mmHg (n = 13, $P < 0.05$) at 15 min. Thereafter, MAP returned to the pre-LPS value at 1 h (i.e., 108 ± 3 vs. 114 ± 3 mmHg, $P > 0.05$). At 2 h after LPS, there was a continued further fall in MAP to 84 ± 4 mmHg at 4 h (Fig. 1B). In the sham-operated (i.e., control) group, there was no significant change of MAP during the experimental period (i.e., from 113 :t 3 at time 0 to 107 ± 4 mmHg at 4 h, n = 8, $P > 0.05$). Post-treatment of rats with DA (3 or 10 μg kg^{-1} min^{-1}) exerted no significant acute effect on MAP when compared to the LPS group. By ontrast, the administration of DA to endotoxemic rats further enhanced the fall of delayed hypotension (i.e., after 2 h). Thus, the MAP of LPS rats treated with DA was significantly lower than that in the respective LPS control group at 2-4 h (Fig. 1B).

Effects of DA on HR in Rats Treated with LPS

Baseline values for HR of the vehicle- and DA-pre-treated animal groups were between 268 ± 12 and 276 ± 9 beats min^{-1} (n = 38), which did not significantly differ between groups (Fig. 2A). Administration of LPS caused an acute and a sustained increase in HR (i.e., from 276 ± 9 beats min^{-1} at time 0 to 365 ± 18 beats min^{-1} at 4 h, n = 14, $P < 0.05$), whereas in the sham-operated (i.e., control) group, there was no significant change in HR during the experimental period (i.e., from 272 ± 10 to 262 ± 14 beats min^{-1}, n = 8, $P > 0.05$). Pre-treatment of rats with DA (3 or 10 μg kg^{-1} min^{-1}) caused a slightly increase in HR. However, the values of pre-LPS injection (i.e., at time 0) did not significantly differ from those of other groups. Pre-treatment of rats with either doses of DA further enhanced the tachycardia elicited by LPS injection. Thus, the HRs of LPS rats pre-treated with DA were significantly higher than those in the respective LPS control group at 15 min to 4 h (Fig. 2A).

Baseline values for HR of the vehicle- and DA-post-treated animal groups were between 268 ± 13 and 273 ± 10 mmHg (n = 37), which did not significantly differ between groups (Fig. 2B). Administration of LPS caused a significant increase in HR (i.e., from 268 ± 13 beats min^{-1} at time 0 to 366 ± 15 beats min^{-1} at 4 h, n = 13, $P < 0.05$), whereas in the sham-operated (i.e., control) group, there was no significant change in HR during the xperimental period (i.e., from 270 ± 11 to 266 ± 10 beats min^{-1}, n = 8, $P > 0.05$). Post-treatment of rats with DA (3 or 10 μg kg^{-1} min^{-1}) caused a significant acute increase in HR when compared to the LPS group. In addition, the tachycardia induced by LPS was

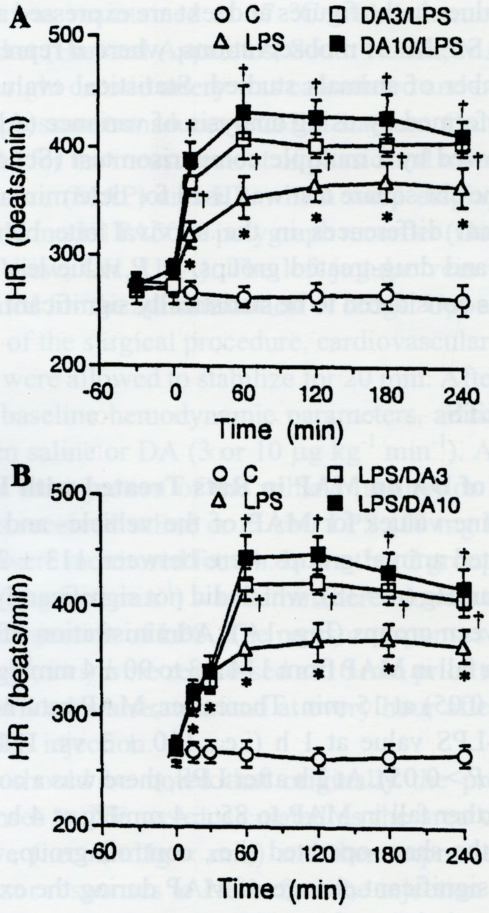

Fig. 2. Effects of (A) pre-treatment and (B) post-treatment of DA on heart rate (HR) in rats treated with endotoxin. Depicted are the changes in HR during the experimental period in different groups of animals which received injections of vehicle (C; n = 8), vehicle plus lipopolysaccharide (LPS; 10 mg kg^{-1}, n = 13-14), or DA (3. or 10 μg kg^{-1} min^{-1}, at 30 min (A) prior to or (B) after LPS) plus LPS (DA3/LPS, n = 8; DA10/LPS, n = 8; LPS/DA3, n = 8; LPS/DA10, n = 8). Data are expressed as the mean ± S.E.M. of n animals. *$P < 0.05$ represents a significant difference between the LPS group and the control group. $^{+}P < 0.05$ represents a significant difference between endotoxemic rats pre-treated or post-treated with and without DA.