changes in all parameters were similar with or without epidural analgesia following pancreatoduodenectomy, "not only neural input from the surgical wound but also perioperative stimulation of cytokine production might be responsible for the development of the stress response."

Although the study otherwise is very important, the applied method in my opinion does not allow this conclusion. First, the epidural block is not well described (dose of mepivacaine?), and it is only active intraoperatively, which hinders interpretation of late postoperative changes, since these may be released by early postoperative afferent input and pain after disappearence of the neural blockade. Second, and maybe more importantly, there is much evidence that a standard thoracic epidural analgesia does *not* provide a total afferent blockade,[2-4] which therefore is the most plausible explanation to the lack of a pronounced inhibitory effect of thoracic epidural analgesia on the surgical stress response in abdominal surgery. Thus, since the authors have not documented sufficient afferent neural blockade, it is impossible to make conclusions as to the relevance and quantitative role of other potential mediators, such as cytokines, as suggested in their study.

Finally, the pronounced inhibition of the stress response after hip surgery by epidural analgesia is well known,[5] and the differences in cytokine responses and endotoxin between the two procedures may be explained by the pronounced intestinal manipulation and subsequent endotoxin release during pancreatoduodenectomy, compared with the clean soft tissue injury in hip replacement.

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In Reply:—Walker and Herschman are concerned that perioperative use of antibiotics or certain other drugs may have affected the stress response of plasma adrenocorticotropic hormone (ACTH) and cortisol during pancreatoduodenectomy. Kehlet points out that a standard thoracic epidural analgesia does not provide a total afferent block during pancreatoduodenectomy and that this may explain the lack of a pronounced inhibitory effect of thoracic epidural analgesia on the stress response of these hormones during surgery. What we did observe, while monitoring plasma ACTH and cortisol levels, was a summated effect of various stimuli, such as pain, unblocked afferent neural input, and cytokines, on the hypothalamo-pituitary-adrenal (HPA) axis. Our observation obtained from animal experiments[1-3] as well as a vast majority of clinical experiences[4] indicated that each of these can induce maximal or near maximal stimulation of ACTH and cortisol secretion. Therefore, as far as this study design is employed, it is quite difficult to analyze which part of the hormonal response should be attributed to a specific stimulus. All we can conclude, therefore, is that "not only neural input . . . but also perioperative stimulation of cytokine production might be responsible for the development of the stress response . . . during and after upper abdominal surgery."

A second point is that Walker and Herschman claim it might be that the postoperative use of analgesics or opioids are responsible for reduction in stress response. Similarly, Kehlet claims that postoperative pain is important in inducing long-lasting hormonal changes after pancreatoduodenectomy. If so, what mechanism is responsible for maintaining the significantly elevated cortisol levels for more than 72 h with no elevation of plasma ACTH levels?[5-7] Pain and other neural inputs stimulate cortisol secretion through increased release of the hypothalamic corticotropic-releasing hormone and the resultant elevation of plasma ACTH levels. On the other hand, what we observed during this period was prolonged elevation of plasma cortisol levels with paradoxically suppressed ACTH level. As we have demonstrated elsewhere,[6] the mechanisms responsible for postoperative stress response are not continuously activated by pain, but an adaptation mechanism at the peripheral level increases the responsiveness of the adrenal gland, in turn increasing cortisol secretion. Therefore, in the present paper, we did not cite in detail any data regarding analgesic use postoperatively.

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A Newly Designed Curved Needle for Percutaneous Cannulation of the Internal Jugular Vein

To the Editor:—Among the reported sites for cannulation of internal jugular vein (IJV) is the notch of the clavicle. Initially described by Rao *et al.*[1] and confirmed by Oshima *et al.*,[2] this technique requires considerable skill and experience. One of the most difficult aspects of the procedure is that the posterior wall of the IJV should not be pierced, thereby avoiding complications such as pneumothorax and arterial puncture. Although the conventional straight needle has a sharply angled tip, it usually pierces both walls of the IJV.[3] To penetrate only the anterior wall of the IJV, we present a new approach for IJV cannulation using a newly designed curved needle (fig. 1).

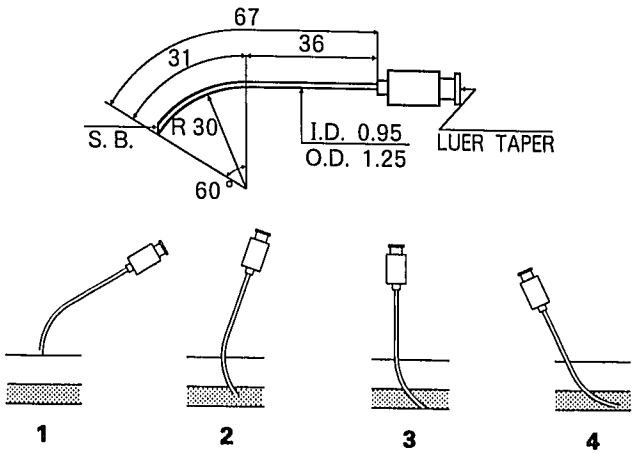

Fig. 1. Schematic diagram of the curved needle and schematic presentation of the internal jugular vein cannulation with the needle.

The needle was designed based on the anatomic profiles of the location of the IJV obtained from computed tomography and magnetic resonance studies. We confirmed that the IJV is located beneath and 1–1.5 cm lateral to the sternal end of the clavicle and parallel to the skin surface of the neck. The distance from skin surface to the axis of the IJV was 22.4 ± 5.1 mm (mean ± SD, n = 36).

The needle is advanced along a curving path to pierce the IJV at a narrow angle with little compression on the vessel wall. Furthermore, the bevel of the needle was shortened to prevent penetration of the posterior wall of the IJV (fig. 1).

With institutional approval and written informed consent from the patients, our new approach was employed for placement of the central venous catheter in 130 (81 males, 49 females) consecutive anesthetized patients ranging in age from 16 to 85 yr. Venipuncture was attempted using the right IJV in all patients. The operator stood at the right side of the patient. Correct identification of the notch of the clavicle was essential. A 23-G probing needle attached to a 2.5-ml syringe was inserted perpendicularly to the skin surface of the neck. The insertion site was about 0.5 cm cephalad from the notch. With the orientation of the probing needle as a guide, the curved needle was introduced. The needle was always advanced perpendicularly to the skin surface of the neck and parallel to the sagittal plane and directed to the thoracic inlet until the IJV was entered. As soon as blood was aspirated, the needle was tilted cephalad. A central venous catheter was then inserted with the aid of a spring guide wire.

The IJV was entered with the probing needle at the first attempt in 129 patients (129 of 130) within 3 cm, usually 1.5–2.0 cm, from the skin surface. In one of the patients, the probing needle did not locate the IJV, so that cannulation with the curved needle was not attempted. The needle was inserted into the IJV in 112 patients (112 of 129) on the first attempt, and on the second attempt in 16 patients.