sponses to twitch and tetanic stimulation, sustained head lift for more than 15 sec, and proper responses to verbal commands ruled out residual neuromuscular block or residual anesthetic drug action as the causative factor in the airway obstruction.

The acute onset of airway obstruction immediately following extubation of the trachea in this patient can be explained on the basis of trauma to the adjacent cranial nerves due to operative manipulation. Consideration of the anatomic location of the cranial nerves helps to identify the cause of airway obstruction in this patient. The glossopharyngeal nerve arises from the posterolateral sulcus of the medulla craniad to the fibers of origin of the vagus and accessory nerves. The afferent pathway of the pharyngeal or gag reflexes is through the sensory fibers of the glossopharyngeal nerve, and the efferent pathway is through the motor fibers of the vagus nerve.[1] The hypoglossal nerve supplies the motor fibers to the tongue. Trauma to the glossopharyngeal nerve results in absence of the gag reflex and impairment of parotid gland secretions. Inability to move or protrude the tongue results from injury to the hypoglossal nerve. Thus, when glossopharyngeal nerve is injured bilaterally, as occurred in this patient, pharyngeal or gag reflexes will disappear, resulting in inability to protect the airway, and injury to

hypoglossal nerve results in the tongue's falling back, leading to airway obstruction.

In retrospect, the lack of gag or pharyngeal reflexes with suctioning and stimulating the pharynx prior to extubation of the trachea should have aroused suspicion regarding the status of patient's cranial nerves. This case points out that airway obstruction could occur in spite of the patient's meeting all the criteria for tracheal extubation following surgical manipulation of the posterior fossa, in the absence of anatomic abnormalities of the tongue, pharynx, and larynx.

Since the tumor distorted the anatomy of the nerves, and as the tumor was removed piece by piece, the surgeon was not aware of the injury to the cranial nerves. The neuro-anesthesiologist should be aware of the surgical anatomy and the structures in the operative field, and should make a practice of inspecting the operative site before closure commences. This would help to predict the possibility of cranial nerve dysfunction in the immediate postoperative course.

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Blood Levels of Bupivacaine after Injection into the Scalp with and without Epinephrine

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Infiltration into the scalp of a local anesthetic is often done for the surgical excision of a seizure focus. This will result in the appearance of the drug in the systemic circulation.[1] Since local anesthetics have anticonvulsant activity, systemic absorption might result in blood levels sufficient to decrease

seizure activity and interfere with the localization of the seizure focus.[2] On the other hand, higher blood concentrations may be associated with symptoms of local anesthetic toxicity. In this report, we present observations on blood levels of bupivacaine following subcutaneous infiltration into the scalp of 0.125 per cent and 0.25 per cent solutions with and without epinephrine, 1:400,000.

METHODS

Twenty-one patients undergoing awake craniotomy for excision of a seizure focus were studied. On arrival in the operating room patients were sedated with 1–2 ml of either fentanyl–droperidol or fentanyl alone administered intravenously. Routine monitoring for these procedures included electrocardiography and continuous arterial pressure monitoring via the radial artery.

TABLE 1. Injection Volumes and Blood Levels of Bupivacaine, and Patient Characteristics (Mean ± SD)

|	Number of Patients	Age (Years)	Weight (kg)	Initial Injection Volume (ml)	Total Injection Volume (ml)	Reinjections (Initial 120 Min)	Total Dose (mg/kg)	Peak Concentration in Blood (µg/ml) (Range)	Duration of Surgical Procedure (Min)
Bupivacaine, 0.125 per cent									
With epinephrine	6	26 ± 12	76 ± 18	41 ± 14	62 ± 18	1.7 ± 0.5	1.03 ± 0.80	0.12 ± 0.06 (0.06 ± 0.12)	604 ± 153
Without epinephrine	5	33 ± 9	64 ± 7	46 ± 8	63 ± 6	2.8 ± 0.8*	1.25 ± 0.20	0.77 ± 0.15 (0.68 ± 0.95)	438 ± 130
Bupivacaine, 0.25 per cent									
With epinephrine	5	23 ± 5	65 ± 9	40 ± 9	52 ± 9	1.2 ± 0.8†	2.02 ± 0.41†	1.01 ± 0.67* (0.20 ± 1.71)	497 ± 76
Without epinephrine	5	28 ± 2	78 ± 10†	51 ± 5‡	61 ± 12	0.6 ± 0.6*†	2.00 ± 0.48*†	1.23 ± 0.49*† (0.83 ± 2.08)	392 ± 118

* $P < 0.05$ compared with 0.125 per cent with epinephrine.
† $P < 0.05$ compared with 0.125 per cent without epinephrine.
‡ $P < 0.05$ compared with 0.25 per cent with epinephrine.

The following solutions of bupivacaine were used to anesthetize the scalp: bupivacaine, 0.125 per cent, with epinephrine, 1:400,000 (six patients); bupivacaine, 0.125 per cent, without epinephrine (five patients); bupivacaine, 0.25 per cent, with epinephrine, 1:400,000 (five patients); bupivacaine, 0.25 per cent, without epinephrine (five patients). The initial injection volume was determined solely by the magnitude of the craniotomy incision, and in no case exceeded the suggested dose for bupivacaine, *i.e.*, 175 mg without epinephrine and 225 mg with epinephrine (1:200,000).[3] Scalp infiltration in each patient also included peripheral nerve injection to block the auricular, occipital, and supraorbital nerves supplying the surgical site. We used the arterial catheter to sample blood for bupivacaine analysis. Blood samples for bupivacaine assay were drawn at 5, 10, 20 and 30 min and then at 30-min intervals for as long as four hours following the initial injection. At the time the study was initiated, Human Studies Review Committee approval for the volume of blood sampled was not required. Bupivacaine was chosen to provide scalp anesthesia because of this agent's long duration of action in relation to the particularly prolonged nature of these procedures. The various bupivacaine solutions tested were all in common clinical use. Bupivacaine assays were done using gas–liquid chromatography.[4] Statistical comparisons between groups were done using Student's t test for unpaired data.

Results

Satisfactory anesthesia was achieved in all patients. Initial injection volumes were similar for the groups receiving 0.125 per cent solutions. The initial injection volume in the group given 0.25 per cent bupivacaine without epinephrine, however, was greater than that in the group given 0.25 per cent with epinephrine ($P < 0.05$) (table 1). All patients tolerated the initial skin incision without discomfort. Subsequent reinjections were sometimes necessary, and were done when a patient expressed discomfort during the scalp incision and craniotomy. Most reinjections were done between the initial 10 min and 120 min period. Reinjections were needed less frequently with 0.25 per cent solutions, but in the group given 0.25 per cent without epinephrine, this may have been partly due to the use of a somewhat greater initial injection volume. The durations of the surgical procedures varied greatly. Mean durations were similar in the group receiving 0.125 per cent bupivacaine without epinephrine and both of the groups receiving the 0.25 per cent solutions. Every patient receiving 0.125 per cent with or without epinephrine needed one or more additional injections during scalp closure; this was not necessary in any patient receiving a 0.25 per cent solution, in procedures lasting as long as ten hours. Peak blood levels of bupivacaine for the groups as a whole were detected within 5–10 min after the initial injections in

FIG. 1. Arterial blood concentrations following injection into the scalp of 0.125 per cent bupivacaine (mean ± SE). *Solid line:* without epinephrine; *dashed line,* with epinephrine.
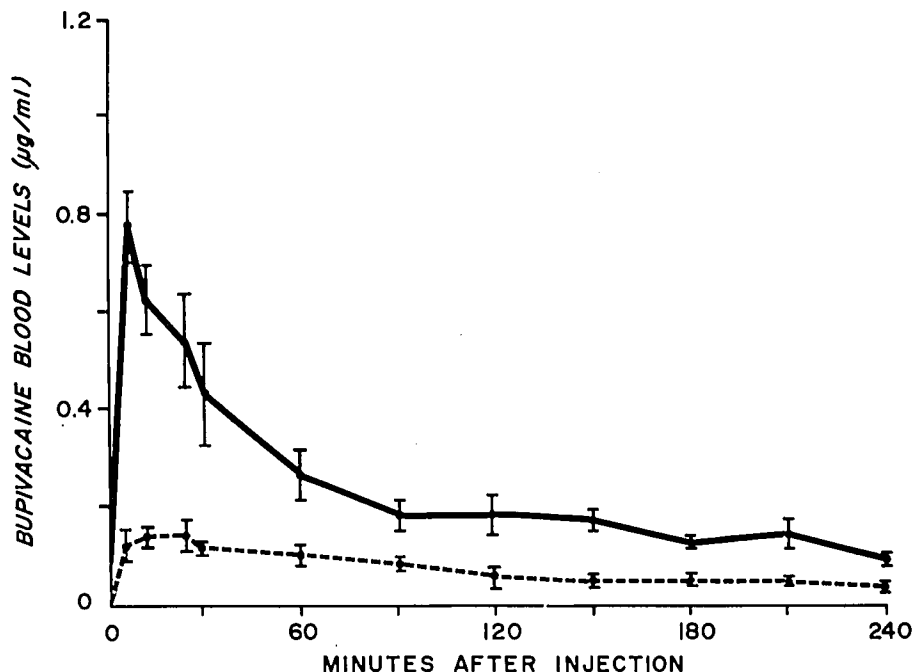
all groups (figs. 1 and 2). In individual patients, however, peak blood levels sometimes occurred later than 10 min and were related to reinjection. Peak levels progressively declined during the initial 120–150 min and then declined at a much slower rate. At the time of the EEG recording to detect the patient's seizure foci, the blood level was less than 0.5 μg/ml in every patient. The absence of anticonvulsant activity could not be determined, but in every patient a seizure focus was successfully located. No sign of local anesthetic toxicity occurred.

Epinephrine, 1:400,000, attenuated the systemic absorption when added to 0.125 percent bupivacaine, but not when added to 0.25 per cent bupivacaine.

FIG. 2. Arterial blood concentrations following injection into the scalp of 0.25 per cent bupivacaine (mean ± SE). *Solid line:* without epinephrine; *dashed line,* with epinephrine.
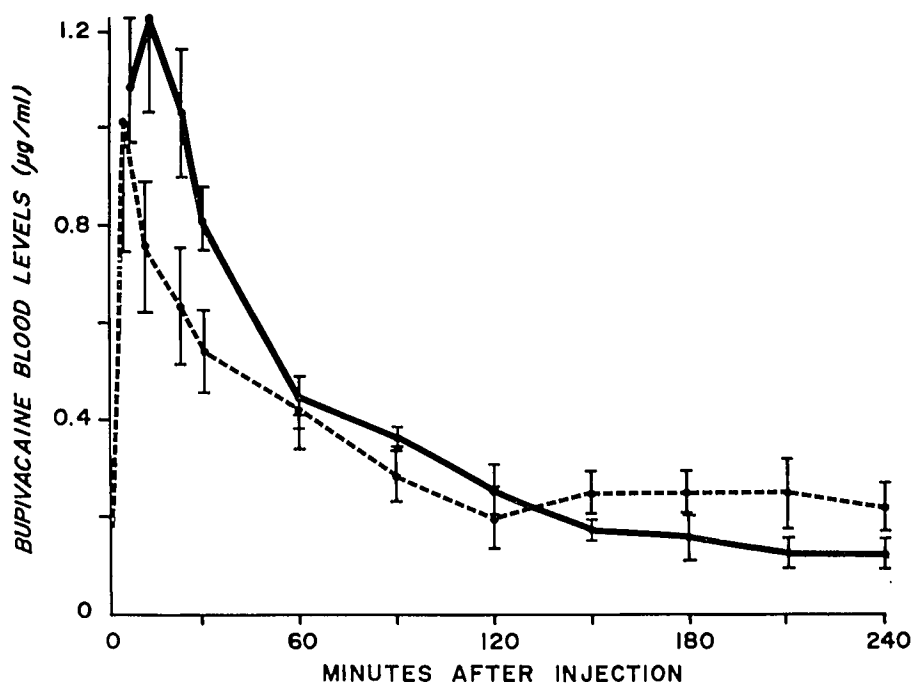
Peak blood levels of bupivacaine in patients receiving 0.125 per cent bupivacaine without epinephrine were nearly sixfold higher than those in patients receiving the same concentration of bupivacaine with epinephrine (table 1; fig. 1). With 0.25 per cent bupivacaine, epinephrine, 1:400,000, had no apparent effect on peak systemic blood levels (table 1; fig. 2). The insignificantly (22 per cent) higher peak blood levels seen in the group not given epinephrine probably reflect the 28 per cent greater initial injection volumes used in that group.

DISCUSSION

Subcutaneous injection of local anesthetics has been associated with peak blood levels lower than those seen following injections in other sites.[5] Presumably, the lower peak blood level is related to a lesser vascularity. Because of the vascular nature of the scalp, injection into the scalp might be expected to result in higher peak blood levels than would subcutaneous injections at other locations. In the present study, scalp infiltration carried out using 0.25 per cent bupivacaine with and without epinephrine, 1:400,000, produced peak blood levels that were in the range found by Moore et al.[6] following lumbar peridural injection of a comparable total dose of bupivacaine, i.e., 150 mg.

The blood levels of bupivacaine resulting in convulsant activity are not known. Venous blood levels of 2.3 $\mu g/ml$[7] and 3.0 $\mu g/ml$[8] have been reported to be associated with convulsions following accidental intravenous injection of bupivacaine during epidural anesthesia. Venous blood levels of local anesthetics are frequently considerably less than are arterial blood levels,[6,9] and are regarded by Scott[10] as a poor indicator of systemic toxicity. Both Jorfeldt et al.[11] and Moore et al.[6,9] failed to observe convulsions in man when arterial blood levels of bupivacaine were less than 4.0 $\mu g/ml$. The peak arterial blood levels observed here were considerably below these levels.

These observations suggest that epinephrine-induced vasoconstriction was antagonized by the higher concentration of bupivacaine, but not by the 0.125 per cent solution. Aps et al.[12] found that 0.125 per cent bupivacaine had vasoconstrictor activity when injected intradermally, whereas 0.5 and 0.25

per cent bupivacaine produced local vasodilatation. Without epinephrine, the peak blood level seen with 0.125 per cent bupivacaine, however, was approximately 75 per cent of the peak blood level seen with the 0.25 per cent solution, indicating the 0.125 per cent bupivacaine did not, itself, cause vasoconstriction.

Blood loss was not measured, but hemostasis was subjectively perceived to be adequate. Because fewer supplemental injections were needed at the higher bupivacaine concentration, 0.25 per cent solutions were felt to be preferable. The use of this higher concentration did not result in systemic toxicity, and allowed detection of an epileptic focus in every patient.

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