

## CASE REPORTS

250,000 fiberoptic gastroscopies, a 10% mortality was observed in surgically treated perforations as compared to 37% in medically treated perforations,<sup>4</sup> and mortality decreased from 56% to 0% when intubation-related esophageal perforations were treated by early (less than 12 h) surgical intervention.<sup>7</sup>

Surgical intervention includes the local repair at the site of perforation and, most importantly, the separation of the perforation site at the neck and the primary site of operation, the mediastinum. This separation of operation fields is of prime importance, because it increases the chances that a potential infectious process may be limited to the neck rather than expanding into the mediastinum.

The NPO regimen until radiologic confirmation of consolidation of the local repair and absence of a fistula originating from the site of perforation is important, because penetration of food particles into a fistula could induce major infectious complications.

Interviewing patients on swallowing difficulties can alert the anesthesiologist to a yet unknown narrowing of the orogastric pathway and potentially prevent future perforations by TEE probes. Also, inserting the TEE

probe under direct vision using the laryngoscope is advisable should resistance be encountered at the initial attempt of blind insertion of the TEE probe.

## References

1. Daniel WG, Erbel R, Kasper W, Visser CA, Engberding R, Sutherland GR, Grube E, Hanrath P, Maisch B, Dennig K: Safety of transesophageal echocardiography: A multicenter survey of 10,419 examinations. *Circulation* 83:817-821, 1991
2. Seward JB, Khandheria BK, Oh JK, Freeman WK, Tajik AJ: Critical appraisal of transesophageal echocardiography: Limitations, pitfalls, and complications. *J Am Soc Echocardiogr* 5:288-305, 1992
3. Dawson J, Cockel R: Oesophageal perforation at fiberoptic gastroscopy. *Br Med J* 283:583, 1981
4. Miller G: Komplikationen bei der Endoskopie des oberen Gastrointestinaltraktes. *Leber Magen Darm* 17:299-304, 1987
5. Silvis SE, Nebel O, Rogers G, Sugawa C, Mandelstam P: Endoscopic complications: Results of the 1974 American Society for Gastrointestinal Endoscopy Survey. *JAMA* 235:928-930, 1976
6. Konstadt S, Reich DL, Thys DM, Aronson S: Transesophageal echocardiography, Cardiac Anesthesia. Edited by Kaplan JA. Philadelphia, WB Saunders, 1993, pp 342-385
7. Dubost C, Kaswin D, Duranteau A, Jehanno C, Kaswin R: Esophageal perforation during attempted endotracheal intubation. *J Thorac Cardiovasc Surg* 78:44-51, 1979

Anesthesiology  
82:583-586, 1995  
© 1995 American Society of Anesthesiologists, Inc.  
J. B. Lippincott Company, Philadelphia

## Early Thrombus Formation on Heparin-bonded Pulmonary Artery Catheters in Patients Receiving Epsilon Aminocaproic Acid

Mark E. Dentz, M.D.,\* Thomas F. Slaughter, M.D.,† Jonathan B. Mark, M.D.‡

ADMINISTRATION of antifibrinolytic drugs to prevent bleeding has become an increasingly widespread prac-

tice during cardiac surgery.<sup>1,2</sup> The potential for antifibrinolytic therapy to promote pathologic thrombosis in the perioperative period remains unclear. A recent report describes the rapid formation of thrombus on a heparin-bonded pulmonary artery catheter after aprotinin administration.<sup>3</sup> To date, no reports have described this complication in association with the synthetic antifibrinolytics (*i.e.*, epsilon aminocaproic acid and tranexamic acid). We describe two recent cases during which large thrombi were detected on heparin-bonded pulmonary artery catheters shortly after administration of epsilon aminocaproic acid and the subsequent precautions we have taken to minimize the risk of this complication recurring with the administration of antifibrinolytic therapy.

\* Associate.

† Assistant Professor.

‡ Associate Professor.

Received from the Department of Anesthesiology, Duke University Medical Center and the Durham Veterans' Affairs Medical Center, Durham, North Carolina. Submitted for publication May 23, 1994. Accepted for publication September 20, 1994.

Address reprint requests to Dr. Dentz: Department of Anesthesiology, Duke University Medical Center, Durham, North Carolina 27710.

Key words: Coagulation, thrombus: epsilon aminocaproic acid. Equipment: pulmonary artery catheter.



## Case Reports

## Case 1

A 42-yr-old, 86-kg man was scheduled for coronary artery bypass graft surgery. The patient received heparin for unstable angina until 3 days before surgery. Preoperative values for prothrombin time, activated partial thromboplastin time, and platelet count were normal. Before induction of anesthesia, an 8.5-Fr introducer catheter (SI-09880, Arrow International, Reading, PA) was placed into the right internal jugular vein, followed by a 7.5-Fr thermodilution, heparin-coated pulmonary artery catheter (93A-831H-7.5F, Baxter Healthcare, Irvine, CA). Before incision, epsilon aminocaproic acid was administered through the side port of the introducer catheter (10 g over 30 min), followed by a continuous infusion of 2 g/h for 5 h. Anesthesia consisted of midazolam, fentanyl, and pancuronium. After tracheal intubation, a biplane transesophageal echocardiography probe (Hewlett Packard, Andover, MA) was placed for intraoperative monitoring. At this time (20 min after pulmonary artery catheterization and 15 min after initiation of the epsilon aminocaproic acid infusion), a  $1 \times 1.5$ -cm echo-dense, freely mobile mass with fimbriated edges adherent to the pulmonary artery catheter was observed in the right atrium (figs. 1 and 2). Before aortic cannulation, 300 U/kg porcine heparin was administered *via* the side port of the introducer catheter. Activated coagulation time was maintained for more than 480 s. Cardiopulmonary bypass proceeded uneventfully. After cardiopulmonary bypass, the right atrial mass remained adherent to the pulmonary artery catheter as detected by transesophageal echocardiography. After administering protamine, the pulmonary artery catheter was removed during aspiration of the introducer catheter side port. Several soft dark red thrombi were recovered (fig. 3). A second pulmonary artery catheter was placed, and no subsequent thrombus formation was observed by transesophageal echocardiography. The patient's postoperative course was unremarkable, and the patient was discharged home on postoperative day 11.

## Case 2

A 73-yr-old, 54-kg man with a history of chronic obstructive pulmonary disease and angina pectoris was scheduled for elective cor-



Fig. 1. Intraoperative transesophageal echocardiographic image of pulmonary artery catheter thrombus.

Anesthesiology, V 82, No 2, Feb 1995

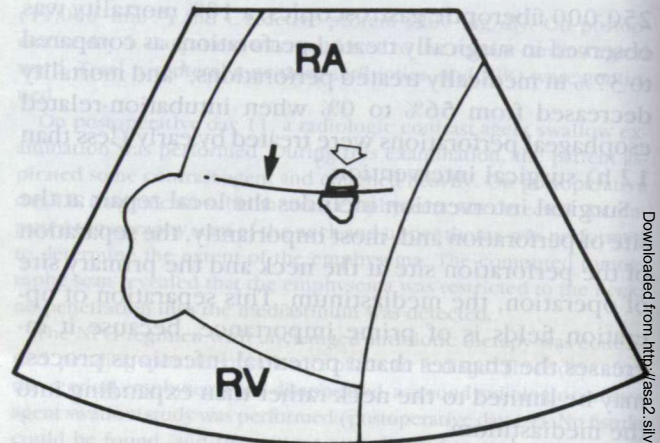


Fig. 2. Diagrammatic representation of figure 1. RA = right atrium, RV = right ventricle; open arrow = mass; filled arrow = catheter.

onary artery bypass graft surgery. A heparin-coated pulmonary artery catheter (93A-831H-7.5F, Baxter Healthcare) was inserted through an introducer catheter in the right internal jugular vein. Ten grams of epsilon aminocaproic acid was administered through the side port of the introducer catheter over 30 min, followed by an infusion of 1 g/h for 5 h. General anesthesia consisted of fentanyl and midazolam. After tracheal intubation, an Omniplane transesophageal echocardiographic probe was placed for intraoperative monitoring. Heparin (300 U/kg) was administered before aortic cannulation resulting in an activated coagulation time of 488 s. Cardiopulmonary bypass and myocardial revascularization proceeded uneventfully. At the conclusion of cardiopulmonary bypass, 160 mg intravenous protamine was given to neutralize the heparin. Examination by transesophageal echocardiography revealed an echodense freely mobile mass ( $0.3 \times 1.5$  cm) attached to the pulmonary artery catheter in the right atrium. Repeat examination by transesophageal echocardiography 45 min later failed to reveal the mass previously identified on the pulmonary artery catheter. The patient's postoperative course was complicated by supraventricular dysrhythmias and pulmonary lobar atelectasis; however, on postoperative day 16, the patient was discharged home in good condition.

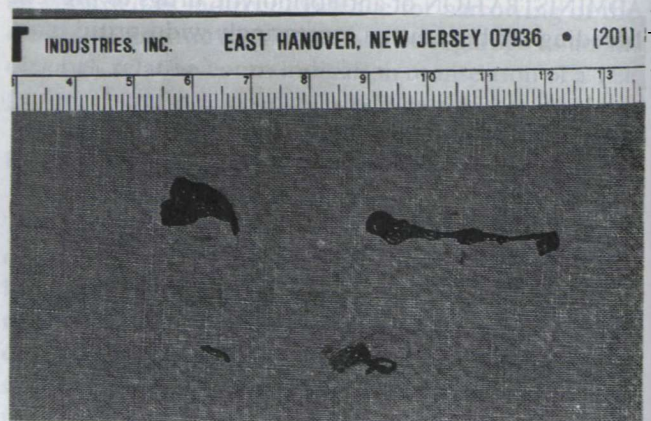


Fig. 3. Aspirated thrombi.



## Discussion

Before the development of heparin-bonded catheters, clot formation on pulmonary artery catheters frequently occurred and occasionally caused serious complications.<sup>4,5</sup> Heparin bonding has been demonstrated to prevent or at least significantly delay the formation of catheter-associated clots.<sup>6</sup> Whether pharmacologic therapy aimed at reducing blood loss during heart surgery will increase the risk of clot formation on pulmonary artery catheters remains unclear. Bohrer *et al.* previously reported an association between high-dose aprotinin and pulmonary artery catheter thrombosis<sup>3</sup>; however, no reports of pulmonary artery catheter thrombosis after therapy with epsilon aminocaproic acid have appeared in the literature. The two cases reported here suggest that epsilon aminocaproic acid may be associated with early clot formation on pulmonary artery catheters.

Epsilon aminocaproic acid is a synthetic lysine analog with a plasma half-life of 90 min.<sup>7</sup> The mechanism of action of synthetic antifibrinolytic drugs occurs through competitive inhibition of the lysine binding sites of plasminogen and plasmin, thereby preventing the formation of a complex with fibrin.<sup>8</sup> Numerous investigations have demonstrated a hemostatic effect after the administration of epsilon aminocaproic acid before cardiac surgery. To suppress fibrinolytic activity, Hardy *et al.* recommended administering epsilon aminocaproic acid as a 100–150-mg/kg loading dose, followed by a continuous infusion of 10–15 mg·kg<sup>-1</sup>·h<sup>-1</sup>.<sup>8</sup> However, the minimal effective dose for fibrinolytic suppression during cardiac surgery remains unclear. Prior investigations suggest that antifibrinolytic therapy must be administered before cardiopulmonary bypass to achieve an optimal hemostatic effect.<sup>1,9</sup>

The major concern related to antifibrinolytic therapy remains the theoretical risk of promoting pathologic thrombus formation in the perioperative period. Despite heparin administration, coronary artery bypass surgery generates increased prothrombin activation and fibrin formation.<sup>10</sup> Concomitant fibrinolytic activity may provide a protective mechanism by regulating the extent of thrombus formation.<sup>11</sup>

We hypothesize that the pulmonary artery catheter thrombosis observed in these patients was related to high localized concentrations of epsilon aminocaproic acid achieved around the pulmonary artery catheter during administration of epsilon aminocaproic acid through the side port of the introducer catheter. Subsequent to these cases, we altered our practice by in-

fusing epsilon aminocaproic acid through a peripheral intravenous catheter after heparin administration before initiation of cardiopulmonary bypass. Administration of epsilon aminocaproic acid after heparin may decrease the risk of thrombus formation. Van Riper *et al.* demonstrated that antifibrinolytic therapy may be administered after heparin with no loss of hemostatic activity.<sup>12</sup> During the 6 months after our alteration in the administration of epsilon aminocaproic acid, no subsequent episodes of pulmonary artery catheter thrombosis have been observed by intraoperative transesophageal echocardiography.

In summary, this report describes two cases of early thrombus formation on heparin-bonded pulmonary artery catheters in patients receiving epsilon aminocaproic acid during cardiac surgery. We speculate that administration of epsilon aminocaproic acid into the central circulation before administration of heparin increases the risk of pulmonary artery catheter thrombosis. Although antifibrinolytic therapy has proved highly efficacious in reducing bleeding after cardiac surgery, additional investigations are needed to determine the potential risks for perioperative thrombosis.

## References

1. Del Rossi AJ, Cernaianu AC, Botros S, Lemole GM, Moore R: Prophylactic treatment of postperfusion bleeding using EACA. *Chest* 96:27–30, 1989
2. Horrow JC, Hlavacek J, Strong MD, Collier W, Brodsky I, Goldman SM, Goel IP: Prophylactic tranexamic acid decreases bleeding after cardiac operations. *J Thorac Cardiovasc Surg* 99:70–74, 1990
3. Bohrer H, Fleischer F, Lang J, Vahl C: Early formation of thrombi on pulmonary artery catheters in cardiac surgical patients receiving high-dose aprotinin. *J Cardiothorac Anesth* 4:222–225, 1990
4. Hoar PF, Stone JG, Wicks AE, Edie RN, Scholes JV: Thrombogenesis associated with Swan-Ganz catheters. *ANESTHESIOLOGY* 48:445–447, 1978
5. Yorra FH, Oblath R, Jaffe H, Simmons DH, Levy SE: Massive thrombosis associated with use of the Swan-Ganz catheter. *Chest* 65:682–684, 1974
6. Hoar PF, Wilson RM, Mangano DT, Avery GJ, Szarnicki RJ, Hill JD: Heparin bonding reduces thrombogenicity of pulmonary-artery catheters. *N Engl J Med* 305:993–995, 1981
7. Verstraete M: Clinical application of inhibitors of fibrinolysis. *Drugs* 29:236–261, 1985
8. Hardy JF, Desroches J: Natural and synthetic antifibrinolytics in cardiac surgery. *Can J Anaesth* 39:353–365, 1992
9. Modell AI, Hallman GL, Bloodwell RD, Beall AC, Yashar JJ, Cooley DA: Epsilon-aminocaproic acid for bleeding after cardiopulmonary bypass. *Ann Thorac Surg* 11:577–582, 1971
10. Slaughter TF, LeBleu TH, Douglas JM, Leslie JB, Parker JK, Greenberg CS: Characterization of prothrombin activation during cardiac surgery by hemostatic molecular markers. *ANESTHESIOLOGY* 80:520–526, 1994



11. Nossel HL: Relative proteolysis of the fibrinogen B beta chain by thrombin and plasmin as a determinant of thrombosis. *Nature* 291:165-167, 1981

Anesthesiology

82:586-587, 1995

© 1995 American Society of Anesthesiologists, Inc.

J. B. Lippincott Company, Philadelphia

## Tension Pneumothorax Presenting as Ischemia of the Hand

Lynda T. Wells, M.D., F.R.C.A., Teodulo Aves, M.D.

PNEUMOTHORAX, a recognized complication of central venous catheterization especially when the subclavian route is used, can result in rapid collapse of the ipsilateral lung, mediastinal shift, and cardiovascular collapse.

We report a case of iatrogenic tension pneumothorax under general anesthesia presenting as ischemia of the contralateral hand.

### Case Report

A 51-yr-old woman who had a subarachnoid hemorrhage was brought to the operating room for craniotomy and clipping of an intracerebral aneurysm. Bilateral ulnar artery circulation was confirmed by Allen's test and palpation of ulnar artery pulses. Noninvasive monitoring was commenced, and anesthesia induced with 4  $\mu\text{g}/\text{kg}$  fentanyl, 2 mg/kg propofol, and 0.1 mg/kg vecuronium. Tracheal intubation was performed without difficulty. The lungs were ventilated mechanically with oxygen and nitrous oxide 50:50 and 0.2% isoflurane. A 20-G cannula was placed in the left radial artery. Several attempts by the surgical team to cannulate the right subclavian vein failed. The right internal jugular vein was cannulated at the first attempt. Throughout the case, hemoglobin oxygen saturation ( $\text{SpO}_2$ ) was 100% with unchanged cardiovascular parameters (blood pressure (BP) 110/65-130/80 mmHg, pulse rate 80-90/min). End-expired carbon dioxide ( $\text{ETCO}_2$ , 26 mmHg) and other ventilatory parameters (tidal volume ( $\text{Vt}$ ) 620-670 ml; peak inspiratory pressure ( $\text{Pip}$ ) 25-27  $\text{cmH}_2\text{O}$ ) remained unchanged. Chest radiography was performed to check the position of the central venous catheter.

Five minutes after radiography and 20 min after attempts to cannulate the right subclavian vein were abandoned, the  $\text{SpO}_2$ , which

12. Van Riper DF, Horrow J, Strong MD, Malik BA: Tranexamic acid is hemostatic when administered only during heparinization (abstract). *ANESTHESIOLOGY* 79:A93, 1993

was being monitored *via* a probe on the left index finger, decreased to 95%, and the arterial blood pressure trace became damped. The left hand was noted to be pale, cold, and without circulation, although perfusion above the wrist appeared unchanged. No changes in perfusion were observed in the right arm. The oximeter probe was replaced on the index finger of the right hand.  $\text{SpO}_2$  remained at 95%. Chest auscultation revealed diminished breath sounds on the right side. Depth of insertion of the endotracheal tube was unchanged.  $\text{ETCO}_2$ , and ventilatory parameters continued unchanged.

Six minutes after radiography, the chest radiograph became available. It showed a large right-sided pneumothorax (70%) with significant mediastinal shift to the left (fig. 1). Additionally, the heart shadow was displaced cephalad. Immediately, nitrous oxide was dis-

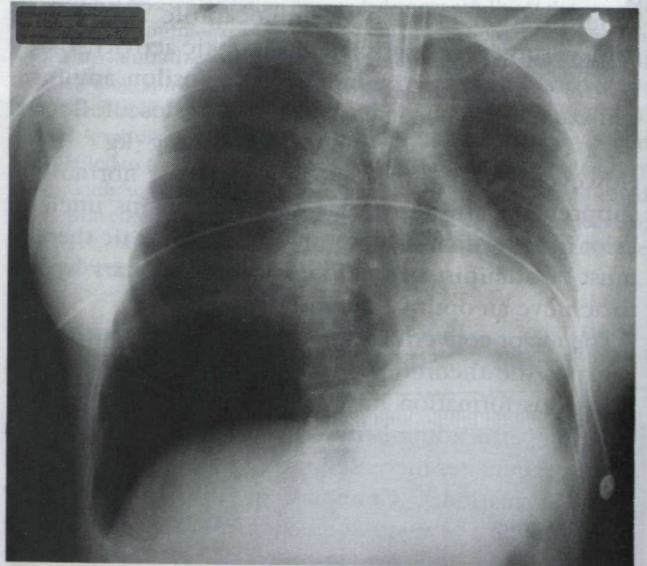


Fig. 1. A large right-sided pneumothorax with shift of the heart shadow into the left hemithorax and cephalad displacement such that the aortic knuckle lies behind the lower edge of the medial end of the clavicle. This would produce a mechanical distortion of the left subclavian artery in the region of the clavicle and first rib. The endotracheal tube is shifted to the left. The right hemidiaphragm is displaced downward. The patient has scoliosis with deviation of the thoracic spine to the right.

Received from the Department of Anesthesiology, University of Texas Health Science Center at Houston, Houston, Texas. Submitted for publication May 5, 1994. Accepted for publication October 3, 1994.

Address reprint requests to Dr. Wells: Department of Anesthesiology, University of Texas Health Science Center at Houston, 6431 Fannin Street, 5.020 MSMB, Houston, Texas 77030.

Key words: Complications: ischemia; pneumothorax.