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INTRODUCTION. Aortic cross-clamping may, although blood pressure is controlled by vasodilatators, lead to an increase in atrial stress by an increased in the loading conditions of the heart. Atrial natriuretic peptide (NAP), which is stored in cardiac atria may be secreted in response to atrial distension if induced by aortic cross-clamping. Since ANP has been shown to have natriuretic and marked vasorelaxant effects it may play a role in the regulation of circulatory volume during aortic surgery.

The purpose of the present study therefore was to determine wether ANF plasma levels are increased in response to either infrarenal or thoracic aortic cross-clamping in patients undergoing aortic surgery.

## METHODS.

<u>Patients</u>. Twenty five patients (mean  $62 \pm 15$ ) undergoing vascular surgery were included in this study. All gave informed consent after approval by our Ethics Committee. Patients were divided into 3 groups according to the surgical procedure: group A (n = 10) patients undergoing reconstruction of the abdominal aorta requiring an infrarenal aortic cross-clamping, group T (n = 5) patients undergoing surgical resection of thoracic aortic aneurysm without shunting technique, group C (n = 10) patients undergoing carotid endarterectomy (control group).

Anesthetic management. All patients had preoperative insertion of radial artery catheter. Patients scheduled for aortic surgery of Swan-Ganz catheter was established.

Anesthetia was induced with flunitrazepam 0.02 mg.kg-l, fentanyl 5 mcg.kg-l and pancuronium 0.1 mg.Kg-l. After tracheal intubation, anesthesia was maintained with N2O 50 % in O2, increments of fentanyl and isoflurane. Intraoperatively systolic blood pressure was maintained by adjusting the inspired concentration of isoflurane. Hemodynamic measurements and ANF plasma levels, determined by radioimmuno assay on blood drawn via the radial artery catheter, were obtained at the following times:

- TO: before anesthesic induction
- Tl : during the surgical procedure before the aorta or the carotid artery was clamped.
- T2 and T3 : respectively 2 min and 30 minutes after clamping.

All data were expressed as mean  $\pm$  SEM. Two way analysis of variance was used for statistical analysis.

RESULTS. ANF plasma levels and main hemodynamic parameters are reported in the table. At clamping SBP remained towards preinduction values in all patients studied, by increasing the inspired concentration of isoflurane (Table 1).

No change in ANF plasma levels was noted in patients at the clamping of the carotid artery and at infrarenal aortic cross-clamping. By contrast ANF plasma levels increased significantly in response of thoracic aortic clamping. After thoracic aortic clamping (T2 and T3) ANF plasma levels are significantly higher in group T than in groups C and A.

## DISCUSSION.

The ANF plasma levels were highly and significantly increased only in patients who had a thoracic aortic cross-clamping. Such an increase was observed while systolic blood pressure was controlled and although RAP did not increase. These data suggest that although SBP and RAP are controlled, thoracic aortic cross-clamping increases atrial wall stress. Moreover, the effects of ANF which decreases both afterload and circulatory volume could play a role in the cardiovascular changes induced by thoracic aortic cross-clamping.

|                |             | CONTROL  | ANESTHESIA  | XC+2min  | XC+30min   |
|----------------|-------------|--|---|--|--|
| ANF<br>pg.ml-1 | C<br>A<br>T | 89 <u>+</u> 17<br>81 <u>+</u> 16<br>85 <u>+</u> 35 | 75 <u>+</u> 14<br>82 <u>+</u> 16<br>87 <u>+</u> 29  | 76 <u>+</u> 14<br>91 <u>+</u> 12<br>187 <u>+</u> 70 <b>*</b> | 68 <u>+</u> 14<br>80 <u>+</u> 13<br>∆318 <u>+</u> 116 <b>*</b> ∆ |
| HR<br>b.min-l  | C<br>A<br>T | 61 <u>+</u> 7<br>70 <u>+</u> 4<br>65 <u>+</u> 3    | 65 <u>+</u> 6<br>71 <u>+</u> 3<br>73 <u>+</u> 7     | 65 <u>+</u> 5<br>72 <u>+</u> 3<br>86 <u>+</u> 4              | 62 <u>+</u> 5<br>68 <u>+</u> 3<br>87 <u>+</u> 8                  |
| SBP<br>mmHg    | C<br>A<br>T | 147 <u>+</u> 8<br>141 <u>+</u> 6<br>135 <u>+</u> 4 | 121 <u>+</u> 6<br>122 <u>+</u> 6<br>128 <u>+</u> 10 | 130 <u>+</u> 6<br>101 <u>+</u> 14<br>138 <u>+</u> 8          | 121 <u>+</u> 5<br>103 <u>+</u> 3<br>111 <u>+</u> 13              |
| RAP<br>mmHg    | C<br>A<br>T | 9 <u>+</u> 2<br>7 <u>+</u> 3                       | 10 <u>+</u> 1<br>19 <u>+</u> 2                      | 10 <u>+</u> 1<br>20 <u>+</u> 2*                              | 8 <u>+</u> 2<br>21 <u>+</u> 3 <b>*</b>                           |
| CO<br>l.min-l  | C<br>A<br>T | 6.1 <u>+</u> 0.3<br>4.4 <u>+</u> 0.4               | 5.7 <u>+</u> 0.4<br>5.3 <u>+</u> 0.8                | 5.3 <u>+</u> 0.4<br>4.7 <u>+</u> 1                           | 5.8 <u>+</u> 0.5<br>7 <u>+</u> 1 <b>*</b>                        |

Table 1  $\bigstar$  p < 0.01 vs control ;  $\Delta$  p < 0.01 vs gr. C  $\bullet$  p < 0.01 vs gr. A

## REFERENCE

1. De WARDENER HE, CLARKSON EM. : Concept of natriuretic hormone. Physiol. Rev. 65 : 658-759, 1985