

Communications

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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 Catheter was abruptly withdrawn (thick arrow) from pulmonary artery to right atrium while position of transducer and gain settings were unchanged.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.

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CHEST, 74: 4, October, 1978
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 abnormalities 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 ulcerations and cratering of the vocal cords, maceration of the vocal cords, ulceration of the arytenoid cartilage and corniculate tubercle, ulceration of the glottic surface of the epiglottis, ulceration of the proximal posterior tracheal 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 development of laryngeal injury using the fiberoptic bronchoscope. 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 performed 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 aspiration 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 establishing 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 endotracheal tubes are usually 0.5 to 1 mm smaller in diameter 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 position 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 prolonged intubations, our study demonstrated long-term oral endotracheal intubations produced significantly greater laryngeal injury than nasal intubations. There-

Figure 1. Injury to vocal cords.

Figure 2. Injured larynx, showing evidence of ulceration, cratering, and maceration of vocal cords and cartilaginous edema.