cause cardiac dysrhythmias, especially in digitalized patients receiving digitalis. Pancuronium has a vagolytic effect,\textsuperscript{2,3} a direct sympathomimetic effect,\textsuperscript{9} an indirect sympathomimetic effect by increased release of norepinephrine,\textsuperscript{10} and by preventing reuptake of norepinephrine\textsuperscript{11–13} results in increased circulating catecholamine levels.\textsuperscript{14} This may be the cause for increased incidence of dysrhythmias following pancuronium administration in our group of patients who were receiving digitalis.

In this clinical investigation, the doses of muscle relaxants used are much smaller than one would use during rapid sequence induction. Added to this, during rapid sequence induction, the patients are not ventilated for about 45 s to 1 min during which time mild hypercarbia occurs. Thus, if these relaxants are used in rapid sequence induction in patients receiving digitalis, the effects on cardiovascular system might be more profound than those reported here.

In conclusion, we found that succinylcholine is associated with a lower incidence of dysrhythmias than pancuronium in patients receiving digitalis. Thus, caution should be exercised in administering pancuronium to such patients. Succinylcholine need not be withheld in patients receiving digitalis requiring the relaxant for rapid sequence endotracheal intubation.

REFERENCES


Acute Pulmonary Embolism during Therapeutic Arterial Embolization with Silicone Fluids

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Arterial embolization with gelfoam, silicone spheres, polyvinyl alcohol foam (PVA), isobutyl-2-cyanoacrylate (IBCA), or silicon fluid mixtures, has been used clinically to terminate the blood supply of arteriovenous malformations (AVM).\textsuperscript{1–6} Serious pulmonary embolization with this method usually does not occur\textsuperscript{1,7} because most of the embolizing substance is trapped within the malformation leaving only a small quantity to reach the pulmonary circulation.\textsuperscript{1,8} However, we describe a patient who developed severe pulmonary embolism with silicone liquid during this procedure.

REPORT OF A CASE

A 27-year-old woman was admitted for embolization of an arteriovenous malformation (AVM) of the right thigh. She first noted discoloration over the right calf 13 years ago. This gradually increased in size leading to ulceration and recurrent bleeding. Angiography at that time demonstrated an extensive AVM of the right leg. Two un-
successful surgical attempts were made to control the malformation eight and ten years prior to this admission. A right above-knee amputation was done seven years ago because of a chronic bleeding ulcer over the lesion. One year ago, she developed an ulcer over the stump. The thigh during the present admission appeared swollen. A non-healing ulcer, 3 x 2 cm in size, was observed over the stump. Angiographic studies showed a large femoral AVM of the thigh. Surgical intervention was thought not possible because of the size of the lesion. Intraarterial embolization of the AVM was contemplated in place of surgery. Because the patient wanted to be asleep during the procedure, general anesthesia was chosen. The patient had no other medical problems. Preoperative laboratory values were within normal limits. The ECG was normal and the chest roentgenogram showed normal lung fields and mild cardiomegaly probably resulting from chronic, high cardiac output caused by the fistula. Analysis of arterial blood gases were not done preoperatively. The patient was brought to the neuroradiology suite unanesthetized. Her blood pressure was 120/70 mmHg and heart rate 80/min. Thiopental, 500 mg, and succinylcholine, 80 mg, were given iv and an orotracheal tube inserted without difficulty. Halothane 0.6% and N2O 70% were inhaled for maintenance of anesthesia. Breathing was spontaneous for the first three hours of the procedure. A #9F introducer sheath was inserted into the left femoral artery. Through this, a double-lumen balloon catheter was advanced into the right iliac artery under fluoroscopic control. At this time, the arterial blood pressure was 100/60 mmHg, heart rate 70/min, and respiratory rate 18-20/min. pH was 7.35, PaO2 110, and PaCO2 46 mmHg. The tip of the catheter then was advanced into a feeding vessel and the balloon inflated. Angiography indicated satisfactory control of blood flow through the AVM. A total of 4.5 ml radiopaque silicone fluid was injected to occlude the nidus of the malformation. Immediately after the injection, respiratory rate increased to 40/min, arterial blood pressure decreased to 70/40 mmHg, and the heart rate to 50/min with normal sinus rhythm. Within three minutes, heart rate increased to 110/min and arterial blood pressure to 100/60 mmHg. Fio2 during this episode was increased to 1.0, halothane was turned off and ventilation controlled at 20/min (Ve = 700 ml). Fluoroscopy during injection detected some radiopaque silicone in the regional veins. Manual pressure was applied over the right groin to prevent further vascular entry of silicone fluid. Breath sounds were clear. The lung fields first were examined fluoroscopically, followed by a chest roentgenogram which showed bilateral miliary opacities. A right radial arterial line was inserted ten minutes after the hypotensive episode and blood-gas analysis showed pHc of 7.39, PaCO2, 43 mmHg, and PaO2 294 mmHg with an A-aDO2 of 369 mmHg. At this time, N2O 50% was introduced into the inspired mixture. A subsequent analysis of arterial blood gases revealed pHc, 7.38, Pao2 31 mmHg, and Paco2 194 mmHg. Minute ventilation was reduced to 8 l/min. To prevent further embolization, the right femoral vein was occluded. During plication, arterial blood pressure remained 100/80 mmHg and the heart rate 90/min. Forty-five minutes after the embolization, pHc was 7.35, PaCO2 41, and PaO2 181 mmHg with Fio2 of 0.5. Further embolization was postponed. Neuromuscular block was reversed by standard doses of neostigmine and atropine and trachea extubated. The immediate postoperative course was uneventful and blood pHc was 7.34 mmHg, PaCO2 43 mmHg, and PaO2 181 mmHg on 70% oxygen by mask. From the second to the eleventh postoperative days her body temperature varied from 37.5°C to 39°C. She also complained of left-sided pleuritic chest pain. A chest roentgenogram taken on the first postoperative day showed bilateral multiple radiopaque densities throughout both lung fields and blunting of both costophrenic angles (fig. 1). Serial sputum cultures revealed no organism. Chest pain and fever resolved on the twelfth postoperative day without antibiotic therapy. Pulmonary function studies done three days after the embolization showed mild decrease of all lung volumes, probably secondary to pleuritic pain. A chest roentgenogram taken 20 days later still showed silicone opacities but was otherwise normal (fig. 2).

**DISCUSSION**

The appearance of the chest roentgenograms leaves little doubt that acute silicone pulmonary embolism occurred during the procedure. This was associated with systemic hypotension, tachypnea, increased A-aDO2 (369 mmHg), and alveolar dead space (PaCO2 of 41 mmHg at a Ve of 14 l/min). The characteristic pleuritic chest pain together with poor inspiratory effort and blunting
of costophrenic angles further support the diagnosis. Previous reports reviewing experience with intraarterial AVM embolization failed to describe symptomatic pulmonary embolization.\textsuperscript{1,9,10} The reported pulmonary emboli were clinically asymptomatic or occurred several days after the procedure, suggesting that the delayed embolization was thromboembolic in nature but not caused by the foreign substance.\textsuperscript{5,11}

The extent of the embolic pulmonary artery occlusion in this case is difficult to determine. McIntyre \textit{et al.},\textsuperscript{12} correlated angiographically documented extent of the pulmonary arterial bed occlusion with arterial oxygenation and pulmonary artery pressure. They found that decreased $\text{PaO}_2$ was seen with as little as 13% pulmonary arterial bed obstruction. When occlusion exceeded 30%, mean pulmonary artery pressure consistently reached 30 to 40 mmHg. Since we neither measured pulmonary artery pressure nor performed an angiogram, accurate quantitation of the obstruction was impossible. However, the sudden onset of tachycardia, systemic hypotension, decreased $\text{PaO}_2$, together with the diffuse opacities seen in the chest roentgenogram (fig. 1), suggest that at least 15–20% of the pulmonary vasculature was embolized. The amount of injected silicone that escaped into the pulmonary circulation appears to be insufficient to cause respiratory or hemodynamic changes seen in this patient. Fluoroscopic observation during the injection suggested that 50–60% of the injected material remained in the malformation. Acute neurohumorally induced pulmonary vasoconstriction may have been responsible for the transient respiratory and hemodynamic changes. Short-lived symptomatic cardiorespiratory changes have been induced experimentally during pulmonary embolization with autologous blood clot and barium sulfate.\textsuperscript{13} A similar mechanism also may be present during pulmonary embolism in humans.\textsuperscript{14}

The passage of embolizing material into the venous system is clearly dependent on the physical and chemical nature of the injectate and the anatomic and hemodynamic characteristics of the lesion.\textsuperscript{6,8,10} Silicone liquid does not adhere to the vessel wall\textsuperscript{3,4} and produces no local inflammatory response.\textsuperscript{15} Blood flow through the AVM must be interrupted by inflating the balloon catheter for a sufficient period of time to allow hardening or vulcanization of the liquid to form a cast of the abnormal vascular bed.\textsuperscript{3,4,6} A significant amount of afferent blood flow will persist through the AVM if the balloon deflates prematurely in the presence of large collateral circulation. Thus, it is imperative to assure a temporary interruption of flow to an AVM if a nonadhesive material such as silicone fluid is used. In our case adequate blood flow control could not be accomplished probably due either to the abundant number of feeding arteries resulting in a collateral circulation or premature deflation of the balloon. Isobutyl-2-cyanoacrylate (IBCA) is considered to be one of the most desirable agents for embolizing high-flow fistulous communications because it has high tissue adhesiveness.\textsuperscript{3,4} We used this material to embolize the residual malformation three months later without causing pulmonary embolism.

The use of radiopaque embolizing substance has the advantage of enabling the physician to make diagnosis without invasive monitoring. Silicon, PVA, and IBCA can be made radiopaque by adding either barium sulfate or talentum powder.\textsuperscript{3,4,8} The use of continuous endtidal $\text{CO}_2$ monitoring also may be beneficial in these patients to monitor pulmonary embolism noninvasively.

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\textbf{REFERENCES}