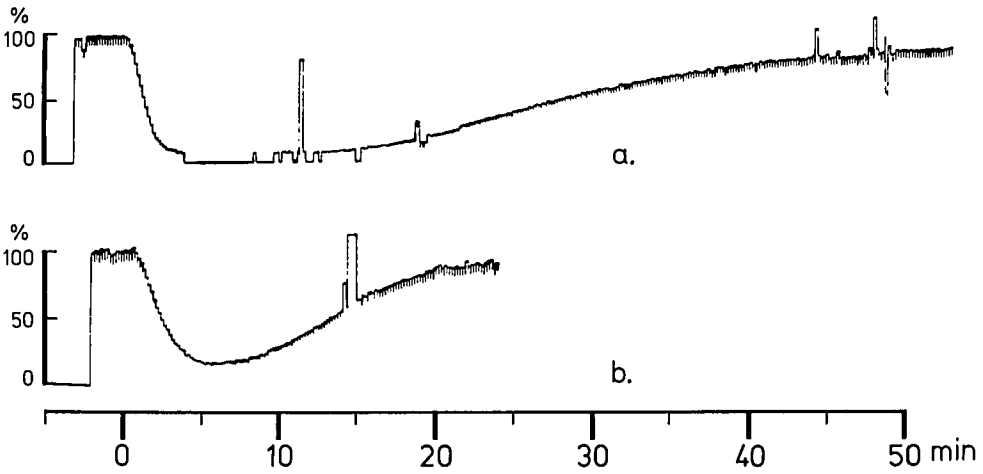

FIG. 1. Electromyographic recording of the effect of vecuronium with (*a*) and without (*b*) oral dantrolene pretreatment. At t = 0, an iv bolus of 45 μg/kg vecuronium was given.

In summary, our case report suggests that pretreatment with oral dantrolene may prolong the duration of muscle paralysis caused by vecuronium.

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Diagnostic Application of an Axillary Block in an Infant

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Although anesthesia of the upper extremity by means of a brachial plexus regional block is well described in pediatrics,[1,2] its use in infants and children for surgical procedures frequently is restricted. We report the use of the axillary approach to blocking the brachial plexus to provide excellent conditions for electromyographic (EMG) study of the upper extremity in an infant who was suspected of having an infantile form of myasthenia gravis.

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REPORT OF A CASE

The patient, an 18-month-old male infant (weight 12.2 kg), was hospitalized since birth for multiple problems following an apparent episode of perinatal asphyxia and meconium aspiration. He was a 3,015-g product of a 37-week gestation in a 20-year-old woman with no history or clinical signs of myasthenia gravis. Labor was complicated by variable and late decelerations. Immediately after delivery, his trachea was intubated, and meconium suctioned from below the vocal cords, with immediate extubation. Apgar scores were 3 at 1

min and 5 at 5 min. Subsequent metabolic and respiratory acidosis required reintubation of the trachea in the neonatal intensive care unit. While there was no radiographic evidence of cardiopulmonary disease, repeated trials of extubation and spontaneous ventilation resulted in respiratory distress requiring reintubation of the trachea. At 1 month of age, bronchoscopy revealed supraglottic and subglottic edema, and a tracheostomy was performed. Although the infant was maintained on continuous positive airway pressure (CPAP), respiratory compromise secondary to atelectasis and pneumonia required increasing intervals of mechanical ventilation. Cardiac catheterization revealed elevated pulmonary artery pressures accompanying the episodes of hypoventilation and hypercarbia but normal pulmonary artery pressure with adequate ventilation. Eventually, he became totally dependent upon mechanical ventilation.

The patient was unable to tolerate feedings due to significant regurgitation. He underwent a Nissen fundoplication, and a gastrostomy was performed. Bolus feedings were not tolerated, and constant infusion feedings were required due to a "dumping syndrome." An esophogeal sump was required due to poor esophageal motility.

At nine months of age the patient (weight: 6.4 kg) had a prolonged complex motor seizure, which responded to treatment with phenobarbital. The patient was noted to be generally hypotonic at all times, with a developmental delay in motor function that was greater than that in cognitive and social areas.

Due to his inability to ventilate adequately, an EMG was scheduled to rule out myasthenia gravis. Despite inadequate ventilation, the patient had spontaneous, generalized but weak motor activity of his extremities. Anesthesia would be required in order to provide an electrophysiologically silent background for an adequate EMG study. Blockade of the brachial plexus with light sedation was considered. Methohexital (360 mg) was administered rectally with inadequate effect after 12 min, so ketamine (36 mg) was administered im, causing sedation with minimal motor activity. A 27-gauge metal hub needle insulated down to the needle bevel with a 24-gauge plastic catheter was used with electrical stimulation by a Digistim II® nerve stimulator to verify proximity to nerves in the axillary sheath. With his left arm abducted 90 degrees, the patient's axillary artery was palpated and the needle inserted at a 45-degree angle to the skin along an axis parallel to the artery. Stimulus–synchronous flexion of the hand was noted with 0.5 mA of current being passed through the needle but not with 0.1 mA. The needle was advanced, restoring hand flexion with the lower stimulation intensity, and 8.8 ml of 1% lidocaine (7 $mg \cdot kg^{-1}$) with 1:200,000 epinephrine was injected. Over the next 20 min, the patient gradually showed signs of increased motor activity and awareness; however, he was unable to move his left upper extremity. The patient did not react when current was passed through the stimulating electrodes employed for neurologic study. The nerve conduction study showed no evidence of deficits. The EMG recording from the opponens policis with medial nerve stimulation revealed the typical decrement in compound action potential amplitude consistent with a myasthenic syndrome. Within 2 h the patient recovered motor activity in the left arm. In spite of a trial of neostigmine, the patient continued to require ventilatory support and hospitalization.

Discussion

In order for an EMG to provide accurate information concerning the function of the neuromuscular junction, spontaneous background motor activity and motor response to the occasionally painful stimuli must be absent. Adults usually are able to cooperate sufficiently, but children and especially infants will not. Although general anesthesia would provide adequate conditions, anesthetic levels of all iv and volatile general anesthetics interfere with the neuromuscular junction by altering the kinetics of the acetylcholine activated ion channel.[3–5] Although sedating doses could be employed, anesthetic doses of these drugs could complicate conclusions of EMG studies by altering neuromuscular transmission. The axillary block provides an effective means of anesthetizing the upper extremity so that the EMG study can be performed. Local anesthetics have been shown to enhance the effect of neuromuscular blocking agents; however, they have not had any effect on twitch tension in and of themselves.[6,7] The serum levels expected in this infant (2–3 $\mu g \cdot ml^{-1}$) for an axillary block with the dose employed (7 $mg \cdot kg^{-1}$) are 10–20% of the levels employed to demonstrate synergism with neuromuscular blockers. These levels are far less likely to interfere with neuromuscular function than general anesthetics at clinical levels.

The myasthenic syndromes in infancy show variable clinical expression. Transitory neonatal myasthenia represents that syndrome of myasthenia in infants who were born to a myasthenic mother. Treatment with anticholinesterase drugs usually provides sufficient treatment until the circulating globulin that binds the acetylcholine receptor protein disappears from the infant's circulation. Juvenile myasthenia represents that form of the syndrome in which children develop acetylcholine antibody to acetylcholine receptor protein, and onset in the majority of cases is after the age of 10 years, with no evidence of disease seen during the first year of life.[9] Congenital myasthenia and familial infantile myasthenia represent two syndromes that may be apparent at birth and constitute intrinsic diseases of the myoneural junction. One congenital form is associated with end-plate acetylcholinesterase deficiency and is refractory to anticholinesterase drugs.[9,10] Other congenital myasthenic syndromes have been demonstrated to be due to a defect in the acetylcholine receptor channel or a defect of acetylcholine resynthesis or mobilization and may show partial improvement with anticholinesterase therapy.[10] Exact identification of the specific nature of the end-plate defect requires microelectrophysiologic and cytochemical investigation of neuromuscular junctions of affected muscle. Such studies were not performed in this infant.

In summary, brachial plexus block provides a suitable and quiet electrophysiologic field in which to perform electromyographic studies in infants or patients otherwise unable to cooperate. Regional anesthesia reduces interference due to the effects of general anesthetic drugs on the neuromuscular junction.

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A Comparison of Two Automated Indirect Arterial Blood Pressure Meters: With Recordings from a Radial Arterial Catheter in Anesthetized Surgical Patients

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Most clinical measurements of arterial blood pressure are made by sphygmomanometry. Recently, automated sphygmomanometric blood pressure meters have been developed as substitutes for the manual sphygmomanometer. We evaluated two such devices: the Dinamap 845®** and the Infrasonde 4000®.†† Both devices automatically inflate a cuff to above systolic arterial pressure and determine arterial pressure by incremental deflations of the cuff. They differ in the physical measure used: the Dinamap 845® analyses pressure fluctuations sensed by the occluding cuff,‡‡ while the Infrasonde 4000® uses a microphone to detect infrasound (20–30 Hz) waves associated with motion of the arterial wall.§§ Both devices display heart rate, and systolic, diastolic, and mean blood pressures.

MATERIALS AND METHODS

Forty male patients, who required arterial cannulation for medical reasons, participated in this study. All patients meeting this criterion, and given general anesthesia in the supine position, were included in succession until the study was complete. The experimental protocol was reviewed and approved by the Veterans Administration Research and Development (Human Studies) Committee. We compared the Dinamap 845® (N = 20) and the Infrasonde 4000® (N = 20) with a common direct measure, obtained from a radial artery catheter.

For the direct measurements, a radial artery was cannulated with a Criticon Catholon-IV® 20 G 1-1/4 catheter. A Gould/Statham® P23 pressure transducer was connected to the patient via a Sorensen Research® monitoring kit, which contained pressure tubing, an Intra-flo® automatic flushing device, and a stopcock. The total length of tubing in the system was 161 cm.

Direct arterial pressure measurements may be seriously

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** Dinamap® Adult/Pediatric Vital Signs Monitor, model 845. Criticon Inc., Northwest Shore Blvd, Tampa, Florida 33607.

†† Infrasonde D4000® Automatic Digital Blood Pressure Monitor, Puritan–Bennett, 12655 Beatrice St., Los Angeles, California 90066.

‡‡ Operations Manual, Dinamap® Adult/Pediatric Vital Signs Monitor, model 845. Criticon, Inc., 1981.

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