# Effect on atrial natriuretic peptide by bracing in scoliosis

## Boston brace removal studied in 10 cases

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The immediate effect of Boston brace removal on plasma levels of atrial natriuretic peptide and arginine vasopressin was studied in 10 patients with idiopathic scoliosis ranging in age from 12 to 16 years. Blood samples were drawn via the antecubital vein in the supine position with the brace on and 10 min after brace removal. ANP and AVP plasma levels were measured by radioimmunoassay. Although no differences were found in the plasma concentrations of arginine vasopressin while on and after removal of the brace, atrial natriuretic peptide levels showed an approximately 80 percent increase 10 min after brace removal, presumably due to central volume redistribution. The changes of atrial natriuretic peptide plasma levels observed may be related to previously reported changes of the glomerular filtration rate and urinary sodium excretion following brace removal.

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Bracing in scoliosis exerts increased external pressure on thoracic and abdominal organs (Aaro et al. 1981). The pressure could affect respiratory as well as renal function (Aaro and Berg 1982, Kennedy et al. 1989). The application of the brace results in reduced urinary sodium excretion, whereas removal of the brace results in normalization of sodium excretion (Aaro and Berg 1982). This has been attributed to hemodynamic changes resulting from pressure exerted by the brace; but the role of atrial natriuretic peptide, which can modify urinary sodium excretion, has not been studied.

In this study, we investigated the immediate effect of Boston brace removal on plasma atrial natriuretic peptide (ANP) and arginine vasopressin (AVP) in patients with idiopathic scoliosis.

## Patients and methods

Ten patients with idiopathic scoliosis, 4 girls and 6 boys, treated with the Boston brace (TLSO) for a period ranging from 3 to 36 months, median 12 months, were included in the study. The median age was 14 (12–16) years.

The scoliotic curves were measured using the Cobb method on an anteroposterior radiograph of the erect spine before and after application of the brace. Six of the curves were convex to the right. Two were pure thoracic, two were lumbar, and the remaining six were thoracolumbar. The Cobb angle without the brace ranged from 19° to 37° (median 25°). The immediate angle correction by the Boston brace was 55° ± 12° (mean ± SD).

Patients with cardiac, renal, and hepatic failure or those that had received any drug treatment during the week prior to the study were excluded. There were no dietary restrictions before the study, but the patients were studied after an overnight fast. After informed consent from the parents and the patients, the ensuing protocol was followed.

The patient, who was already wearing the brace, was placed in the supine position for 10 min, and arterial blood pressure was measured with a standard sphygmomanometer. The diastolic reading was made at the level where the sounds disappear (Korotkoff's phase V). The heart rate was measured by cardiac auscultation for a whole minute. For plasma ANP and AVP determinations (with the brace), 5 mL of blood was drawn via the antecubital vein, and the brace was subsequently removed. The patient remained in the supine position for 10 more minutes, at which time a second measurement of blood pressure and heart rate was taken and a second blood sample was drawn for ANP and AVP plasma determination without the brace.

The blood was drawn into plastic tubes containing EDTA. The plasma was separated immediately by centrifugation and kept at –20 °C until extraction. Plasma ANP and AVP concentrations were measured by RIA. The methodology for the ANP and AVP assays has been described in detail elsewhere (Margioris et al. 1990, Meyorhoff et al. 1990). The sensitivities of the ANP and AVP assays were respectively 8 pg/mL and 0.2 pg/mL. Plasma levels in the range of 50 pg/mL for ANP and 1.0 pg/mL for AVP were considered normal. In one of the 10 patients, AVP plasma levels were not measured because of insufficient plasma quantity.

The mean arterial blood pressure (MAP) was calculated using the following equation :

MAP = diastolic pressure + [(systolic/diastolic pressures)/3].

### *Statistics*

The Student's paired *t*-test and the Pearson correlation coefficient were used for statistical analysis. The 5 percent level was considered significant. Summarized data are expressed as mean ± SD.

## Results

Although there was no difference in the mean arterial blood pressure with and without the brace (paired *t*-test, $P = 0.5$), the heart rate was decreased 10 min after brace removal from a value of 80 beats/min to a value of 75 beats/min (paired *t*-test $P = 0.0001$; Table 1).

Plasma AVP concentrations remained unchanged after brace removal (0.9 ± 0.5 pg/mL vs 0.9 ± 0.7 pg/mL). In contrast, plasma ANP concentrations showed an 83 percent increase 10 min after brace removal: viz., from 46 pg/mL to 83 pg/mL (paired *t*-test $P = 0.002$; Figures 1 and 2).

No correlation was observed between the changes of ANP and AVP, heart rate, mean arterial blood pressure, or Cobb's angle.

**Table 1. Mean arterial blood pressure (MAP), heart rate, arginine vasopressin (AVP), and atrial natriuretic peptide (ANP) plasma levels with and without the Boston brace. Mean *SD***

| Variable | n | With Brace | | Without Brace | | *P*-value |
|---|---|---|---|---|---|---|
| MAP (mm Hg) | 10 | 88 | *10.5* | 90 | *9.4* | NS |
| Heart rate (b/min) | 10 | 80 | *8.2* | 75 | *8.1* | 0.0001 |
| AVP (pg/mL) | 9 | 0.9 | *0.5* | 0.9 | *0.7* | NS |
| ANP (pg/mL) | 10 | 46 | *26.8* | 83 | *44* | 0.002 |

## Discussion

Our study clearly shows that in patients with idiopathic scoliosis atrial plasma natriuretic peptide levels increase 10 min after Boston brace removal. This change must reflect changes in circulation causing atrial distension, because the latter is known to increase plasma concentrations of alpha atrial natriuretic peptide in the circulation (DeBold et al 1981, Epstein et al. 1989).

The plasma half-life of human alpha-atrial natriuretic peptide is 2–3 min (Yandle et al. 1986), and the elevation we observed 10 min after removal of the brace is compatible with recent or concurrent secretion of atrial natriuretic peptide.

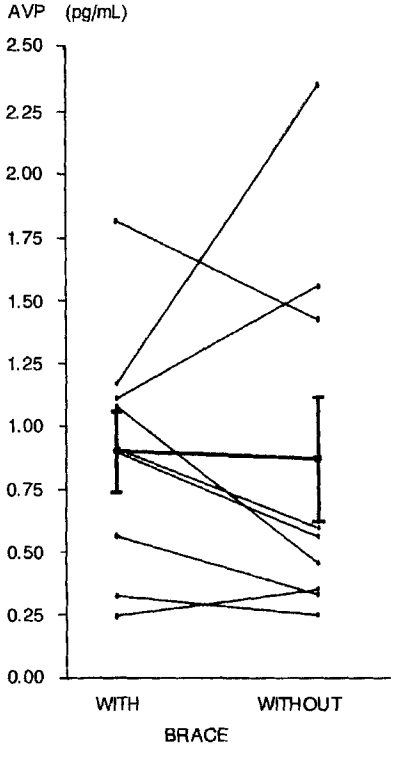


Figure 1. Plasma arginine vasopressin levels (AVP) in each patient with and without the brace ($P = 0.999$).

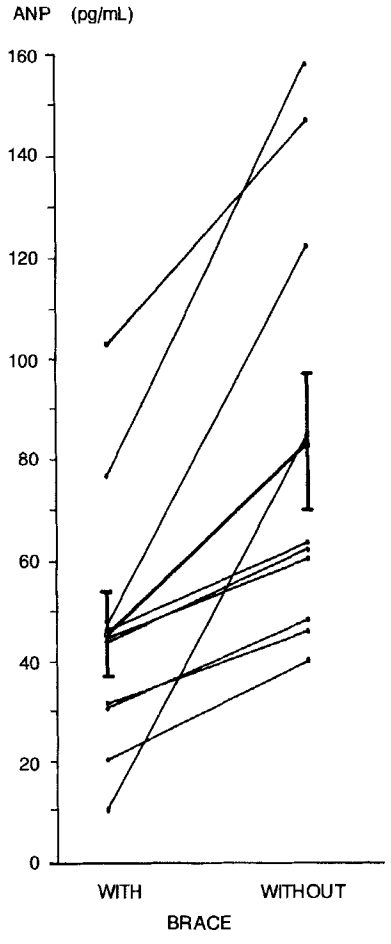


Figure 2. Plasma atrial natriuretic peptide levels (ANP) in each patient with and without the brace ($P = 0.0023$).

Plasma immunoreactive atrial natriuretic peptide rises in response to acute sodium volume loading, the central shift of volume produced by lying down or by immersion, rapid increases in blood pressure, dynamic exercise, and the administration of glucocorticoids or mineralocorticoids (Weidmann et al. 1989). Similarly, atrial natriuretic peptide plasma levels are elevated in conditions such as cardiac renal or hepatic failure (Keyon and Jardine 1989, Weidmann et al. 1989). The latter is not pertinent to our patients, who have no evidence of such failure.

The atrial natriuretic peptide plasma levels with the brace on as measured in the supine position were found to be within the adult "normal" range (46 ± 27 pg/mL). However, the fact that atrial natriuretic peptide plasma levels rise with age (Tan et al. 1989) should be kept in mind, as our subjects were all young. Ten minutes after brace removal, atrial natriuretic peptide plasma levels—in the absence of any known stimulus for atrial natriuretic peptide secretion—showed an increase corresponding to approximately 80 percent of the mean initial value with the brace. This increase of atrial natriuretic peptide could be attributed to atrial distension secondary to a central volume shift caused by brace removal. Brace wear has been associated with a reduction in lung volume and urinary sodium excretion (Aaro and Berg 1982, Kennedy et al. 1989). These changes, which are due to the compressive effect exerted by the brace, are functional and thus reversible by brace removal. The reduction of urinary sodium excretion during brace treatment is accompanied by a reduction in glomerular filtration rate (GFR), although it remained within the normal range. This reduction was claimed to be the result of hemodynamic changes occurring at the level of the inferior vena cava or the renal veins (Aaro and Berg 1982). Theoretically, a partial compression of the inferior vena cava during bracing can explain the volume shift and the subsequent increase in atrial natriuretic peptide plasma levels observed after brace removal. The simultaneous decreased heart rate observed supports the above theory, although "stress" elimination by the end of the study period cannot be excluded as a causative factor.

The arginine vasopressin plasma levels were found to be within the normal range during bracing, as well as 10 min after brace removal. Although there is evidence of physiologic antagonism between atrial natriuretic peptide and arginine vasopressin (Keyon and Jardine 1989), no correlation was found between changes in the concentrations of these hormones ($r = 0.22$, $P = 0.63$). Moreover, the normal values of plasma arginine vasopressin concentrations found in these patients with and without the brace preclude the possibility of significantly reduced blood volume during bracing.

Over the last few years, it has become apparent that atrial natriuretic peptide has natriuretic, diuretic, and vasodilatory properties (Keyon and Jardine 1989). On the other hand, brace removal in patients with idiopathic scoliosis has been found to restore the reduction of glomerular filtration rate and urinary sodium excretion induced by bracing (Aaro and Berg 1982). Although these data suggest a causative role for atrial natriuretic peptide, more studies are needed to clarify whether the increase in plasma atrial natriuretic peptide after brace removal is a crucial factor for the restoration of glomerular filtration rate and urinary sodium excretion.

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