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.

Richard A. Brunswick, M.D., F.C.C.P.,
Director, Children’s Cardiovascular Services; and
Thomas A. Gionis, M.D.,
Chief Resident, Tulane University,
School of Medicine, New Orleans

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 vivo.

Thrombogenesis of the Swan-Ganz pulmonary artery catheter has been demonstrated previously, and recently the use of heparin-bound catheters, as reported by Hoar and associates, appears to reduce the thrombogenicity of pulmonary artery catheters, although longterm 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. Although the thrombogenicity of such catheters has been attributed to the surface properties of these catheters, the role, if any, of glove powder in stimulating thrombosis remains undefined.

At one time considered uncommon, the incidence of indwelling catheter-related central venous thrombosis has been reported by us and others 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
prospective double-blind animal study of the thrombogenesis of this powder might clarify the issue at hand.

William R. Bradway, D.O.; and James C. Giudice, D.O., F.C.C.P., UMDNJ-New Jersey School of Osteopathic Medicine, Stratford, New Jersey

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3. Hoar PF. Personal communication, February 16, 1982

Restrictive Defect in Klinefelter's Syndrome

To the Editor:

Huseby and Petersen's (Chest 1981; 80:31-33) observation that nine of 24 patients with Klinefelter's syndrome had a restrictive defect on pulmonary function studies is of particular interest to us.

In studying pulmonary function tests in nine patients with karyotype-proven Klinefelter's syndrome (Table 1), we found a restrictive defect in five patients (four had TLC less than 80 percent of predicted and one had VC less than 85 percent with a normal FEV1/FVC ratio). Unlike Huseby and Petersen, we did not observe a reduced FRC in those without a restrictive defect. Our nine patients, all white, ranged in age from 28 to 61 years. All smoked and six were symptomatic. There was no obvious explanation for the restrictive defect observed in five patients, as none had chest wall abnormalities and only two exceeded their ideal body weight by more than 20 percent. One patient had a PiMS phenotype and a serum alpha1-antitrypsin level of 100 mg percent.

These results, published as an abstract in 1976, the first time, pointed out the increased incidence of restrictive defect in Klinefelter's syndrome. Prior to 1976, there were two patients with Klinefelter's syndrome and restrictive defect described, but both had other diseases that could account for the defect.

Combining data from Huseby and Petersen's study and our data, it is noted that 14 of 33 patients with Klinefelter's syndrome had a restrictive defect. We wish to re-emphasize that restrictive defect, not obstructive defect as implied in an earlier report, is the predominant finding in patients with Klinefelter's syndrome.

Basil Varkey, M.D., F.C.C.P., and Akira Funahashi, M.D., F.C.C.P., Pulmonary Section, Veterans Administration Medical Center, Wood and Medical College of Wisconsin, Milwaukee

REFERENCES


Table 1—Patient Data*

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*Volumes and Dco expressed as percent predicted.