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Corrigendum

Corrigendum to 'The role of endothelin and nitric oxide in modulation of normal and spastic cerebral vascular tone in the dog' [Eur. J. Pharmacol. 277 (1995) 77–87] ☆

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Abstract

To investigate the roles of endothelin and nitric oxide (NO) in the regulation of cerebral vascular tone under basal conditions and in cerebral vasospasm following subarachnoid hemorrhage in dogs, we used BQ-123 (cyclo(-D-Trp-D-Asp-L-Pro-D-Val-L-Leu-) sodium salt), an endothelin ET_A receptor antagonist, L-arginine, a substrate for the formation of NO, and N^G-nitro-L-arginine methyl ester, an NO synthesis inhibitor, and measured the angiographic diameter of the basilar artery in vivo. In normal dogs, intracisternal (i.c.) injection of BQ-123 (0.6 mg/kg) produced a $29.4 \pm 6.11\%$ ($P < 0.01$) increase in the basal diameter 24 h after injection. N^G-nitro-L-arginine methyl ester (0.6 mg/kg i.c.) produced a $19.3 \pm 2.93\%$ ($P < 0.05$) decrease in the basal diameter 2 h after injection. This decrease was significantly attenuated by both BQ-123 (0.06–0.6 mg/kg i.c.) and L-arginine (6 mg/kg i.c.), but not by D-arginine. In the two-hemorrhage canine model, BQ-123 significantly inhibited the development of cerebral vasospasm ($36.9 \pm 4.11\%$ decrease on day 5 and $42.0 \pm 4.54\%$ decrease on day 6 in controls vs $21.7 \pm 4.75\%$ decrease ($P < 0.05$) on day 5 and $20.8 \pm 4.14\%$ decrease ($P < 0.05$) on day 6 for 0.6 mg/kg i.c.). Furthermore, in this model, L-arginine (6 mg/kg i.c.) significantly attenuated the cerebral vasospasm on day 4 from a $30.9 \pm 5.78\%$ decrease (before) to a $12.6 \pm 5.99\%$ decrease (after). The immunoreactive endothelin-1 levels in the endothelial layer and the adventitia of the basilar artery were much higher on days 3 and 7 after the injection of autologous blood than on day 0 before blood injection. These results suggest that endogenous endothelin and NO both participate in regulating the basal tone of cerebral arteries, and, therefore, the development of cerebral vasospasm following subarachnoid hemorrhage may be at least partially attributed to an impairment of the balanced action of endothelin and NO. Furthermore, endothelin ET_A antagonists or NO products may be useful in the treatment of cerebral vasospasm following subarachnoid hemorrhage.

Keywords: BQ-123; Endothelin; Nitric oxide (NO); Cerebral vasospasm; Subarachnoid hemorrhage

In our above-mentioned paper, the top panel of Fig. 3 should read 'Decrease in diameter' instead of 'Increase in diameter'. On the next page please find the corrected figure.

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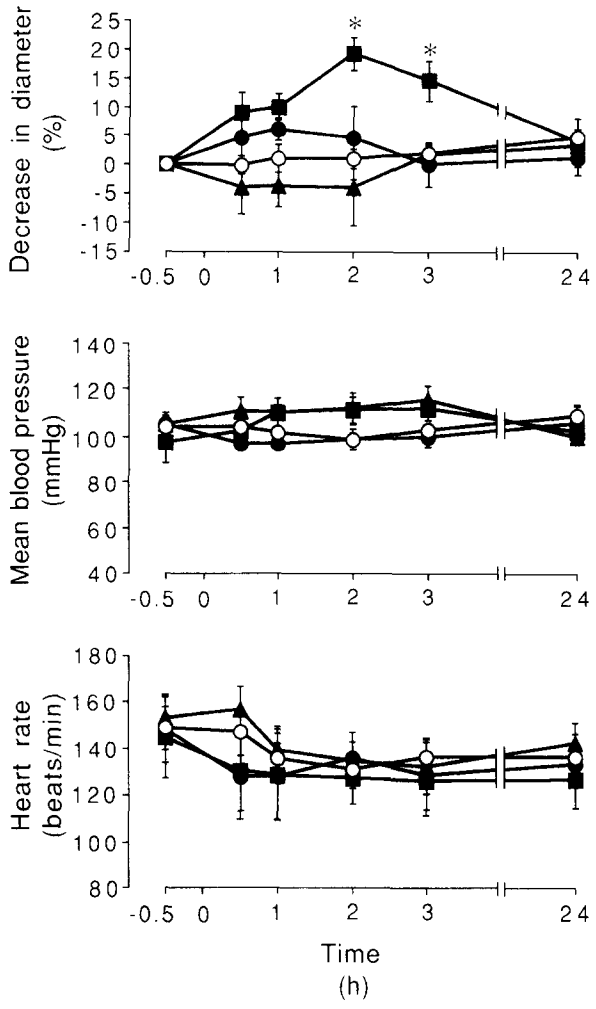

Fig. 3. Effect of intracisternal injection of saline (○), L-arginine (6 mg/kg (▲)) and N^G-nitro-L-arginine methyl ester (0.12 mg/kg (●) and 0.6 mg/kg (■)) on the diameter of the basilar artery (upper), mean blood pressure (middle) and heart rate (lower) in normal dogs. Decreases in the diameter are expressed as a percentage of the basal diameter measured 30 min before injection in each dog. The basal diameters were 1.09 ± 0.03 mm (control), 1.00 ± 0.08 mm (L-arginine), 1.10 ± 0.04 mm (N^G-nitro-L-arginine methyl ester, 0.12 mg/kg) and 1.07 ± 0.04 mm (N^G-nitro-L-arginine methyl ester, 0.6 mg/kg). $^*P < 0.05$ significantly different from the value in the control (saline-treated) group. The values are mean ± S.E. for five animals. The Newman-Keuls test was used.