FIGURE

cliniques en une dizaine de jours et la disparition du pneumatocèle, constatée par une contrôle TDM, à la troisième semaine.

Une injection accidentelle d'air dans l'espace sous-arachnoïdien peut survenir alors même qu'aucune extériorisation de LCR ne soit notée. Cette situation particulière peut s'expliquer en obstétrique par une très faible marge de manoeuvre liée à l'étroitesse de l'espace péri-dural encore accentuée par le bombement de la dure-mère lors des contractions. Le pneumatocèle, sans soustraction de LCR, crée alors les conditions d'une hypertension intracrânienne. Les céphalées, assez banales au décours des anesthésies locorégionales obstétricales, sont ici d'apparition immédiate et non calmées par le décubitus dorsal.²

Bien que le traitement soit conservateur une évocation précoce du diagnostic est utile permettant d'éviter immédiatement l'emploi du protoxyde d'azote en cas d'anesthésie générale et plus tardivement épargnant à la patiente de nombreuses explorations diagnostiques. Avantages et inconvénients de mandrins gazeux et liquides peuvent être rediscutés à la lumière de ces observations.³

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Propranolol protection from bupivacaine toxicity

To the Editor:

We read with interest Drs. Kinney, Kambam and Wright's article which indicated the propranolol pretreatment reduced the incidence of cardiorespiratory arrest in Sprague Dawley rats given injections of bupivacaine plain but not bupivacaine containing epinephrine.¹ These findings were "unexpected" as the authors hypothesized that the negative inotropic effect of propranolol would potentiate the cardiodepressant effect of bupivacaine. We would like to offer the following explanation for their results.

Bupivacaine, like most local anaesthetics, decreases sodium and calcium conductance in nerves and muscles by blocking ion channels.² Amide anaesthetics are thought to bind to a receptor located inside the channel pore. In order for the blocker to access its binding site, the channel must be in the open or activated state. Increasing the frequency of channel opening, increases the rate of channel blockade. This effect is referred to as "use-dependence" of channel blockade.³ Use-dependency is thought to be the basis for the therapeutic effect of several antiarrhythmic drugs which selectively suppress high frequency discharges but allow the conductance of normal action potentials. Recovery from blockade may also be use-dependent if the channel must be in the open state for the drug to dissociate from its binding site. Closure of the channel may trap the local anaesthetic and delay the recovery from blockade.⁴

It is possible that propranolol reduced the frequency of sodium or calcium channel opening thereby reducing the number of available bupivacaine receptors. Epinephrine would increase channel activity and antagonize the protective effects of propranolol. Use-dependence would explain the protective effect of propranolol given *prior* to the injection of bupivacaine, it would also suggest that propranolol would increase toxicity if it were given *after* the drug had bound to its receptor.

In summary, the protective effect of propranolol given prior to the injection of bupivacaine plain may be explained by use-dependency of channel blockade. Proprano-

lol would likely be harmful if used for the treatment of bupivacaine overdose.

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Long-term interpleural analgesia using a subcutaneous implantable infusion system

To the Editor:

Interpleural analgesia has been used for postoperative analgesia as well as treatment for malignant and post-traumatic pain.¹⁻³ We report the successful treatment of chronic unilateral non-malignant chest pain utilizing a fully implantable delivery system for interpleural analgesia.

A 51-yr-old male was referred with a six-month history of constant searing unilateral chest wall pain following tube thoracostomy and tetracycline pleurodesis. Successful placement of a percutaneous interpleural catheter was performed and the patient underwent a series of interpleural injections with 20 ml bupivacaine 0.5% with epinephrine 1:200,000. A dome subcutaneous port with Groshong catheter was implanted subcutaneously anterior to the anterior axillary line at T₈-T₉ to enable continued outpatient injections. The catheter was tunnelled posteriorly; the tip was located posteromedially at T₇-T₈ (Figure). Placement of this catheter was complicated by the development of a 15-20% pneumothorax which required tube thoracostomy for three days. Three months after the last injection the catheter and port were removed uneventfully.

FIGURE Intraoperative radiograph showing interpleural catheter. Arrow points to radioopaque column of dye in the right paravertebral gutter.

Although the exact mechanisms of interpleural analgesia have yet to be fully elucidated, one can speculate that the local anaesthetic diffused across the pleura to block the intercostal somatic nerves. One might also expect that neurons in the thoracic sympathetic chain might be affected by the local deposition of anaesthetic. We suggest that the progressive improvement in the pain relief our patient exhibited lends credibility that some, if not all, of his chest wall pain was due to a sympathetic mediated cause, i.e., causalgia secondary to the chemical pleurodesis. We chose to use an implantable delivery system to reduce the likelihood of bacterial contamination and infection of the pleura. The system employed is more commonly used for the chronic ambulatory administration of antibiotics or chemotherapeutic agents.

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