Prolonged Endotracheal Intubation

To the Editor:

It is common practice to perform a tracheostomy on patients who require prolonged endotracheal intubation in order to avoid laryngeal damage. There appears to be no argument regarding the need for tracheostomy when a patient must be cared for outside a special care unit. On the other hand, prolonged nasal or oral intubation avoids the disadvantages associated with tracheostomy such as operative intervention and microbial colonization of the wound. However, the upper limits for safe endotracheal intubation have not been established.

Before the development of soft cuff implant tested materials, laryngeal damage resulted from prolonged endotracheal intubation.1,2 Recent experience from a number of sources indicates that modern tubes are well tolerated.3-5

We have examined by fiberoptic bronchoscopy, 21 patients who had been treated by endotracheal intubation for respiratory insufficiency, usually due to pneumonia. They were intubated on 26 occasions with soft cuff tubes, usually of the Lanz variety. The average duration of intubation was 14.6 days, range four to 71 days. Three patients were intubated 27 or more days without evidence of pathology at the time of extubation. Fifteen procedures produced no visible pathology (average 14.0 days, range six to 31 days). Eleven intubations were followed by mild changes varying from minimal hyperemia and edema to small superficial ulcerations (average 15.4 days, range four to 71 days). The latter complication occurred in only three instances and seems unrelated to the duration of intubation as the average was 8.6 days, compared to 14.6 days for all procedures. Pathologic changes appeared to occur at the vocal cords, balloon site, and rarely, at the tip of the tube. There were no severe complications such as tracheomalacia, deep ulceration or stricture, commonly associated with the older tubes.

Although nasal tubes appeared to be more comfortable to patients, they did not appear to cause less injury than oral tubes. Both tubes tended to produce pathologic changes at pressure points.

All of the abnormalities which we observed following prolonged endotracheal intubation appeared to be mild enough to resolve completely. There were no prolonged adverse clinical sequela. Apparently, ischemia due to pressure, sometimes aggravated by shock, is the most important cause of tracheomalacia and stenosis. Modern tubes such as the Lanz are not easily hyperinfated and are not likely to cause ischemia.3

In most hospitals, it is not difficult to maintain patients on endotracheal tubes, as specialized personnel can easily change the tubes when necessary. Tracheostomy under these circumstances seems to offer little advantage except avoidance of laryngeal lesions which, in our experience, are not serious. We agree that tracheostomy should be employed in all patients who require prolonged intubation where specialized assistance is not available. However, where specialized assistance is available, prolonged endotracheal intubation appears to be safe.

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A New Observation on Papillary Muscle Dysfunction

To the Editor:

The systolic murmur of papillary muscle dysfunction is variable and is frequently associated with a loud first heart sound (S1), a fourth heart sound (S4), and third heart sound (S3), especially in the absence of heart failure.1,2 A characteristic feature of the murmur is its post-extrasystolic diminution.1,2 There is usually a post-extrasystolic accentuation of the third heart sound.3 To our knowledge, post-extrasystolic diminution or disappearance of the third heart sound (S3) in papillary muscle dysfunction has not been previously reported. I describe a patient with this rare and interesting finding.

A 69-year-old man, with a previous inferior myocardial infarction, was admitted with acute anterior myocardial infarction. Physical examination revealed: pulse rate 75/min; BP 130/75 mm Hg; loud first heart sound; third and fourth heart sounds and a grade 3 apical crescendo-decrescendo systolic murmur. Post-extrasystolic diminution in intensity and duration of systolic murmur and disappearance of third heart sound were observed (Fig 1). There was no evidence of heart failure. During hospitalization, S3 became normal and systolic murmur and S4 disappeared, but S4 persisted.
Starch as a Cause of Thrombus with Swan-Ganz Catheters

To the Editor:

Bradway et al (Letter to Editor, Chest 1981; 80:335-36)

cite an appreciable incidence of internal jugular venous thrombosis secondary to temporary cannulation with a Cordis sheath and Swan-Ganz catheter. An observation made in our institution is worthy of note.

A 32-year-old woman with incomplete endocardial cushion defect and mild pulmonary hypertension underwent corrective surgery. After induction of anesthesia, a Swan-Ganz catheter was placed through a Cordis sheath introduced into the internal jugular vein. Upon opening the right atrium, a segment of the catheter was seen coated by a thin layer of products of coagulation. The coagulum was examined microscopically and found to contain refractile elements consistent with starch particles, mixed with fibrin, platelets and cellular debris.

We postulated that powder from the anesthesiologist’s gloves, retained on the catheter, incited coagulation on the otherwise “inert” surface. Because of this observation, we have wiped the operator’s gloves with a damp sponge to remove all powder. Since instituting this precaution in numerous cases of repair of congenital lesions when a Swan-Ganz catheter was observed intraoperatively, no clot has been observed.

Attention to elimination of starch from glove surfaces might decrease the incidence of intravascular thrombosis.

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To the Editor:

Drs. Brunswick and Gionis report a case in which clots containing starch particles were found adherent to a Swan-Ganz pulmonary artery line intraoperatively. Although anecdotal, this case implies an interesting concept, which to our knowledge has not previously been addressed: that powder from the operators’ gloves might contaminate the Swan-Ganz catheter and subsequently stimulate clot formation in vitro.

Thrombogenesis of the Swan-Ganz pulmonary artery catheter has been demonstrated previously,1 and recently the use of heparin-bound catheters, as reported by Hoar and associates,2 appears to reduce the thrombogenicity of pulmonary artery catheters, although long-term use has not been studied. It is interesting that Hoar’s method of insertion of the Swan-Ganz catheter does not routinely involve rinsing the operators’ gloves prior to placement.3 Although the thrombogenicity of such catheters has been attributed to the surface properties of these catheters,4 the role, if any, of glove powder in stimulating thrombosis remains undefined.

At one time considered uncommon,5 the incidence of indwelling catheter-related central venous thrombosis has been reported by us and others6-10 at an alarming rate recently. The pathogenesis is not known. Several potential factors favoring thrombogenesis include: venous wall injury from venipuncture technique, the intrinsic thrombogenic properties of the catheter itself, and diminished venous blood flow either from mechanical obstruction by the catheter or the altered hemodynamics of the patient. What role, if any, contamination of catheters with powder plays in the pathophysiology of catheter associated central venous thrombosis, remains speculative but intriguing. Although a cause-and-effect relationship is not proven by Brunswick and Gionis’ case, the simple act of rinsing one’s gloved hands intraoperatively is easy to perform. Perhaps a

FIGURE 1

A radionuclide angiogram revealed hypokinesia of apicoinferior segment and an ejection fraction of 53 percent (normal 65 ± 8 percent).

Postextrasystolic compliance of the left ventricle remains unchanged,4 and cannot be the explanation for postextrasystolic accentuation or disappearance of the third heart sound. Postextrasystolic potentiation of contractile strength of ischemic but viable papillary muscle and myocardium, increased diastolic filling and reduction in afterload because of declining aortic pressure during the compensatory pause causes increased forward flow and decreased backward regurgitation. More complete emptying of the left ventricle results in a smaller postextrasystolic end-systolic volume and decreased backward regurgitation results in less ventricular filling from left ventricle during diastole. The compensatory pause also allows increased diastolic time for coronary blood flow and may be responsible for a transient decrease in myocardial ischemia. Whether the postextrasystolic S3 remains or disappears depends upon the degree of papillary muscle dysfunction, the overall left ventricular contractility, and the degree of its postextrasystolic potentiation.

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