tor unit. This is associated with clinical improvement in motor strength weeks and months after an acute attack of polio. After decades, a postpoliomyelitis syndrome can develop, with muscle atrophy potentially progressing to complete paralysis. This syndrome is thought to occur secondary to increased functional demands, or overuse, of the giant motor unit, which results in the death of its sprouts.[2,3]

There have been a few reports over the years using succinylcholine in patients with pathology similar to that seen in PPS. For example, succinylcholine-induced hyperkalemia and circulatory collapse were reported in a patient with acute idiopathic anterior horn cell disease[4]; the serum potassium during this cardiac arrest was 7.9 mEq/l. Another study of denervated baboons found an increase in intravascular potassium up to 5.5 mEq/l.[5] PPS is similar in pathophysiology to the baboon denervation study, and one could assume that hyperkalemia could also be seen in PPS patients. There have been numerous reports of hyperkalemia in patients with neuromuscular disease.[4–8] It would have been informative to have had the prepotassium and postpotassium measurements from the patient in the report of Wernet *et al.*[1] to determine the magnitude and time frame of the increase of serum potassium.

The avoidance of neuraxial anesthesia was also discussed by Wernet *et al.* Successful neuraxial anesthesia in patients with PPS has been reported without adverse complications.[9,10] Many clinicians provide regional anesthesia for labor and delivery in patients with a history of PPS.[11]

If general anesthesia needs to be induced, the potential hazard of using succinylcholine in patients with PPS has been acknowledged.[12] If the need for rapid sequence induction exists in a PPS patient, we believe one should choose a short-acting nondepolarizing muscle relaxant in lieu of succinylcholine; the only caveat would be to consider using a decreased dose because of the increase risk of muscular weakness.[13]

The mere fact that succinylcholine was used in the current case does not preclude the possible occurrence of severe, acute hyperkalemia in subsequent cases in patients with PPS. We do not believe that one can conclude from this single case that succinylcholine should be used in patients with PPS.

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In Reply:—We thank Drs. Connelly and Abbot for their interesting comments on our case report.[1] They give us the opportunity to clarify the choice our anesthetic strategy in the current case.

Drs. Connelly and Abbot mention a number of cases reported in the 1970s in which life-threatening hyperkalemia induced by succinylcholine was observed in patients with denervation pathology. As emphasized in our letter, the use of succinylcholine in patients with postpoliomyelitis syndrome remains controversial because of the risk of fatal hyperkalemia observed in patients with pathology close to postpoliomyelitis syndrome. The molecular mechanism of succinylcholine-induced hyperkalemia has been nicely described by Martyn and Richtsfeld[2] and results primarily from motor endplate receptor up-regulation. This explains why the expected dangerous increase in serum potassium occurs over a very brief period of time, and why normal preoperative potassium levels have limited value in predicting the magnitude of potassium increase. In our case, preoperative blood potassium was 3.4 mM. Although we did not obtain a postoperative potassium determination, we did not observe any significant electrocardiographic modification, such as T-wave changes indicative of hyperkalemia, in the current case after succinylcholine injection.

Drs. Connelly and Abbott also discuss the choice of general *versus* regional anesthesia in our case. We agree that regional anesthesia has been successfully used in some cases of patients with postpoliomyelitis syndrome. However, we did not consider it as a first-choice strategy because the risk of exacerbating the motor deficit of the limbs due to a toxic action of local anesthetics on the motoneurons could not be excluded. Moreover, there was a lack of symptoms suggesting a diagnosis of postpolio-related central disorder. We disagree with Drs. Connelly and Abbott that the use of a nondepolarizing muscle relaxant for rapid sequence intubation always represents an effective and safe alternative to succinylcholine. None of the nondepolarizing muscle relaxants available to date have the same rapidity of onset and reversal of action as that exhibited by succinylcholine. This particular kinetic profile makes this agent preferable to any other nondepolarizing muscle relaxants to decrease the risk of inhalation of the gastric contents, as was particularly important here for cesarean delivery. We agree that careful titration (dose reduction) and monitoring of succinylcholine effects are necessary, which was performed in our case.

Finally, we did not state, as mentioned by Drs. Connelly and Abbot, that succinylcholine *should be used* in patients with postpoliomyelitis syndrome. From this case, we concluded that the anesthetic strategy must be chosen after an extensive risk–benefit discussion, and that succinylcholine may represent a possible alternative in such patients, pending careful titration and monitoring of its effects.

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Beach Chair Position: Is It Really Feasible in Laparoscopic Procedures?

To the Editor:—In the November 2007 issue of ANESTHESIOLOGY, Valenza *et al.*[1] described how they used the application of positive end-expiratory pressure and the beach chair position to counteract the respiratory derangements in morbidly obese patients undergoing laparoscopic gastric surgery. The authors state that although both the beach chair position and the addition of positive end-expiratory pressure each similarly improved lung volumes, oxygenation, and respiratory mechanics at baseline, it was only the combination of both positive end-expiratory pressure and the beach chair position that improved oxygenation during pneumoperitoneum. Because it is often the practice of surgeons to ask their anesthesiology colleagues to place the patients in some degree of Trendelenburg to "facilitate" the exposure during laparoscopic surgeries, we are forced to question how feasible it would be to use the beach chair position in any laparoscopic procedure, let alone in morbidly obese patients. Moreover, in certain types of laparoscopic surgeries (prostatectomies, and some gynecologic surgeries), extreme Trendelenburg is requested, thus making the use of beach chair position even more impractical. A number of variables contribute to the respiratory derangements in all anesthetized patients; notable among them are the general anesthetic, paralytics, pneumoperitoneum, and positioning. The authors describe performing recruitment maneuvers (three consecutive inspiratory holds of 5 s at 45 cm H_2O airway pressure) in both positions, with and without pneumoperitoneum, to improve respiratory derangements. Although it is not always possible to consistently maintain improved oxygenation for a prolonged period of time after performing the recruitment maneuvers, it is quite more practical to perform these maneuvers as opposed to attempting to use the beach chair position when performing laparoscopic procedures.

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In Reply:—Drs. Singh Heir and Gottumukkala are right: If a surgeon asks for an "extreme Trendelenburg" position, one cannot use the head-up position as a strategy to counteract hypoxemia, unless in an emergency!

The negative effects of the Trendelenburg position have been investigated by others.[1–3] Any conclusion taken from our data on the Trendelenburg position is merely speculative, provided we set out to investigate the beach chair position and positive end-expiratory pressure (PEEP).[4] Nevertheless, our data allow some speculations on the aspect brought up by Drs. Singh Heir and Gottumukkala in their letter.

In a sedated and paralyzed patient, pleural pressures displace the relaxed diaphragm downward, hence increasing end-expiratory lung volume, while intraabdominal pressure (IAP) acts as a counter pressure on the diaphragm. When the bowels are allowed to slide under gravity in the head-up position, bladder pressure increases, as we have shown (from 17.87 ± 5.45 to 23.92 ± 4.35 cm H_2O; $P < 0.01$). The increase of bladder pressure in head-up position may thus correspond to a reduction of pressure pushing the diaphragm upward. When we estimated pleural pressure as Ppl = (PEEP + PEEPi) × Ew/(El + Ew)[5] in the supine and beach chair positions with and without PEEP, and considered changes of IAP due to the beach chair position (ΔIAP) to obtain an indicative "push-down pressure" (Ppl + ΔIAP), we found that this was positively correlated with end-expiratory lung volume ($R^2 = 0.857$, $P < 0.001$, m = 0.67, multiple linear regression analysis). We did not measure IAP changes occurring in the extreme Trendelenburg position. However, we would anticipate that an increase in the forces shifting the diaphragm upward would ensue following the same physical rule. In this condition, PEEP (hence Ppl) may be used as a counterpressure that opposes diaphragm upward shift, thus preserving lung volume.

Recruitment maneuvers proposed by Drs. Singh Heir and Gottumukkala to improve oxygenation have been discussed in our article.[4] They are possibly even more important in the extreme Trendelenburg position than in the supine position to open up the lung. However, their effects are short-lived,[6] whereas PEEP is known to maintain lung units open, once recruited.

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