CORRESPONDENCE

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In Reply:—The comment by Cold *et al.* to our article "Cerebral Vasoconstriction by Indomethacin in Intracranial Hypertension"[1] addresses important problems within neuro-intensive care: Should posttraumatic brain swelling be treated with pharmacologic vasoconstriction? If so, what drug and dosage should be chosen? In our study of induced intracranial hypertension in piglets,[1] we found that indomethacin reduced intracranial pressure (ICP) by constriction of precapillary resistance vessels only when cerebral blood flow (CBF) was reduced to a level where progressive changes in $CavO_2$, vpH, and electroencephalography occured, indicating ischemia. We concluded that our data did not support the use of indomethacin in patients with intracranial hypertension due to head injury. In their comment, Cold *et al.* refer to clinical publications not available at that time. Some of these are still in press, but we had the opportunity to review the manuscripts.

The study by Biestro *et al.*[2] included 10 patients with severe head injuries and raised ICP. The patients were given an indomethacin bolus (50 mg) followed by a continuous infusion (21.5 ± 11.4 mg/h). The authors measured significant decreases in ICP and increases in cerebral perfusion pressure (CPP) during the treatment, but no data were given regarding CBF, $CavO_2$, vpH, electroencephalography, or other physiologic parameters. Four of the ten head-injured patients were reported dead, but no data regarding the cause or the time of death were presented.

In the study by Dahl *et al.*,[3] physiologic parameters that included ICP, CPP, CBF, and $CavO_2$ were studied in 14 patients with head injuries (mean ICP 14.8 mmHg before treatment). The study compared the effect of a bolus indomethacin (30 mg intravenously) with that of induced hypocapnia. Indomethacin was found to decrease ICP and CBF and increase $CavO_2$ significantly. It is remarked by the authors that: "Although ischaemia evaluated from CBF, $SvjO_2$, and LOI (lactate/oxygen index) were indicated in individual cases after both hyperventilation and indomethacin, it was not reflected in outcome." However, the increase in ICP before treatment was very modest, and "patients in poor clinical condition indicating transtentorial herniation were excluded." There is no indication in the study that a bolus injection of indomethacin had any beneficial effect on outcome.

In their letter, Cold *et al.* claim that, during continuous intravenous indomethacin treatment, "the ICP-reducing effect is sustained and the increase in $AVDO_2$, the jugular venous lactate and EEG are transitory" and that they "find a low mortality of 10% in indomethacin treated severe head injury." We have been unable to find the data supporting these statements. In the report by Jensen *et al.*[4] indomethacin was administered intravenously throughout a 7-h period. During this period, the decrease in global CBF and the increase in $CavO_2$ remained. At 7 h after start of infusion, ICP seemed to have returned close to pretreatment level (28 ± 3 mmHg *vs.* 25 ± 11 mmHg). There is a lack of information regarding physiologic parameters in patients with severe head injuries when indomethacin infusion is continued beyond 7 h. Cold *et al.* also said that "results in an experimental model of a focal mass expanding lesion in pigs cannot rightly be transferred to the pathophysiological events of severe human head injury." Although this is obviously true, it seems to us that the clinical experiences by Biestro *et al.*[2] and Dahl *et al.*[3] are in good agreement with our experimental study: indomethacin reduces increased ICP, but at the expense of decreased CBF and impending cerebral ischemia. We agree with Cold *et al.* that indomethacin is contraindicated in patients suffering from cardiac ischemia, renal insufficiency, gastric bleedings, and coagulation disorders. Unfortunately, patients with severe head trauma may, quite often, experience multi-organ failure, which may limit the clinical use of indomethacin even further.

In our opinion, cerebral vasoconstriction is only one of the components, though important, in the treatment of posttraumatic brain swelling. Our treatment concept was published recently.[5] The concept aims at a reduction of ICP through decreased CBF and increased absorption of cerebral interstitial fluid. The latter is accomplished through reduction of intracapillary pressure by a combination of moderate precapillary vasoconstriction (DHE) and reduction of mean arterial blood pressure. We are currently reviewing our clinical results since 1989 that show that mortality in patients with closed head injuries has been almost eliminated, with no increase in the number of patients surviving in a vegetative state (Eker *et al.*, in preparation).

Although Cold *et al.* and our group seem to disagree partly regarding the clinical use of indomethacin, we share the view that pharmacologic vasoconstriction shows promise for the treatment of increased ICP. Probably, the optimal drug should combine a pronounced constriction of cerebral venous capacitance vessels and a moderate constriction of cerebral precapillary resistance vessels with no or insignificant effects in other vascular territories.

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Ultrasonography as an Aid to Internal Jugular Vein Cannulation: I

To the Editor:—Troianos *et al.*[1] used real-time ultrasonography with the Site Rite device (Dymax, Pittsburgh, PA) to clarify the relation between internal jugular vein and carotid artery in adults.[1]

In this study, Polaroid photographs of the relevant images displayed on the screen of the Site Rite were taken with a modified camera, but 11.2% (127/1136) were subsequently rejected, partly because of poor image quality. There is an alternative method of data collection when using this device. It is simple, effective and inexpensive.

Transparent acetate sheets used with overhead projectors are cut into 6 cm × 4.5 cm rectangles. One of these is then temporarily applied to the screen of the Site Rite device. The dimensions of the rectangle match that of the screen, and the housing of the screen holds it in place. Tilting of the device will add to stability, but adhesive is not needed. The anatomic structure under study is then surveyed by the investigator, and the relevant image is displayed on the screen. At this time, a co-investigator records relevant details from the screen image by drawing around salient images, recording them on the acetate rectangle with fine-tipped marker pens, which may be color-coded for clarity. The acetate is removed for study, and a new one can then be applied. The shape, size, and relation of vessels are, thereby, easily recorded.

I concede that photography may be viewed as a "gold standard," but I believe the method I describe is an acceptable alternative. Problems of photographic lighting, costs of film, camera, and modifications are avoided, and accuracy is not sacrificed.

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