface of
the epiglottis, ulceration of the proximal posterior tra-
cheal wall approximating the glottic opening, supra-
glottic edema, abnormalities of glottic closing, anterior
adhesions of the vocal cords, and laryngeal webs.

During our study, we carefully followed the develop-
ment of laryngeal injury using the fiberoptic bron-
choscope. Every 48 to 72 hours during intubation and
within 24 hours after extubation, the vocal cords and
surrounding laryngeal tissue were directly visualized. If
we believed that the severity of laryngeal injury would
cause prolonged phonation or functional impairment (or
both), we recommended that tracheostomy be per-
formed by our surgical colleagues. In long-term follow-
up examinations, we have found that the majority of the
laryngeal injuries will heal within a period of two weeks
to approximately two months. Our major concern has
been with the loss of functional integrity of the laryngeal
apparatus, which may result in acute problems of aspira-
tion within 48 hours following extubation or in a chronic
process of aspiration.

We found that the route of intubation, as well as the
duration, appeared to be a significant factor in establish-
ing the severity of laryngeal injury. Nasal endotracheal
tubes produced approximately 50 percent less laryngeal
injury than oral endotracheal tubes. We believe that
nasal tubes produce more stability and are less affected
by the patient's movement of his head. Nasal endo-
tracheal tubes are usually 0.5 to 1 mm smaller in diame-
ter than oral endotracheal tubes and, therefore, produce
less laryngeal injury because of their size. The majority
of patients intubated in our study exhibited ulceration
and cratering, which is due to the posterior force that the
endotracheal tube exerts on the posterior laryngeal
apparatus. This pressure is caused by the lever action of
the tube as it bends over the tongue in an oral intubation
and over the palate in a nasal intubation.

We had hypothesized that anatomic variants involving
the length of the hard palate, the portion of the first
cervical vertebrae, and other factors might be important
in explaining the variability of damage seen in patients;
however, despite careful cephalometric analysis of 17
patients, we could detect no certain aggravating factors.

Although laryngeal damage was found after all pro-
longed intubations, our study demonstrated long-term
oral endotracheal intubations produced significantly
greater laryngeal injury than nasal intubations. There-

![Figure 1. Injury to vocal cords.](image1)

![Figure 2. Injured larynx, showing evidence of ulceration, cratering, and maceration of vocal cords and cartilaginous edema.](image2)