Lumbar puncture is usually required for the diagnosis of this condition, and it is unfortunate that this procedure may make the headache worse by increasing CSF leakage.

The vast majority of our medical colleagues have never heard of epidural blood patches for the treatment of low CSF pressure headache and are not aware of its potential value in treating this disabling condition, whether it be of a spontaneous onset, or following lumbar puncture.

We see the benefit of an epidural blood patch as being two-fold in these chronic patients: firstly, it may seal the CSF leak which caused the headache in the first instance; secondly, it will remove the iatrogenic component introduced by the performance of lumbar punctures.

Epidural blood patch is now considered to be a very safe procedure which will produce dramatic relief of low-CSF pressure headache in the majority of cases. The use of this procedure should be considered more often in patients with symptoms suggestive of low CSF pressure.

P. B. Gaukroger FFARCS
Department of Anaesthesia
Adelaide Children's Hospital
South Australia
P. Brownridge FFARCS
Department of Anaesthesia
Flinders Medical Centre
South Australia

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REPLY

I would like to thank Drs. Gaukroger and Brownridge for their interesting comments and observations. I agree with most of their comments but I would like to recommend caution regarding their implied widespread use of epidural blood patch for treatment of chronic headache until more definitive research on the mechanism, aetiology and diagnosis of these headaches is

done. The diagnosis of low CSF pressure headache is difficult to make in the absence of a diagnostic lumbar puncture which may, itself, produce such a headache. Consequently, the diagnosis is usually a tentative one of exclusion after evaluating headache patients whose symptoms resemble patients with diagnosed low CSF pressure headache or post-dural puncture headache. It is in those selective patients with negative neurological and radiological studies that I would recommend consideration of an epidural blood patch.

Further, Drs. Gaukroger and Brownridge have correctly postulated that suggested mechanisms of low-CSF pressure headaches may be (1) diminished CSF production, (2) increased CSF absorption and (3) CSF leakage. However, I would like to question their implication that the mechanism of action of epidural blood patch in treating post-dural puncture or low-CSF pressure headache is "to seal the CSF leak which caused the headache in the first instance." Clinical experience indicates that the pain relief is immediate (five minutes or less) and even though the CSF leak is sealed by the blood introduced into the epidural space, the choroid plexus takes several hours to restore the CSF volume to its original state. Consequently, the mechanism of action of epidural blood patch is unclear at this time. One may have to examine newer biochemical hypotheses for a possible answer to the mechanism of action of epidural blood patch.

Winston C.V. Parris MD
Pain Control Center, Department of Anesthesiology
Vanderbilt University Hospital
Nashville, Tennessee 37232

Evaluation of the Humphrey A.D.E. system

To the Editor:

I read with interest the article by Artru and Katz.¹ The authors contribute clinically useful material regarding the safe use of the multipurpose A.D.E. system^{2,3} for both spontaneous and controlled ventilation, thereby answering some of the questions posed in an Editorial by Byrick and Rose.⁴ Quite clearly the A.D.E. system needed careful evaluation, especially as our original observation of its greater efficiency in its Mapleson A mode for spontaneous respiration over the definitive Magill (Mapleson A) system was surprising.² However, Dixon *et al.* confirmed our findings⁵ and we are further reassured to see that, yet again, our low values as to the mean fresh gas requirements of the A.D.E. system² have been independently confirmed by Artru and Katz.

I would like to emphasize two points of clinical value. Firstly that we have not observed any differences in the function of the dual lever system (studied by Artru and Katz) and the more recent simpler single lever version.^{2,3} Secondly the FGF of 51 ml · kg⁻¹ · min⁻¹ for spontaneous respiration reported by ourselves² was a mean value; by definition this flow will cause rebreathing in some

patients. In clinical practice we have observed that the lean more muscular males tended to rebreathe at the mean flow and therefore require higher FGFs; Artru and Katz confirm these trends and report a mean 20 per cent increase in FGF for males compared to females. They also quite rightly point out that only by taking the mean value plus two standard deviations can one be sure that there will be no rebreathing in nearly all patients (97.2 per cent). They thus recommended an overall FGF of $66 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, a value very close to that calculated in retrospect from our own results ($62 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$).² On the other hand using the same criteria, the Bain system required $192 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ some three times greater and hence much less economical!

Since Artru and Katz have recommended that a FGF of $66 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ is a safe flow for spontaneous respiration, a suggestion we made in the discussion of our paper on the use of the A.D.E. system during controlled ventilation becomes more pertinent.³ On changing from controlled to spontaneous respiration we wrote: "Only minor adjustment in FGF between 50 and $70 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ is necessary, though some anaesthetists may find it convenient to leave the FGF at the higher flow, allowing a good margin of safety during spontaneous respiration." Since Artru and Katz have also shown that the FGF of $70 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ is satisfactory for controlled ventilation, our recommendation would appear all the more valid as this flow is higher than even their conservative suggestion for spontaneous respiration. Thus, once set, the FGF need not be adjusted. Such a recommendation would make the A.D.E. system even simpler to use; with the FGF set at $4.9 \text{ L} \cdot \text{min}^{-1}$ (calculated for a 70 kg patient) all an anaesthetist needs to do is to set the lever up for spontaneous or manual ventilation, and down for controlled ventilation. As has been pointed out before, the inclusion or exclusion of the reservoir bag, exhaust valve and ventilator is automatic.

Bain makes a plea for a system that is safe, simple, economical and convenient;⁶ may I suggest that this has been achieved. Since Artru and Katz make no comment on any clinical advantages the A.D.E. system may have, it remains for anaesthetists to evaluate any such merits during routine anaesthesia and to compare any advantages of the A.D.E. system to current alternative systems such as the Bain.

David Humphrey MBBS DA
Dept of Physiology
University of Natal
P.O. Box 17039
4013 Congella
Durban, South Africa

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REPLY

Thank you for the opportunity to reply to Dr. Humphrey's letter.

I appreciate Humphrey's comments on our recent article and his summary of previously reported work with the A.D.E. breathing system. The second portion of his letter makes one recommendation and poses a question on clinical use of the A.D.E. system. He states that the system is safe and proposes that it may be more convenient to maintain a fresh gas flow (FGF) of $70 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ for both spontaneous and controlled ventilation. While our data suggest that a FGF of $70 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ should be safe for virtually all patients, I believe that the prudent anaesthesiologist should not rely solely on predetermined FGF values but also should employ a reliable means to indicate that the patient's ventilation is adequate. As regards clinical use of the A.D.E. system, I have experienced no mechanical difficulties with it. The system is simple and convenient to use and is far less bulky than the traditional circle systems used at our hospital.

Alan A. Artru MD
Department of Anesthesiology, RN-10
University of Washington School of Medicine
Seattle, WA 98195