

Headache after Attempted Epidural Block

The Role of Intrathecal Air

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Background: Postmeningeal puncture headache (PMPH) is typically attributed to the loss of cerebrospinal fluid (CSF). However, when it occurs after an attempted epidural puncture, it may be due to either CSF loss or, potentially, to the subarachnoid injection of air used as a part of "loss-of-resistance" testing. This study was performed to examine the relation between intrathecal air and PMPH.

Methods: Using a loss-of-resistance test with an air-filled ($n = 1,812$; air group) or saline-filled ($n = 1,918$; saline group) syringe, epidural block was performed in patients with acute or chronic pain. The dura was judged to be perforated not only when backflow of CSF was recognized in the needle but also when signs and symptoms solely attributable to meningeal perforation were seen, such as high spinal blockade or severe motor blockade. The incidence, onset time, and duration of PMPH in the air and saline groups were compared. In all patients with signs of meningeal perforation, brain computed tomography was examined.

Results: The incidence of PMPH in the air group (32 cases) was significantly higher than that in the saline group (5 cases), although the occurrences of meningeal perforation between the air (48 cases) and saline (51 cases) groups did not differ significantly. Intrathecal air bubbles were detected on brain computed tomography in both the deep supraspinal structures such as the ventricles, Sylvian fissures and cisterns, and the superficial subarachnoid space in 30 of 32 patients with PMPH in the air group, whereas no intrathecal air bubbles were seen in the saline group. PMPH was significantly more rapid in onset and shorter in duration in the air group than that in the saline group.

Conclusions: The use of air for loss-of-resistance testing during epidural block was associated with a higher incidence of PMPH, which might be attributable to subarachnoid air injection and CSF leakage. (Key words: Computed tomography; loss-of-resistance test; meningeal perforation; supraspinal structure.)

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Received from the Department of Anesthesiology, Niigata University School of Medicine, Niigata, Japan. Submitted for publication January 29, 1997. Accepted for publication August 21, 1997.

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POSTMENINGEAL puncture headache (PMPH) after spinal block has long been considered to be caused by low cerebrospinal fluid (CSF) pressure subsequent to CSF leakage.¹⁻⁴ Similarly, after epidural block, we sometimes encounter severe headache, when the dura is unintentionally perforated by a needle tip. Therefore, headache occurring after epidural block has also been attributed to CSF leakage.⁵⁻¹⁰

To confirm that the needle tip is in the epidural space, loss-of-resistance is usually tested with an air- or saline-filled syringe.^{10,11} If air in the syringe is introduced into the intrathecal space during the block procedure, it may stimulate central nervous system structures and produce headache, in the same manner as intrathecal air introduced during pneumoencephalography causes headache.¹² In fact, several cases of headache immediately after epidural block using the loss-of-resistance method with an air-filled syringe have been reported and were attributed to intrathecal air bubbles introduced during the procedure.¹³⁻¹⁵ Headache due to intrathecal air injection during spinal anesthesia has been also demonstrated.¹⁶

To confirm this thesis, we studied the relation between intrathecal air and PMPH after epidural block and examined the air in the CSF.

Methods

The study was performed after receiving the approval of the University Human Research Committee, and informed consent was obtained from 2,975 patients. Cervical, thoracic, or lumbar epidural block (3,730 total; excluding caudal block) was performed using the loss-of-resistance method¹⁰ with a Tuohy needle (15 cm, 20 gauge) once in 2,823 patients and two to seven times in 152 patients (907 blocks) with low back pain syndrome or *herpes zoster*. The blocks were performed in the lateral spinal tap position by the same person (S.A.) in patients consulting our pain service between 1989 and 1996.

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In 1,812 blocks, a 5-ml glass syringe was internally wetted and filled with 4–5 ml air. Loss of resistance was then used to enter into the epidural space (air group). In the other 1,918 blocks, a syringe filled with 4–5 ml saline was used (saline group). In the saline group, air was completely excluded from the saline-filled system (syringe and needle) to strictly avoid injecting even one air bubble. A total of 1–5 ml air or saline was injected during the loss-of-resistance test. After loss of resistance was confirmed, 10 ml 0.5% lidocaine was applied in each block (table 1).

The loss-of-resistance technique was altered on a weekly basis; that is, during 1 week patients were entered into the air group, whereas during the next week only saline-filled syringes were used (saline group). Follow-up evaluation of headache was carried out by physicians excluding the first author (that is, the second to fourth authors), and the grouping was blinded to these physicians and to the patients.

After the block procedure, patients were allowed to rest in a supine position and were observed for at least 60 min in the nerve block room. Blood pressure and heart rate were monitored every 5 min. The patients were observed for signs and symptoms including headache (according to a questionnaire, "have you headache after the epidural block?"), muscle weakness (Bromage scale), and loss of pain sensation (pin-prick test). All patients were asked to call us when headache or other neurologic symptoms occurred within 10 days after the block (returning home).

In the present study, the dura was judged to be perforated not only when backflow of CSF was recognized in the needle but also when signs and symptoms solely attributable to meningeal perforation were seen, such as high spinal blockade (apnea) or severe motor blockade (grade 3 in Bromage scale). When CSF backflow was evident, the block procedure was discontinued (lidocaine was not injected). In all the patients with evident and suspected meningeal perforation, brain computed tomography (CT) was immediately performed. Further, when intrathecal air was found, CT was repeated 3 days later.

Although criteria for PMPH (due to CSF leakage) were proposed by Driessen *et al.*,¹⁷ all headaches that newly developed after the loss-of-resistance test were considered PMPH. However, preexisting headaches were excluded. When PMPH occurred, the patient was hospitalized, observed at rest, maintained in a slight head-down position, infused with 5% glucose solution,^{9,10} medi-

cated with rectal diclofenac (50 mg), or all three until PMPH disappeared.

Values were expressed as the median and range. Numeric data were analyzed using the chi-square test, Fisher's exact probability test, or the Mann-Whitney U test, and *P* values < 0.05 were considered significant.

Results

The meninges were punctured in 48 and 51 (2.6% and 2.7%, not significant) patients in the air and saline groups, respectively. Cerebrospinal fluid backflow occurred in 30 and 32 patients in the air and saline groups, respectively; the punctures in the remaining 18 and 19 patients in the air and saline groups were judged on signs and symptoms after local anesthetic injection (high spinal blockade, severe motor blockade, or both). Thirty-two of 48 (66.7%) and 5 of 51 (9.8%) patients with evidence of meningeal perforation in the air and saline groups (*P* < 0.01), respectively, reported PMPH (1.8% and 0.3%, respectively; *P* < 0.01 by Fisher's exact probability test). No patient without evidence of meningeal puncture reported PMPH (table 2).

In the air group, supraspinal intrathecal air bubbles were found on brain CT examination in 39 of 48 patients with signs of meningeal perforation. In 30 of 32 patients with PMPH in the air group, bubbles were spread widely throughout both the deep supraspinal structures, such as the lateral (17 cases), third (19 cases), and fourth (21 cases) ventricles; Sylvian fissures (23 cases); and the great (20 cases) and chiasmatic (20 cases) cisterns (deep supraspinal air bubbles); and over the convexity of the brain (30 cases; superficial supraspinal air bubbles) (fig. 1). Intrathecal air bubbles were found in 16 of 18 patients without obvious CSF backflow in the air group. Nine patients in whom only a few air bubbles in the superficial subarachnoid space were detected did not report PMPH. However, no supraspinal air bubbles were detected in two patients with PMPH. Brain CT examinations showed that the air bubbles were absorbed spontaneously within 3 days in all patients (table 2).

In the saline group, there was no intrathecal air in any of the 51 patients with signs of meningeal perforation, nor in the 5 patients who reported PMPH (table 2).

Onset of PMPH in the air group was significantly more rapid (*P* < 0.01 by the Mann-Whitney U test) and the duration was significantly shorter (*P* < 0.05 by the

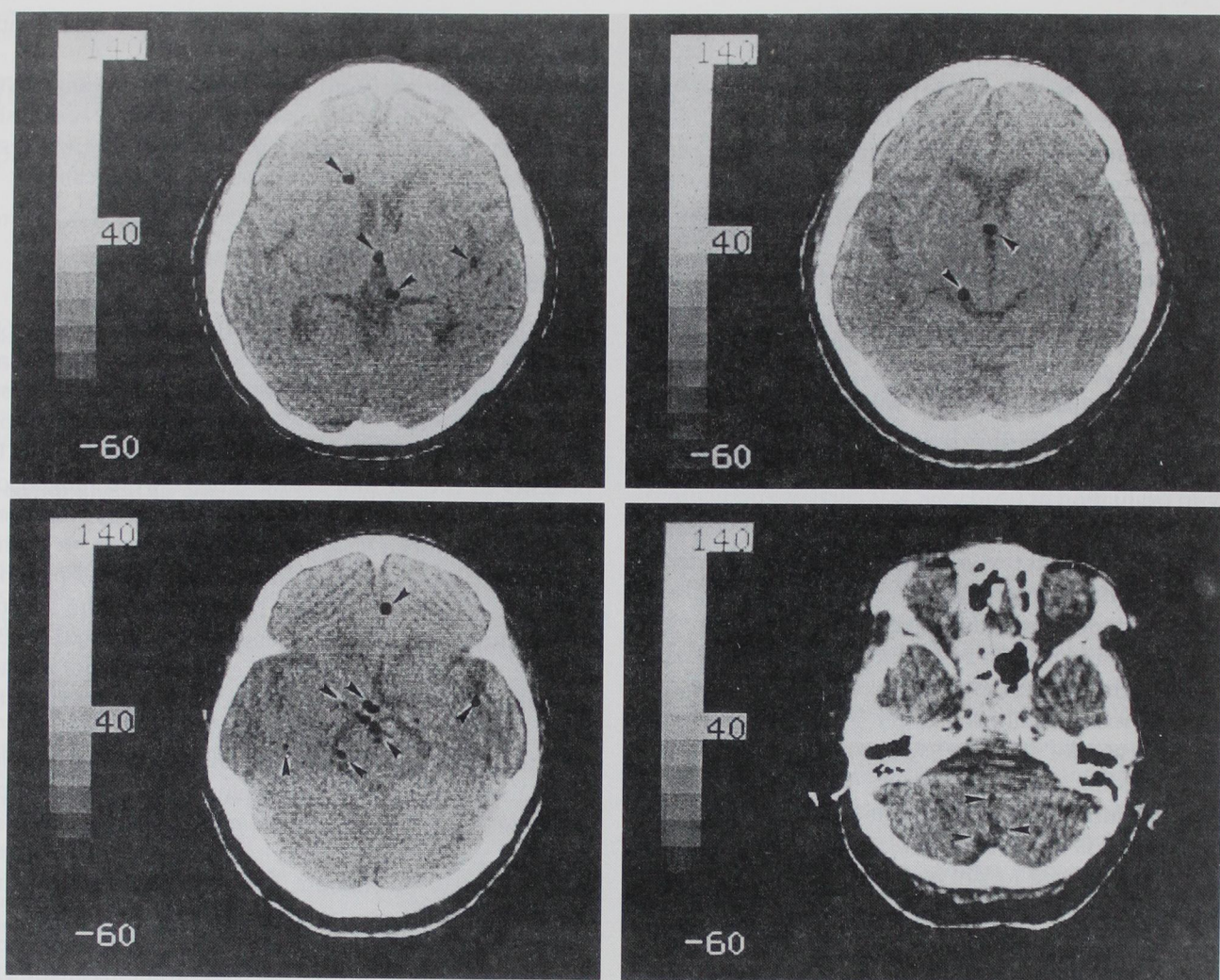


Fig. 1. Brain computed tomographic (CT) images in the patient with postmeningeal puncture headache (PMPH) in the air group. The CT was performed immediately after onset of PMPH. Intrathecal air bubbles (black spots indicated by arrowheads) were found at the deep-supraspinal structures and superficial surface of the brain, including the lateral, third, and fourth ventricles; Sylvian fissures; and great and chiasmatic cisterns.

Mann-Whitney U test) than those in the saline group (figs. 2, 3).

There were no significant differences in age (Mann-Whitney U test), sex, and number of blocks (by the chi-square test) between the two groups (table 1). No pyrexia, leukocytosis, elevation of serum C-reactive protein, or increased erythrocyte sedimentation rate was observed in any of 99 hospitalized patient with PMPH after the block procedure.

Discussion

In most patients with PMPH in the air group (table 2), air bubbles were found in the intrathecal space.

Intrathecal air is known to produce headache,¹² thus suggesting that intrathecal air is one cause of PMPH after epidural block using the loss-of-resistance method. In the saline group, PMPH was also seen, although no intrathecal air was found. Therefore, PMPH in the saline group was considered due to CSF leakage. Based on these findings, it is suggested that there are two different mechanisms of PMPH after epidural block: CSF leakage and intrathecal air.

The incidence of PMPH in patients with signs of meningeal perforation in the air group was significantly higher (more than six times) than that in the saline group, although the occurrence of meningeal puncture did not significantly differ between the groups. The

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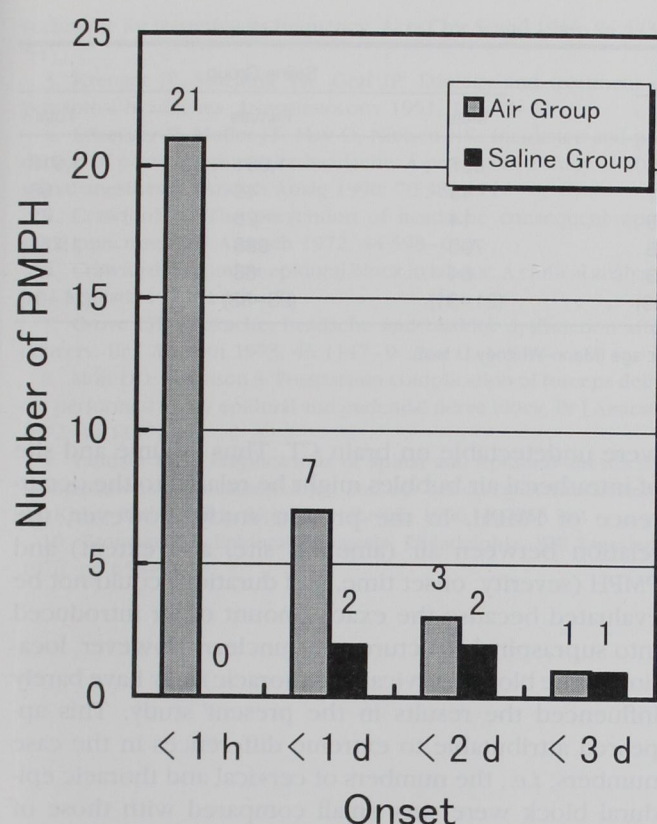


Fig. 2. Onset time of postmeningeal puncture headache (PMPH). The onset time of PMPH was significantly more rapid ($P < 0.01$ by the Mann-Whitney U test) in the air group than in the saline group.

existence of many patients with PMPH due to intrathecal air injection might contribute to the high incidence of PMPH after unintentional meningeal puncture during epidural block. In addition, intrathecal air was shown in 16 of 18 patients without obvious CSF backflow in the air group.⁹

Onset time of PMPH in the air group was short, whereas that in the saline group was long (fig. 1). Air bubbles might have been vigorously pushed out of the syringe, when resistance was suddenly released during loss-of-resistance test, and rapidly migrate in the CSF, reaching the deep supraspinal structures (even though the patients were in the lateral spinal tap position and then supine). As a result, air bubbles may directly stimulate the central nervous system structures to produce PMPH immediately after the block. However, air remaining within the spinal arachnoid space might also migrate upward after position change and might induce or aggravate headache. Thus two types of PMPH due to

intrathecal air injection and CSF leakage showed similar aggravation when the patient changed to an erect or sitting position. This may be due to the fact that air moves upward while CSF flows downward.

In contrast, a rather long onset time might be required for the onset of PMPH due to CSF leakage, because the needle hole was too small for leakage to induce low CSF pressure syndrome within 1 h.⁹ Therefore, the onset time would be expected to be prolonged in the saline group. There might be other mechanisms causing PMPH, such as increased CSF pressure or infection. However, 1–5 ml intrathecal air is considered insufficient to increase CSF pressure significantly,¹⁸ and there were no signs and symptoms suggesting meningitis or encephalitis in the present study.

Duration of PMPH in the air group varied, whereas that in the saline group was prolonged (fig. 2). These results suggest that there were multiple factors influencing the duration of PMPH. Therefore, PMPH with rapid onset and short duration (within 1 h) was consid-

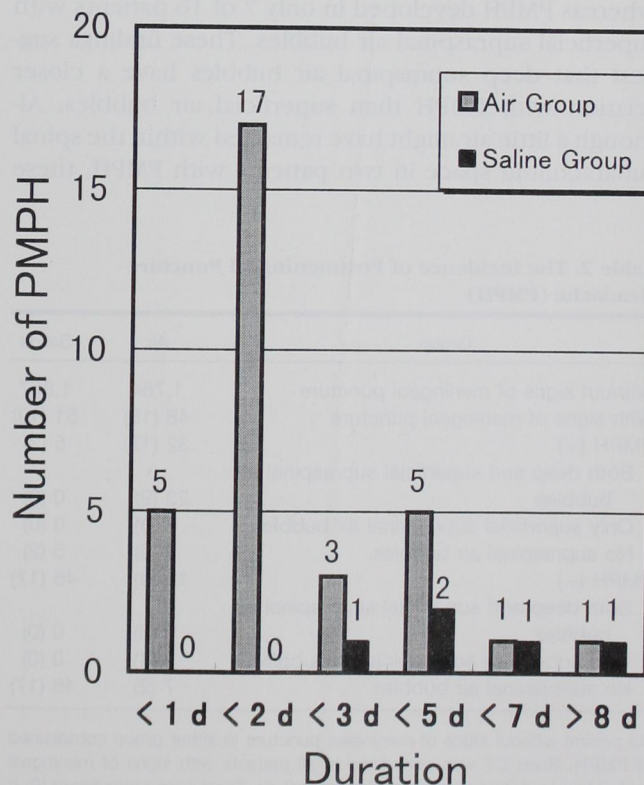


Fig. 3. Duration of postmeningeal puncture headache (PMPH). The duration of PMPH was significantly shorter ($P < 0.05$ by the Mann-Whitney U test) in the air group than in the saline group.

Table 1. Summary of Patients Receiving Epidural Block

| | Air Group | | | Saline Group | | |
|----------|-----------|---------|---------|--------------|---------|---------|
| | Male | Female | Total | Male | Female | Total |
| No. | 845 | 967 | 1,812 | 857 | 1,061 | 1,918 |
| Cervical | 39 | 42 | 81 | 43 | 45 | 88 |
| Thoracic | 19 | 16 | 35 | 14 | 28 | 42 |
| Lumbar | 787 | 909 | 1,696 | 790 | 988 | 1,778 |
| Age (yr) | 59 | 67 | 63 | 64 | 66 | 65 |
| (range) | (18–89) | (16–84) | (16–89) | (21–91) | (23–88) | (21–91) |

There were no significant differences in block numbers, gender (chi-square test), or age (Mann-Whitney U test).

ered to be produced by intrathecal air, which will be absorbed spontaneously within a few days with uneventful recovery. Late-onset PMPH (more than 1 h) is thought to be caused by CSF leakage. When the onset is rapid and duration long, two types of PMPH due to intrathecal air injection and CSF leakage might coexist.

It was noted that all patients demonstrating intrathecal air in the deep supraspinal structures such as the ventricles, Sylvian fissures, and cisterns reported PMPH, whereas PMPH developed in only 7 of 16 patients with superficial supraspinal air bubbles. These findings suggest that deep supraspinal air bubbles have a closer relation with PMPH than superficial air bubbles. Although a little air might have remained within the spinal subarachnoid space in two patients with PMPH, these

were undetectable on brain CT. Thus volume and site of intrathecal air bubbles might be related to the occurrence of PMPH. In the present study, however, the relation between air (amount, site, and extent) and PMPH (severity, onset time, and duration) could not be evaluated because the exact amount of air introduced into supraspinal structures was unclear. However, location of the block (cervical and thoracic) may have barely influenced the results in the present study. This appeared attributable to extreme differences in the case numbers; *i.e.*, the numbers of cervical and thoracic epidural block were very small compared with those of lumbar epidural block.

Diagnoses of the cause of PMPH may be possible from the onset time and duration, as described before. For PMPH due to CSF leakage, treatments including infusion and an epidural blood patch have been described.^{9,10} However, these treatments may not be effective for PMPH due to intrathecal air. When PMPH occurred immediately after the block and intrathecal air was found by brain CT examination, patients can probably be treated only by a head-down position. When PMPH was protracted, treatments such as a blood patch and infusion may be required.

In conclusion, two types of PMPH after epidural block were suggested by the present study: one is attributable to intrathecal air injection and the other is the result of CSF leakage. However, both types of PMPH may occur simultaneously. The use of air for loss-of-resistance testing during epidural block was associated with a higher incidence of PMPH.

Table 2. The Incidence of Postmeningeal Puncture Headache (PMPH)

| Group | Air | Saline |
|---------------------------------------------------|---------|---------|
| Without signs of meningeal puncture | 1,764 | 1,867 |
| With signs of meningeal puncture | 48 (18) | 51 (19) |
| PMPH (+) | 32 (12) | 5 (2) |
| Both deep and superficial supraspinal air bubbles | 23 (9) | 0 (0) |
| Only superficial supraspinal air bubbles | 7 (3) | 0 (0) |
| No supraspinal air bubbles | 2 (0) | 5 (2) |
| PMPH (–) | 16 (6) | 46 (17) |
| Both deep and superficial supraspinal air bubbles | 0 (0) | 0 (0) |
| Only superficial supraspinal air bubbles | 9 (4) | 0 (0) |
| No supraspinal air bubbles | 7 (2) | 46 (17) |

No patient without signs of meningeal puncture in either group complained of PMPH. Brain CT was performed in all patients with signs of meningeal perforation to detect supraspinal intrathecal air. There was a significant ($P < 0.01$ by Fisher's exact probability test) difference in the incidence of PMPH between the groups, although the occurrences of meningeal puncture between the groups did not significantly differ. Numbers in the parentheses represent patients without obvious cerebrospinal fluid backflow.

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