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.

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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 fax cerebri. 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.

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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 $_1$ α-adrenoceptors exist in the airway smooth muscle,[2] the signal transduction of $_1$ α-adrenoceptors in the airway is not fully understood. The present study was designed to clarify whether $_1$ 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 ^{3}[H]*myo*-inositol in the presence of carbachol, phenylephrine or norepinephrine. ^{3}[H]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

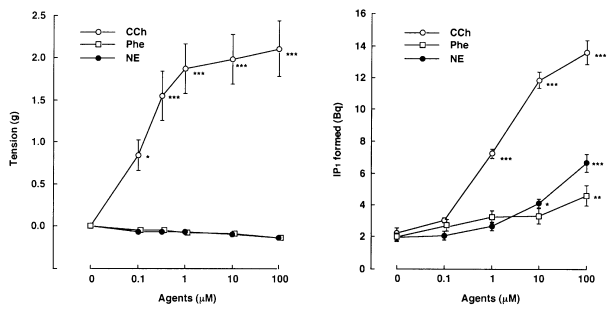

FIGURE The effects of carbachol (CCh), phenylephrine (Phe) and norepinephrine (NE) on resting tension and IP_1 accumulation of rat trachea (mean ± SE; n=8). Bq: becquerel, IP_1: inositol monophosphate, $*P<0.05$, $***P<0.001$ *vs* 0.

between PI response and airway smooth muscle contraction stimulated by carbachol. However, neither phenylephrine nor norepinephrine could cause tracheal ring contraction in spite of increased IP_1 accumulation, suggesting that there is no direct relationship between PI response and airway smooth muscle contraction stimulated by $_1$ α-adrenoceptor agonists in rat trachea.

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IV butorphanol reduces analgesia but not pruritus or nausea associated with intrathecal morphine

To the Editor:

Intrathecal morphine provides excellent postoperative analgesia, but it is often accompanied by troublesome adverse effects, i.e., pruritus and nausea.[1,2] Some investigators have reported that epidural butorphanol in combination with epidural morphine effectively reduces adverse effects without reversing analgesia.[3–5] We tested the hypothesis that prophylactic *iv* butorphanol might reduce pruritus and nausea associated with intrathecal morphine.

In this randomized prospective double-blind pilot study, 20 patients undergoing total abdominal hysterectomy received spinal anesthesia with tetracaine 15 mg, and morphine 0.15 mg. Fifteen minutes after the administration of spinal anesthesia the patients received saline 1 mL *iv*, (Group A) or butorphanol 2 mg *iv*, (Group B). The intensity of pain, pruritus and nausea at three, five, seven and 24 hr after spinal anesthesia, the time of first request for additional analgesics (duration of analgesia), and the consumption of antipruritics and antiemetics were assessed.

The intensities of pain, pruritus and nausea are shown in the Table. The intensity of pain at three, five, and seven hours after spinal anesthesia in group B was significantly higher than that in group A. The duration of analgesia in group B (1126 ± 80 min) was significantly shorter than that in group A (1499 ± 159 min). There was no significant difference in the intensity of either pruritus or nausea or in consumption of antipruritic or antiemetic drugs between the groups.

Based on these preliminary results, we conclude that prophylactic *iv* butorphanol reduces analgesia but not pruritus or nausea associated with intrathecal morphine.

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