We were consulted as to the efficacy and safety of epidural blood patch in this patient. Our primary concern was the injection of HIV-infected blood into the epidural space of a patient who had no evidence of HIV involvement of the central nervous system. Our colleagues' opinions regarding this issue were solicited, and our concerns were shared with the patient. We discussed our lack of experience with this situation and informed the patient of the dearth of experience in the published literature; nevertheless, the patient agreed to blood patch therapy. An epidural blood patch was performed using a 17-G Hustead needle and 15 ml autologous blood. The patient had immediate relief of his PDPH symptoms and was discharged pain-free from the recovery room. He was transferred to domiciliary care 3 days after the procedure. Intermittent follow-up over the subsequent 19 months has revealed no signs of neurologic involvement as a result of the blood patch. The patient has been discharged from domiciliary care and is functioning well in an independent environment.

We present this case to share our single experience with long-term follow-up on epidural blood patch in an HIV-positive patient and solicit additional advice from other practitioners who have dealt with this dilemma.

BRIAN K. BEVACQUA, M.D.
Chairman, Anesthesia Section
Assistant Professor, Clinical Anesthesia
VA Medical Center
10701 East Boulevard
Cleveland, Ohio 44106

ALEXEY V. SLUCKY, M.D.
Resident, Anesthesiology
Case Western Reserve University
2074 Abington Road
Cleveland, Ohio 44106

REFERENCES

1. Frame WA, Lichtmann MW: Blood patch in the HIV-positive patient. ANESTHESIOLOGY 73:1297, 1990

(Accepted for publication January 25, 1991.)

Anesthesiology
74:953, 1991

Post-dural Puncture Headache in the HIV-positive Patient

To the Editor:—The recent report by Frame and Lichtmann[1] about blood patch in the human immunodeficiency virus (HIV)-positive patient raises an interesting question—can injecting HIV-positive blood into the epidural space increase the risk of HIV infection in the central nervous system? The authors invite others to share their experiences with this problem. A 25-yr-old man, HIV-positive, was admitted for evaluation of shortness of breath and fever. As part of the diagnostic evaluation, a lumbar puncture was performed. During the next 2 days, the patient had a persistent positional headache believed to be related to the dural puncture. On the second day the patient was discharged receiving oral analgesics. The patient's headache increased in severity after discharge, and by the 6th day after the lumbar puncture the patient's internist asked me to perform an epidural blood patch because the headache was causing severe nausea and vomiting, as well as pain. It was apparent that the patient had not had a trial of conservative therapy; that is, although told to increase fluid intake he could not tolerate this. Therefore, the patient was admitted on the evening of the 6th day and received an intravenous infusion of 0.45% saline at 100 ml/h. On the evening of the 8th day the patient rated his headache as very mild (1 10), whereas on days 4 and 5 it had been very severe (10 10). His nausea and vomiting had resolved. Since admission he had been able to increase his oral intake. By the morning of the 9th day, his headache had completely resolved and he was discharged.

Most spinal headaches will spontaneously resolve, and until we have more information about the infectiousness of the HIV virus in the central nervous system, I believe that blood patch should be used only when conservative measures truly have failed. This, of course, means that if the patient is not able to tolerate oral hydration, intravenous hydration will be necessary. As in this case, conservative measures often are said to have failed, when in reality, because of nausea and vomiting, hydration has not really been given a chance. Had this patient not responded to intravenous hydration, intravenous caffeine therapy would have been tried next.[2] Does infusion of epidural saline have an advantage over epidural blood patch in this particular situation? If a blood patch is necessary, should it be performed with fresh HIV-negative blood from an appropriate donor? These and other issues need to be considered.

JAMES J. GIBBONS, M.D.
Assistant Professor of Anesthesiology
St. Louis University School of Medicine
3635 Vista Avenue
St. Louis, Missouri 63110-0250

REFERENCES

1. Frame WA, Lichtmann MW: Blood Patch in the HIV-Positive Patient. ANESTHESIOLOGY 73:1297, 1990
2. Jarvis AP, Greenawalt JW, Fagraeus L: Intravenous Caffeine for Postdural Puncture Headache. Anesth Analg 65:316–317, 1986

(Accepted for publication January 25, 1991.)

Anesthesiology
74:953–954, 1991

Does Arterial Baroreflex Play a Role in Response to Acute Hypovolemia during Induced Hypotension?

To the Editor:—I read with great interest the study by Taneyama *et al.*[1] on the arterial baroreceptor reflex response to acute hypovolemia during induced hypotension. There are several questions that need to be addressed. To begin with, there is a question about the arterial

baroreceptor reflex. Rapid blood loss was used to assess the gain of the arterial baroreflex. However, both arterial pressure and cardiac filling pressure decrease with rapid hypovolemia and unload the arterial baroreceptors as well as cardiopulmonary baroreceptors with vagal afferents.[2] Decreases in the activities of both of these groups of receptors augment sympathetic outflow to the circulation. Since vagal afferents were intact in the study by Taneyama *et al.*,[1] they should consider the contribution of the cardiopulmonary baroreflex to the total response.

Secondly, the results clearly show that the slope of baroreflex is steeper when mean arterial pressure (MAP) is reduced by sodium nitroprusside (SNP) compared with prostaglandin E_1 (PGE_1), and the reflex response curve is saturated at approximately 75 mmHg by SNP and at about 65 mmHg by PGE_1 (figs. 1 and 2 of their article), suggesting that the baroreflex gain is depressed during PGE_1-induced hypotension more than during SNP. The authors chose a MAP of 71–74 mmHg for induced hypotension, and they further decreased MAP by rapid blood loss. Since the baseline level of induced hypotension was close to the saturation levels of two baroreflex curves, the baroreflex gain became highly dependent on the pressure where the change in MAP started. Therefore, stating that "(arterial) baroreflex response to acute hypovolemia was better preserved during PGE_1-induced hypotension than with SNP-induced hypotension" is only appropriate when MAP was maintained at some particular level during induced hypotension. If they had maintained MAP at 85 mmHg or more, the baroreflex gain could have been preserved better during SNP-induced hypotension than during PGE_1-induced hypotension. In contrast, if they had chosen 60 mmHg, the result might not have been different between SNP and PGE_1.

Moreover, the same amount of blood loss produced decreases in MAP by 19, 21, and 21 mmHg during SNP-, PGE_1-, and trimethaphan-induced hypotension, respectively. That is, the same degree of further hypotension was elicited in response to acute blood loss. This finding indicates that no drug is superior to others in this respect. As the authors describe, the baroreceptor reflex is important to restore blood pressure in the event of acute blood loss. Moreover, a maximal change in blood pressure in response to acute blood loss is an outcome of reduced blood volume and baroreflex-induced compensation. Since trimethaphan abolished the baroreflex control of sympathetic nerve activity, the equivocal decrease in blood pressure by acute blood loss suggests that baroreflex does not play an important role in regulating blood pressure in such situation. Therefore, the authors' conclusions that "induced hypotension with PGE_1 provides a greater margin of safety than that following SNP when rapid bleeding occurs during surgery" and that "trimethaphan is inferior to PGE_1 and SNP in this respect" are questionable.

SUMIO HOKA, M.D.
Lecturer
Department of Anesthesiology
Faculty of Medicine, Kyushu University
3-1-1 Maidashi, Higashi-ku
Fukuoka 812, Japan

REFERENCES

1. Taneyama C, Goto H, Goto K, Benson KT, Unruh GK, Arakawa K: Attenuation of arterial baroreceptor reflex response to acute hypovolemia during induced hypotension. ANESTHESIOLOGY 73:433–440, 1990
2. Abboud FM, Thames MD: Interaction of cardiovascular reflexes in circulatory control, Handbook of Physiology: The Cardiovascular System. Volume 3. Edited by Shepherd JT, Abboud FM. Washington, DC, American Physiology Society, 1983, pp 675–753

(Accepted for publication January 25, 1991.)

Anesthesiology
74:954–955, 1991

In Reply:—We appreciate Dr. Hoka's comments on our article.[1] In our study, maximum reflex increases in heart rate (HR) and renal sympathetic nerve activity (RSNA) were recorded and compared during acute blood loss; 5 ml/kg over 10 s after a steady state of induced hypotension (mean arterial pressure [MAP] 71–74 mmHg) was established with sodium nitroprusside (SNP), prostaglandin E_1 (PGE_1), and trimethaphan. The article[2] Dr. Hoka cites states that "both arterial pressure and cardiac filling pressure increase with expansion of blood volume and activate the arterial baroreceptors as well as cardiopulmonary baroreceptors with vagal afferents." Therefore, as it states, the contribution of the cardiopulmonary baroreflex should be considered to the total response. However, volume expansion usually is performed slowly by infusing volume expander intravenously. It is apparent that cardiopulmonary baroreflex plays a significant role in the total baroreflex response in such a situation of gradually increasing central blood volume. This is also true when the hypovolemia is produced rather slowly by withdrawing blood from the venous site, and the central blood volume decreases gradually. In our experiment, however, hypovolemia was produced by removing blood rapidly from the arterial site, and we believe that in this situation the primary impact was on the arterial baroreflex. However, reduction of the central blood volume would occur and cardiac pulmonary baroreceptors would be activated eventually. We agree, therefore, with Dr. Hoka that the contribution of cardiopulmonary baroreflex to total response should have been discussed in our article, although it might not have been significant, particularly, as contributing to the maximum gains of HR and RSNA.

The contribution of cardiopulmonary baroreflex to total baroreflex response seems to exist inevitably in the study of arterial baroreflex unless bilateral vagotomy is performed. Phenylephrine and nitro-glycerin have been used for a pressor and a depressor test, respectively, to assess arterial baroreflex sensitivity. However, cardiopulmonary baroreceptors can be affected by increasing or decreasing the central blood volume secondary to peripheral vasoconstriction or vasodilation.[3] How much the cardiopulmonary baroreflex contributes to total baroreflex response in different techniques of pressor tests or depressor tests (blood loss can be considered as one of the depressor tests) has not been studied thoroughly and remains to be elucidated.

We agree with Dr. Hoka, and it is obvious that the degree of the baroreflex gain depends on the pressure where the reduction of MAP starts on the sigmoid shape of the baroreflex response curve. It is therefore true that our statement "arterial baroreflex response to acute hypovolemia was better maintained during induced hypotension with PGE_1 than with SNP" is appropriate only when MAP is maintained at some particular level during hypotension. In our experimental model using mongrel dogs, the particular level of induced hypotension happened to be about 71–74 mmHg MAP, where baroreflex gain is already saturated with SNP but not saturated with PGE_1. Such a particular level of induced hypotension with SNP and PGE_1 is expected to change,