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Grand Mal Seizure after Fentanyl Administration

To the Editor:—We read with great interest the study by Carlsson *et al.*¹ on the effects of high-dose fentanyl on cerebral circulation and metabolism in rats, where they describe seizure activity after high-dose fentanyl (200 or 400 $\mu\text{g}/\text{kg}$). The following is a report of a case where a patient developed grand mal seizures after the administration of 200 μg fentanyl.

A 79-year-old woman weighing 79 kg was admitted for total abdominal hysterectomy for cervical carcinoma. Her medical history was remarkable for a questionable syncopal episode, which was attributed to premature ventricular contraction (PVC) detected by Holter monitoring, for which she was placed on quinidine. The remaining cardiac and neurologic workup was normal.

She was premedicated with Robinul® 0.3 mg 45 min before arrival to the operating room. After placement of the appropriate monitors (ECG V5 lead, direct arterial pressure, and central venous pressure monitoring), anesthesia was induced with diazepam 5 mg iv and fentanyl 200 μg in divided doses. During this time ventilation was assisted with O₂ (100%) and the arterial pressure and heart rate remained unchanged. Within 2 min, generalized clonic motor activity was noted, which was successfully treated with 125 mg thiopental. The operation was canceled, and the patient was allowed to recover from the anesthetic. Her postoperative course was uneventful. A neurologic consult was sought immediately in the recovery room, and the neurologist's initial impression was that she had had a grand mal seizure related to fentanyl. Results of further neurologic examination were negative, and electroencephalographic testing was normal.

The patient underwent an uneventful general anes-

thetic 2 weeks later, and fentanyl was not given. Morphine sulfate was given for pain relief in the postoperative period without untoward effects.

Although it was mentioned in the discussion by Carlsson *et al.*¹ that seizures after narcotic administration have been seen in humans, such seizurelike activity has not been reported following fentanyl administration. The data by Carlsson *et al.* would confirm strongly that a narcotic-induced seizure occurred in our case. It is interesting that 5 mg of diazepam (Valium®) did not prevent the seizure and that the seizure occurred after such a small dose of fentanyl (200 μg). This is at odds with clinical experience and with Carlsson's study in which seizures occurred only after high-dose fentanyl. Nonetheless, we wish to report our experience of a grand mal seizure after a small dose of fentanyl so we may alert our colleagues to the possibility of seizures with fentanyl in humans.

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Collapse after Epidural Injection Following Inadvertent Dural Perforation

To the Editor:—I am intrigued by the possibility that the communications on this topic from Professor Hodgkinson¹ (United States) and Dr. Collier² (Australia) reveal yet another geographically determined variant of outcome of anesthetic practice.

We have inadvertently perforated the dura of 302 obstetric patients during the period from 1968 to the present in my service. Our initial response to this com-

plication is to insert the catheter through another space (almost invariably an adjoining one). We have not encountered evidence that a massive spinal block or a massive subdural block has resulted from injecting a considerable volume of bupivacaine through a catheter so cited. Occasionally clear fluid is seen to drift slowly within the catheter after insertion. We assume that this is some of the cerebrospinal fluid that already has leaked

into the epidural space, but then we administer top-up doses with extra caution, lest the catheter has been placed intrathecally, which had indeed occurred in two cases as demonstrated by an ability to aspirate a continuous flow of spinal fluid.

I wonder if, on the basis of their conjectures, your contributors have considered the distribution of blood injected for an epidural blood patch? To date, we have administered 246 of these in the treatment of headaches, consequent upon either an inadvertent perforation or a spinal block, when, by definition, there is a hole in the dura. In no such circumstance has there been evidence that the blood has reached either the subdural space or the cerebrospinal fluid.

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Pseudoarterialization of the CVP by an Infusion Pump

To The Editor:—We would like to call attention to a problem associated with the use of an Abbott/Shaw Life Care Infusion Pump (Model 3) during induction of anesthesia in a neurosurgical patient. A 20-gauge needle attached to the infusion set was inserted through a rubber port in a long-line central venous catheter that displayed a normal CVP tracing on our monitor. Shortly after initiating the infusion of fentanyl at a rate of 300 ml/h, we observed a sudden increase in the CVP from 10 to greater than 60 mmHg. In addition, the configuration of the tracing was arterial. We were concerned that the tip of the catheter had migrated into the right ventricle. Withdrawal of the catheter did not result in the return of the previously normal CVP tracing. It also was noted at this time that the frequency of these newly observed waves in the CVP tracing appeared to be regular but was not the same as that of the systemic arterial pressure waves being monitored simultaneously. Turning off the infusion pump resulted in the reappearance of a normal CVP tracing. When the infusion was restarted, the abnormal tracing reappeared. At this time, we decided to continue the iv infusion of fentanyl, using a peripheral rather than the central venous access to avoid further confusion.

A study of the Abbott/Shaw Life Care Pump was undertaken *in vivo* to determine the range of pressures that could be generated in the central venous catheter at different infusion rates. In a patient with a CVP of 8 mmHg, four sets of measurements, including systolic, diastolic, and mean pressures, were averaged and are presented in table 1 along with their corresponding stroke and infusion rates. Peak systolic and mean pressures are significantly higher than the baseline CVP at all infusion rates, and all pressures increase gradually

as infusion rates increase. To determine if the pressures generated by the pump in the tubing could be transmitted to the central circulation, a double lumen pulmonary artery catheter was modified by removing about 30 cm of length distal to the proximal (CVP) port, and was inserted into the central venous circulation. No alterations in the CVP tracing monitored distally were observed during infusion of fluid through the proximal port at a rate of 400 ml/h. The system operating manual of the Abbott/Shaw Life Care Pump states that the pressure at the tip of the needle remains essentially similar to that within the cannulated vein, regardless of the pressure developed within the set, pump chamber, and tubing. In view of our findings, we feel that this information is accurate. The high pressures measured in the CVP catheter result from the rapid acceleration of small quantities of fluid by the pump mechanism. It appears that these pressure waves are attenuated almost instantaneously after exiting the infusion set tubing and nee-

TABLE 1. Pressures Generated by the Abbott/Shaw Life Care Pump at Various Infusion Rates in One Patient with a CVP of 8 mmHg

Rates		Pressures*		
Infusion (ml/h)	Strokes (no./min)	Systolic (mmHg)	Diastolic (mmHg)	Mean (mmHg)
10	3	59	14	29
20	5	63	14	30
40	10	63	14	30
100	24	66	16	33
200	48	74	22	39
300	72	82	31	48
400	96	92	40	57

* Average reading after four trials.