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\textsubscript{1} provides a greater margin of safety than that following SNP when rapid bleeding occurs during surgery" and that "trimethaphan is inferior to PGE\textsubscript{1} and SNP in this respect" are questionable.

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(Accepted for publication January 25, 1991.)

In Reply.—We appreciate Dr. Hoka’s comments on our article.\textsuperscript{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\textsubscript{1} (PGE\textsubscript{1}), and trimethaphan. The article\textsuperscript{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.\textsuperscript{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\textsubscript{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\textsubscript{1}. Such a particular level of induced hypotension with SNP and PGE\textsubscript{1} is expected to change.
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(Accepted for publication January 25, 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.1 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.2

Muscles having a referral pattern that includes the abdomen are the rectus abdominis, serratus anterior, external oblique, iliocostalis thoracis, erector spinae, and possibly the intercostal muscles.3,4 The interspinous ligament, costal cartilage and lower intercostal joints may also refer pain to the upper abdomen.5,6 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.1,4 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.

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Anesthesiology
74:955-956, 1991