

## CORRESPONDENCE

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## A Thyroid Shield Can Correct a Laryngeal Mask Airway Air Leak

*To the Editor:*—A patient was anesthetized for an orthopedic procedure and a no. 4 laryngeal mask airway (LMA) was inserted. An air leak around the LMA was observed at approximately 12 cm H<sub>2</sub>O pressure, causing ventilation to be inadequate. I attempted to alleviate the air leak by repositioning the LMA, changing the cuff inflation volume, and repositioning the patient's head, with no success. Accordingly, I prepared to exchange the LMA with an endotracheal tube when the circulating nurse placed a thyroid shield on the patient in preparation for fluoroscopy. The air leak ceased and the case was completed using the LMA.

On several subsequent occasions, I have successfully used a thyroid shield to ameliorate an air leak around an LMA. The patient shown in figure 1 is such an example. The patient had a no. 4 LMA inserted without difficulty, but despite multiple manipulations of the LMA, LMA cuff, and the patient's head, the LMA had a persistent leak at 10–12 cm H<sub>2</sub>O. The patient was breathing spontaneously but entraining room air around the LMA cuff. Placement of a thyroid shield allowed positive pressure ventilation up to 22 cm H<sub>2</sub>O, and the room-air entrainment stopped with spontaneous ventilation. To date, I have had no instances of failure to correct an LMA cuff leak and was able to proceed with the anesthetics planned using an LMA.

The mechanism for this result seems to be that the thyroid shield forces the soft tissue of the anterior pharynx against the LMA cuff. LMA cuff pressure is adjusted to maintain an air leak around the LMA of 20–25 cm H<sub>2</sub>O to lessen the risk of pharyngeal mucosal ischemia.

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## Caution in Performing Epidural Injections in Patients on Several Antiplatelet Drugs

*To the Editor:*—We wish to alert our colleagues about the danger of performing epidural injections in patients who are receiving several antiplatelet drugs. A patient with acute onset of quadriplegia was referred to our hospital with a large cervical epidural hematoma. He developed numbness and weakness of his arms and legs within 30 min after the performance of a cervical epidural steroid injection. At the time of injection, he was taking several antiplatelet drugs (diclofenac, clopidogrel, and possibly aspirin) that had been started after a prior coronary angioplasty. He had a prior uneventful cervical epidural steroid injection 11 days before the incident in question, but it is not clear if he was taking these antiplatelet drugs then. The patient had no history of easy bruisability. His prothrombin time, international nor-



**Fig. 1.** Patient with laryngeal mask airway and thyroid shield in place.

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malized ratio, and partial thromboplastin time were within normal limits, and his platelet count was 160,000/ $\mu$ l but the timing of the last doses of antiplatelet drugs was unclear. He underwent an emergency C3–T3 laminectomy and evacuation of cervical epidural hematoma, which extended from the C2 to C7 vertebral levels, approximately 14 h after the epidural injection. Postoperatively, the strength in the patient's upper extremities improved, but his lower extremities remained paralyzed. Three months after surgery, he had regained his strength in his upper extremities, but the paralysis of his lower extremities remained.

The relative safety of epidural injections in patients on antiplatelet drugs has been established.<sup>1</sup> In the rare case reports of spina-

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hematoma involving antiplatelet drugs, some complicating factors were present: concomitant heparin administration,<sup>2</sup> epidural venous angioma,<sup>2</sup> repeated epidural injections,<sup>3</sup> and technical difficulties in performing the injection.<sup>4-6</sup> The latter factor has been suggested as major risk factor in the development of spinal hematoma.<sup>1,5-8</sup>

The thienopyridine derivatives ticlopidine and clopidogrel represent a newer class of antiplatelet drugs with antiplatelet mechanisms different from that of aspirin. These drugs inhibit induced platelet aggregation, possibly by altering platelet membrane and blocking the interaction between fibrinogen and the membrane glycoprotein receptor, GPIIb/IIIa.<sup>9-11</sup> Clopidogrel and ticlopidine also seem to modulate vascular smooth muscle, resulting in reduced vascular contraction.<sup>12</sup> These drugs inhibit platelet adhesion to the vascular endothelium and shear stress-induced platelet aggregation and secretion.<sup>13</sup> Clopidogrel is 40-100 times more potent than ticlopidine.<sup>14</sup> After discontinuation of clopidogrel therapy, platelet aggregation and bleeding time return to baseline within 5 days. Although there has been no case report of spinal hematoma after a neuraxial block in patients receiving clopidogrel alone, there has been a case of spinal hematoma in a patient on ticlopidine.<sup>6</sup>

The drug package literature for clopidogrel cautions its use with aspirin, warfarin, or nonsteroidal antiinflammatory drugs that have different, and possibly additive, antiplatelet mechanisms.\* Despite the lack of data on the effect of combination of these drugs on duration of antiplatelet inhibition, we recommend that neuraxial blocks be postponed for 5-7 days in patients who are receiving several antiplatelet drugs.

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\* Package literature for Plavix. Princeton, NJ, Bristol-Myers Squibb Company, November 1997.

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