encompasses patients with catastrophic presentations and excludes those with varied presentations. At present, we lack accurate diagnostic tests to confirm or exclude the diagnosis in these mothers with atypical presentations. Therefore, future efforts should be directed towards more clearly delineating the presentation, pathogenesis, diagnosis and outcome of amniotic fluid embolus before abandoning the term altogether.

Sharon Davies MD FRCPC
Toronto, Ontario

References

- 1 Davies S. Amniotic fluid embolus: a review of the literature. *Can J Anesth* 2001; 48: 88–98.
- 2 Clark SL, Hankins GDV, Dudley DA, Dildy GA, Porter TF. Amniotic fluid embolism: analysis of the national registry. *Am J Obstet Gynecol* 1995; 172: 1158–69.

MRI diagnosis of intracranial hypotension

To the Editor:

We read with interest the case report “Postpartum postural headache due to superior sagittal sinus thrombosis mistaken for spontaneous intracranial hypotension” by Chilsholm and Campbell.¹ In that report a patient was eventually diagnosed with a sagittal sinus thrombosis after undergoing a lumbar epidural blood patch (LEBP). Of note, the patient had a magnetic resonance imaging (MRI) of the brain prior to the LEBP reported as normal aside from evidence of venous congestion. MRI is emerging as a useful tool for recognizing intracranial hypotension (IH). MRI studies of patients with IH commonly show on post-contrast image abnormal, intense, diffuse, symmetric, contiguous dural-meningeal (pachymeningeal) enhancement.² This enhancement usually involves much of the supratentorial and infratentorial intracranial dural mater, including the convexities, interhemispheric fissure, tentorium, and foramen cereum. Abnormal leptomeningeal enhancement is usually absent except in more acute states, when abnormal enhancement of the dural venous sinuses may be noted.²

It is unclear in the report if the initial MRI was performed with gadolinium contrast. If this was the case, it would have been unusual to pursue a diagnosis of IH with a MRI showing venous congestion with no post-contrast pachymeningeal enhancement. This would have alerted the clinician about the unlikely diagnosis of IH for the etiology of the patient's

headache and avoided her an unnecessary lumbar epidural blood patch.

Étienne de Médicis MD MSC FRCPC
Oscar A. de Leon-Casasola MD

References

- 1 Chilsholm ME, Campbell DC. Postpartum postural headache due to superior sagittal sinus thrombosis mistaken for spontaneous intracranial hypotension. *Can J Anesth* 2001; 48: 302–4.
- 2 Bakshi R, Mechtler LL, Kamram S, et al. MRI findings in lumbar puncture headache syndrome: abnormal dural-meningeal and dural venous sinus enhancement. *Clin Imaging* 1999; 23: 73–6.

There is no direct relationship between PI response and smooth muscle contraction of rat trachea stimulated by α -agonists

To the Editor:

α -Adrenoceptor agonists are commonly used during anesthesia to cause vascular smooth muscle contraction through the activation of phosphatidylinositol (PI) response. Meurs *et al.* have demonstrated evidences for a direct relationship between PI response and airway smooth muscle contraction induced by muscarinic agonists.¹ Although α_1 α -adrenoceptors exist in the airway smooth muscle,² the signal transduction of α_1 α -adrenoceptors in the airway is not fully understood. The present study was designed to clarify whether α_1 α -adrenoceptor agonists could stimulate PI response, resulting in an induction of airway smooth muscle contraction of rat trachea.

Rat tracheal rings were suspended between two stainless hooks in Krebs-Henseleit (K-H) solution. Contraction was induced with carbachol (a muscarinic agonist), phenylephrine and norepinephrine. The tracheal slices were incubated in K-H solution containing LiCl and ^3H *myo*-inositol in the presence of carbachol, phenylephrine or norepinephrine. ^3H inositol monophosphate (IP_1)^{3,4} a degradation product of PI response, was measured.

Carbachol caused tracheal ring contraction at a dose of 0.1 μM or greater, whereas phenylephrine or norepinephrine could not cause the contraction. Carbachol caused IP_1 accumulation at a dose of 1 μM or greater, and phenylephrine and norepinephrine caused IP_1 accumulation at doses of 100 μM and 10 μM , respectively. There was a direct relationship