

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.<sup>2</sup> 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.*,<sup>1</sup> 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<sub>1</sub> (PGE<sub>1</sub>), and the reflex response curve is saturated at approximately 75 mmHg by SNP and at about 65 mmHg by PGE<sub>1</sub> (figs. 1 and 2 of their article), suggesting that the baroreflex gain is depressed during PGE<sub>1</sub>-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<sub>1</sub>-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<sub>1</sub>-induced hypotension. In contrast, if they had chosen 60 mmHg, the result might not have been different between SNP and PGE<sub>1</sub>.

Moreover, the same amount of blood loss produced decreases in MAP by 19, 21, and 21 mmHg during SNP-, PGE<sub>1</sub>-, 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<sub>1</sub> provides a greater margin of safety than that following SNP when rapid bleeding occurs during surgery” and that “trimethaphan is inferior to PGE<sub>1</sub> 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

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(Accepted for publication January 25, 1991.)

Anesthesiology  
74:954–955, 1991

*In Reply.*—We appreciate Dr. Hoka's comments on our article.<sup>1</sup> 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<sub>1</sub> (PGE<sub>1</sub>), and trimethaphan. The article<sup>2</sup> 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 dis-

cussed 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 nitroglycerin 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.<sup>3</sup> 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<sub>1</sub> 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<sub>1</sub>. Such a particular level of induced hypotension with SNP and PGE<sub>1</sub> is expected to change,

depending on many variables, such as anesthesia techniques, baroreflex sensitivity, or underlying cardiovascular pathology. Therefore, as with most findings from laboratory studies, our findings should not be extrapolated simply to the clinical situation.

The statement by Dr. Hoka that "a maximal change in blood pressure in response to acute blood loss is an outcome of reduced blood volume and baroreflex-induced compensation" may not be true unless full baroreflex compensations (increases in peripheral vascular resistance, heart rate, and cardiac contractility) are achieved at the conclusion of acute blood loss. Our data show that the same amount of blood loss (5 ml/kg) over the same period (10 s) produced almost the same reduction of MAP regardless of whether dogs were in a normotensive state or under induced hypotension before acute blood loss, and regardless of which hypotensive agent was being used before acute blood loss. This indicates that maximum reduction of MAP secondary to acute arterial blood loss may not have been affected by the arterial baroreflex in our experimental procedures. This is because acute arterial blood loss was the initial event and presumably because baroreflex compensation had not been operating fully enough to modulate the impact of acute blood loss on the degree of maximum reduction of MAP. As a matter of fact, maximum reflex increases of HR and RSNA occurred after MAP had reached the lowest level following acute blood loss. We found that the maximum increases in HR and RSNA were greater with PGE<sub>1</sub> than with SNP and therefore stated that "baroreflex response to rapid blood loss was significantly greater during PGE<sub>1</sub> than with SNP-induced hypotension . . . despite the same baseline-induced hypotension . . . and the same degree of further hypotension from rapid blood loss." Although we only measured and evaluated maximum gains in HR and RSNA, a restoration of MAP may have been faster, cardiac output values may have been higher, and the renin-angiotensin system may have been activated more after reaching maximum reduction of MAP due to acute blood loss during PGE<sub>1</sub>-induced hypotension, as compared to SNP-induced hypotension.

CHIKUNI TANEYAMA, M.D.  
*Lecturer*

Anesthesiology  
74:955-956, 1991

## Myofascial Pain Syndrome Can Cause Right Upper Quadrant Pain

*To the Editor:*—Haynsworth and Noe present an interesting case of right upper quadrant pain.<sup>1</sup> In their discussion of the causes of abdominal pain, "trigger points" are mentioned but myofascial pain syndrome (MFPS) is not. Trigger points associated with a MFPS are an important and often overlooked cause of somatic abdominal pain.<sup>2</sup>

Muscles having a referral pattern that includes the abdomen are the rectus abdominus, serratus anterior, external oblique, iliocostalis thoracis, erector spinae, and possibly the intercostal muscles.<sup>2-5</sup> The interspinous ligament, costal cartilage and lower intercostal joints may also refer pain to the upper abdomen.<sup>2,3</sup> Most of these structures are not located within the anterior abdominal area and therefore are not usually palpated during examination of the abdomen.

Visceral complaints as seen in their patient also have been noted as part of a MFPS.<sup>3-5</sup> Perhaps the reproduction of this patient's pain at the T11 area represented a trigger point in the serratus anterior or intercostal muscle. This undiagnosed MFPS may also respond to rhizotomy.

We believe that for completeness, the possibility of a MFPS and its appropriate treatment (TENS trial, trigger point injections, spray and stretch, and physical therapy) should be addressed before the diagnosis of intercostal neuralgia and the relatively destructive treatment of rhizotomy is suggested.

MARK E. ROMANOFF  
*Major, USAF, MC*  
*Director, Pain Management Clinic*

JAY S. ELLIS, JR.  
*Major, USAF, MC*  
*Director, Clinical Anesthesia Services*  
*Department of Anesthesiology*  
*Wilford Hall Medical Center*  
*Lackland Air Force Base, Texas 78236*

*Department of Anesthesiology*  
*Shinshu University School of Medicine*  
*3-1-1 Asahi, Matsumoto 390, Japan*  
*(Previously Research Fellow in Anesthesiology)*  
*Department of Anesthesiology*  
*University of Kansas Medical Center*  
*Kansas City, Kansas 66103*

HIROSHI GOTO, M.D.  
*Professor*  
*Department of Anesthesiology*  
*University of Kansas Medical Center*  
*Kansas City, Kansas 66103*

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*(Accepted for publication January 25, 1991.)*