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## Hemimacroglossia and Unilateral Ischemic Necrosis of the Tongue in a Long-duration Neurosurgical Procedure

*To the Editor:*—Complications of swelling of the tongue<sup>1,2</sup> and head and neck<sup>3</sup> during neurosurgical procedures have been reported. These reports described insertion of oral airways in adults and procedures with fixed positions of the head and neck for 10 h<sup>1</sup> and 14 h,<sup>3</sup> respectively. Both articles suggested that the anatomy of the venous drainage of the tongue and head had been compromised. Ellis *et al.*<sup>3</sup> suggested that bilateral venous occlusion was necessary to cause venous edema. The same articles suggested possible methods of avoiding the positional complications of macroglossia, *i.e.*, 1) avoid the use of an oral airway (which may compress and occlude venous drainage of the tongue); 2) use a bite block (to avoid tongue compression by the teeth); 3) if a chin support bar is used, check the head support (to avoid soft tissue compression); and 4) avoid extreme flexion of the head against the chest (which may compress airway, tube, and tracheal rings against base of tongue). Despite following these recommendations, we still had a severe case of postoperative macroglossia.

A 56-yr-old woman was operated on for a vertex parasagittal meningioma. The trachea was intubated uneventfully with the use of an oral 7.5-mm endotracheal tube, and the breath sounds were equal. In the supine position, the head was fixed with pin head holders. There was no oral airway inserted. The chin was one finger-breadth above the chest, and extreme flexing of the neck was not evident. No venous congestion of the head and neck were noted before draping. The neurosurgical procedure was uneventful and lasted 8 h.

The patient presented with primarily unilateral macroglossia postoperatively and subsequently developed a partial necrotic slough of the superior and anterior surfaces of the tongue. The unilateral swelling was seen immediately postoperatively. A mild upper airway obstruction was noted in the recovery room for a few h postoperatively. A large hematoma at 24 h and subsequent

necrotic slough at approximately 72 h postoperatively was noted by an ENT consultant. The patient experienced severe difficulty with speech and swallowing for 6 days and an extremely sore tongue for 10 days. She was kept on a liquid diet for 3 weeks until the necrotic areas had healed. No severe airway compromise occurred and there were no long-term complications.

Recognizing that precautions were taken to avoid macroglossia, we postulate two mechanisms for this complication: 1) the right side of the tongue was severely compressed by the tube and a necrotic slough occurred; and/or 2) the swelling of the right side may have resulted from venous occlusion of the tongue base with increased tongue size and secondary compression of the tongue against the tube in a confined space.

Previously suggested means to prevent this complication may not be enough to prevent this problem in every instance. Hence, we would additionally recommend that in long procedures where forward flexing of the neck is required or use of a mandibular support bar is necessary, visual checking of the tongue, head, and neck be performed by the anesthesiologist every h during the procedure. If there is any question of compromise of the venous drainage of these areas, then repositioning of the head and neck should be performed.

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$\beta$ -Adrenoceptor Blockade and Tolerance to Potassium

To the Editor:—Treatment with  $\beta$ -adrenoceptor antagonists is increasingly prevalent among surgical patients. Hypokalemia is a well-recognized complication of therapy with  $\beta$ -2-adrenoceptor agonists.<sup>1</sup> In addition, there is increasing evidence that the adrenergic system plays an important role in the modulation of the cellular uptake of potassium ( $K^+$ ). This process is facilitated by activation and hindered by blockade of  $\beta$ -2-adrenergic receptors.<sup>2</sup> Nevertheless, recent reviews of drug interactions have not considered the potential effects of  $\beta$ -adrenoceptor blockade on serum  $K^+$ .<sup>3,4</sup>

To define the relevance of this potential interaction, we retrospectively compared the disposition of  $K^+$  loading during cardiopulmonary bypass (CPB) by patients treated preoperatively with  $\beta$ -1-selective,  $\beta$ -nonselective, or no adrenoceptor antagonists. It has been our practice over the years to maintain serum  $K^+$  at approximately 4.5 mEq/l during CPB. Cardioplegia solution has  $K^+$  20 mEq/l. Supplemental  $K^+$  is administered if serum  $K^+$  is less than 4.5 mEq/l, in the following manner: mEq  $K^+$  supplement = 0.5 body weight in kg  $\times$  [4.5 – serum  $K^+$

in mEq/l]. A review of 55 consecutive anesthesia and CPB records is summarized in table 1. Significantly less  $K^+$  was given to those patients treated preoperatively with the nonselective  $\beta$ -adrenoceptor antagonist propranolol. This group did not differ from the other two with respect to serum  $K^+$  concentration, urine output, or duration of CPB. This observation is consistent with that of Petch *et al.*,<sup>5</sup> who reported mean serum  $K^+$  levels during CPB of 5.22 mEq/l in ten patients receiving propranolol, compared with 4.16 mEq/l in ten patients on the selective  $\beta$ -1-adrenoceptor antagonist metoprolol. The patients reported here received  $K^+$  supplement aimed at maintaining serum  $K^+$  above 4.5 mEq/l, and we expected that serum  $K^+$  would be similar in the three groups. However, the significantly lower amount of  $K^+$  received by those patients receiving propranolol suggests that its preoperative use might impair extrarenal  $K^+$  disposal during anesthesia.

In other patients receiving propranolol, this interaction deserves attention. For instance, steeper rises in serum  $K^+$  may be expected following intravenous  $K^+$  supplementation in patients treated with propranolol, whereas

TABLE 1.  $\beta$ -Adrenoceptor Blockade and Tolerance to a Potassium Load in Anesthetized Patients

Patient Category	Age (years)	CPB Time (min)	Urine Output (ml · h <sup>-1</sup> · m <sup>-2</sup> body surface area)	Serum K <sup>+</sup> (mEq/l)			Total K <sup>+</sup> Load (mEq)
				Preoperative	CPB		
					Maximum	Minimum	
1. <i>β</i> -selective adrenoceptor blockers (n = 11)	55.4 ± 3.4	110.9 ± 11.0*	1180 ± 252*	3.75 ± 0.10	5.34 ± 0.26	3.66 ± 0.12	30.64 ± 2.12†
2. Nonselective adrenoceptor blockers (n = 12)	59.5 ± 3.8	125.2 ± 14.4‡	828 ± 167‡	4.0 ± 0.15	5.10 ± 0.16	4.0 ± 0.12	20.05 ± 2.52‡
3. No adrenoceptor blockers (n = 32)	58.8 ± 1.8	115.0 ± 7.8§	831 ± 125§	3.9 ± 0.05	5.20 ± 0.13	3.7 ± 0.08	30.10 ± 2.52¶

Results presented as mean  $\pm$  SEM.  
Duncan's multiple range test.<sup>a</sup>  
\* 1. *versus* 2.: No significance.  
† 1. *versus* 2.:  $P < 0.05$ .

‡ 1. *versus* 3.: No significance.  
§ 2. *versus* 3.: No significance.  
¶ 2. *versus* 3.:  $P < 0.05$ .