the usual iv preoperative dose of metronidazole necessary to obtain therapeutic blood levels. In humans, the therapeutic metronidazole blood level of 4 ng·ml⁻¹ already is attained and maintained for 6–8 h after slow—20 min—iv administration of 7.5 mg·kg⁻¹ of metronidazole.¹³,¹⁴ Therefore, it is unlikely that following the administration of one usual iv clinical dose of metronidazole a potentiation of vecuronium induced neuromuscular block will occur in normal subjects.

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Persistent Phrenic Nerve Paresis Following Interscalene Brachial Plexus Block

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Accidental, temporary blockade of the phrenic nerve occurs in 36% of patients having interscalene brachial plexus block,¹ but long-lasting injury to the phrenic nerve has not been reported. We describe a phrenic nerve paresis that has persisted more than 3 years following an interscalene brachial plexus block.

Report of a Case

A 64-year-old man, 180 cm tall, weighing 95.5 kg, and in good health except for well-controlled hypertension, was hospitalized for elective shoulder surgery. Admission physical examination was unremarkable except for decreased strength and limited range of motion of the right shoulder. His preoperative chest roentgenogram was normal except for basilar fibrosis.

Premarkedication was with fentanyl 25 µg, droperidol 1.25 mg, pentobarbital 50 mg, and scopolamine 0.4 mg, all intramuscularly. After establishing an iv infusion, electrocardiograph, and (cuff) blood pressure monitoring, the nerve block was attempted using the technique described by Winnie.² A 22-gauge, short-bevel needle was oriented with the bevel parallel to the nerve fibers, and after some difficulty, a paresthesia to the shoulder was elicited. Within 30–45 s following injection of bupivacaine, 50 ml 0.5% solution with epi- nephrine, 1:200,000, the patient had a generalized seizure disorder. He was promptly treated with an oropharyngeal airway, positive-pressure oxygen via mask, and sodium thiopental 125 mg iv. Further details about the performance of the block or the resulting spread of anesthetic are not available from the written medical record.

Surgery was canceled, and the patient was taken to the recovery

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room. A portable chest roentgenogram showed a “significant elevation of the right hemidiaphragm when compared with the previous examination.” An electrocardiogram showed a new intraventricular conduction defect and T-wave changes that resolved during his recovery room stay. While breathing supplemental oxygen, $\text{PH}_2$ was 7.15, $\text{Pa}_\text{O}_2$ 270 mmHg, $\text{Pa}_\text{CO}_2$ 43 mmHg, and base excess was $-14.1$ mEq/l. The acidosis resolved following administration of sodium bicarbonate iv.

The patient remained in the hospital for 3 days, while serial electrocardiograms and cardiac enzyme analyses were obtained to exclude the possibility that he suffered a myocardial infarction during the toxic reaction to the local anesthetic. One week later, an exercise tolerance test was stopped because of dyspnea and fatigue at 45 s into stage 3 (of the standard Bruce protocol), resulting in a functional aerobic impairment of 12% on the sedentary scale. There was no suggestion of myocardial ischemia. He was then readmitted to the hospital and underwent a rotator cuff repair under general anesthesia without incident.

Although the patient had a good functional result from his shoulder surgery, he continued to be troubled by exertional dyspnea. Two months after the block, another radiographic examination showed that the elevation of the right diaphragm was unchanged from the recovery room film. Paradoxic movement of the right diaphragm was demonstrated fluoroscopically with a snifing maneuver. Pulmonary function tests performed at that time showed a restrictive abnormality (see table 1). The patient had a maximal inspiratory effort of 20 cm H$_2$O (20% of the predicted value) and a vital capacity that was lower in the right decubitus position (2.1 l) than in the left decubitus position (2.5 l). These findings are consistent with complete paralysis of the right diaphragm. One year after the injury, a cardiopulmonary progressive exercise test demonstrated a 30% improvement in maximal exercise capacity. His ventilatory impairment no longer limited his exercise performance in that his maximal exercise ventilation was only 60% of his maximal breathing capacity measured at rest.

The report of another chest fluoroscopy performed 30 months postinjury stated that with the patient standing there was wide, normal excursion of the left diaphragm and little, if any, motion on the right. With snifing, some paradoxic movement on the right occurred. Frontal and lateral films showed the right diaphragm to be slightly lower in position than on the previous examination, with a slightly different contour, suggesting some minor recovery occurred.

While pulmonary function tests over the next 3 years showed an improvement in inspiratory effort (to 40 cm H$_2$O) and maximal voluntary ventilation, the vital capacity and total lung capacity did not change in any consistent fashion, supporting the fluoroscopic interpretation of continued diaphragmatic paralysis.

**DISCUSSION**

After a normal preoperative chest roentgenogram, the postblock appearance of a persistently elevated right
diaphragm strongly suggests that the phrenic nerve injury occurred during the procedure, possibly due to needle trauma. Although the procedure also was complicated by a systemic toxic reaction to bupivacaine, it is unlikely that this contributed to the nerve injury, because the needle had been removed prior to the onset of the seizure. Since the 0.5% bupivacaine solution is approved for infiltration on peripheral nerves, local anesthetic neurotoxicity is an unlikely possibility. The relatively large volume employed may have facilitated the spread of local anesthetic to the phrenic nerve, but this should have resulted in only temporary blockade.

At the level of the transverse process of the sixth cervical vertebrae (where the block had been performed), the right phrenic nerve lies on the anterior surface of the anterior scalene muscle, which separates it from the brachial plexus. Thus, anterior misplacement of the needle would have been necessary to injure the nerve. Knoblauch pointed out that the thin investing fascia of the anterior scalene muscle forms the only barricade to spread of local anesthetic solution from the brachial plexus to the phrenic nerve. This explains the frequent occurrence of phrenic nerve block concomitant to the interscalene block as well as to the classic supraclavicular and perivascular approaches to brachial plexus block. Temporary phrenic block also has followed cannulation of the internal jugular and subclavian veins, and injury to the phrenic nerve has occurred with both procedures with reported follow-up periods ranging from 6 months to 3 years.

On the other hand, deliberate blockade of the phrenic nerve with local anesthetic has been employed as treatment for intractable singultus without reported residual paresis. Furthermore, before antituberculous drugs were available, phrenic neuroparalyticus often was produced with a surgical clamp to collapse large tuberculosis cavities in the lung, and the median duration of the resulting diaphragmatic paralysis ranged from 6 to 12 months in various centers. The rare paralysis persisting after 2 years was considered to be permanent.

In summary, phrenic nerve paresis occurred following an interscalene brachial plexus block that also was complicated by a systemic toxic reaction to the local anes-
thetis. The expected time for nerve regeneration has passed, suggesting that the injury is permanent.

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Respiratory Failure Secondary to Homologous Blood Transfusion

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The appearance of alveolar flooding in the perioperative period is often unheralded and usually unwelcome. Accurate diagnosis and prompt treatment are imperative. An example of a severe form of acute pulmonary edema with an uncommon etiology will be described, followed by a differential diagnosis and a review of the available information on this phenomenon.

REPORT OF A CASE

A 32-year-old man with progressive, juvenile rheumatoid arthritis was scheduled to undergo total hip arthroplasty. Preoperative evaluation also revealed a history of paroxysmal atrial tachycardia controlled with digoxin, inflammatory iritis—improved through the use of flumethalone ophthalmic drops—and iron deficiency anemia. Aspirin and steroid therapy had resulted in upper gastrointestinal bleeding several years earlier, necessitating a partial gastrectomy and vagotomy. Previous anesthesics were historically uncomplicated; the family anesthetic history was negative; and he neither smoked cigarettes nor used alcohol. Chronic medications included, in addition to those mentioned above, naproxen, aspirin, and prednisone, and he denied medication allergy. He was small in stature, at 167 cm tall, weighing 52 kg. In addition to the joint deformities associated with rheumatoid arthritis, the patient exhibited considerable restriction of temporomandibular and cervical motion, and anisocoria, with the diameter of the right pupil being approximately 3 mm larger than the left pupil. The preoperative chest roentgenogram showed evidence of early interstitial fibrous changes consistent with rheumatoid arthritis, and the EKG indicated digoxin effect. His hemoglobin concentration was 11.5 g·dl⁻¹ and the hematocrit was 35%; however, the remainder of laboratory data, including quantitative serum immunoglobulins and immunoelectrophoresis, were normal.

Anesthesia was induced with thiopental 300 mg iv, followed by succinylocholine chloride 70 mg iv to facilitate laryngoscopy. Using a conventional technique, only the tip of the epiglottis could be visualized; however, an 8-mm ID cuffed endotracheal tube was inserted successfully. Anesthesia was maintained with an initial fentanyl dose of 10 μg·kg⁻¹·iv, along with isoflurane and oxygen. Following induction, radial arterial and central venous cannulae were inserted for monitoring. With an FiO₂ of 1.0 pH was 7.47, PaCO₂ 33 mmHg, and PaO₂ 379 mmHg. His gas exchange and acid-base status did not change significantly during this 2h operation. A central venous pressure of approximately 10 mmHg was maintained through an infusion of 1,800 ml lactated Ringer’s solution, two units of whole blood were transfused to replace blood loss, and nitroglycerin was administered to reduce the arterial blood pressure by 30% from his baseline. Paralysis was induced by an atracurium infusion titrated to 0.9% train-of-four and electromyogram (EMG) depression; the effects later were antagonized with edrophonium and glycopyrrrolate, and the trachea was extubated.

One hour after arrival in the recovery room, with an FiO₂ of 0.4, the pH was 7.42, PaCO₂ 39 mmHg, and PaO₂ 86 mmHg. His central venous pressure and arterial blood pressure were 8 and 136/74 mmHg, respectively. With a hematocrit of 26% and a heart rate of

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