were purchased from Sigma Chemical (St. Louis, MO, USA). Dopamine hydrochloride was obtained from B. Braun Medical (Emmenbrucke, Switzerland). All solutions were made in saline.

Data Analysis

All values in the figures and text are expressed as the mean ± S.E.M. of n observations, where *n* represents the number of animals studied. Statistical evaluation was performed by using analysis of variance (ANOV A) followed by a multiple comparison test (Scheffe's test). The chi-square test was used for determining the significant differences in the survival rate between control and drug-treated groups. A *P* value less than 0.05 was considered to be statistically significant.

RESULTS

Effects of DA on MAP in Rats Treated with LPS

Baseline values for MAP of the vehicle- and DA-pre-treated animal groups were between 113 ± 2 and 115 3 mmHg (n = 38), which did not significantly differ between groups (Fig. 1A). Administration of LPS caused a fall in MAP from 114 ± 3 to 90 ± 4 mmHg (n = 14, $P < 0.05$) at 15 min. Thereafter, MAP returned to the pre-LPS value at 1 h (i.e., 110 ± 3 vs. 114 ± 3 mmHg, $P > 0.05$). At 2 h after LPS, there was a continuous further fall in MAP to 85 ± 4 mmHg at 4 h (Fig. lA). In the sham-operated (i.e., control) group, there was no significant change of MAP during the experimental period (i.e., from 115 ± 3 at time 0 to 108 ± 4 mmHg at 4 h, n = 8, $P > 0.05$). Pre-treatment of rats with DA (3 or 10 μg kg^{-1} min^{-1}) slightly increased the MAP. However, the values of pre-LPS injection (i.e., at time 0) did not significantly differ from those of other groups. DA (3 or 10 μg kg^{-1} min^{-1}) did not prevent the early (at 15 min) fall in MAP observed in LPS rats treated with vehicle (saline) (Fig. lA). By contrast, pre-treatment of LPS rats with either doses of DA further enhanced the delayed hypotension (i.e., after 2 h). Thus, the MAP of LPS rats pre-treated with DA was significantly lower than that in the respective LPS control group at 2-4 h (Fig. lA).

Baseline values for MAP of the vehicle- and DA-post-treated animal groups were between 112 ± 4 and 115 ± 3 mmHg (n = 37), which did not significantly differ between groups (Fig. 1B). Administration of LPS caused a fall in MAP from 114 ± 3 to 92 ± 4

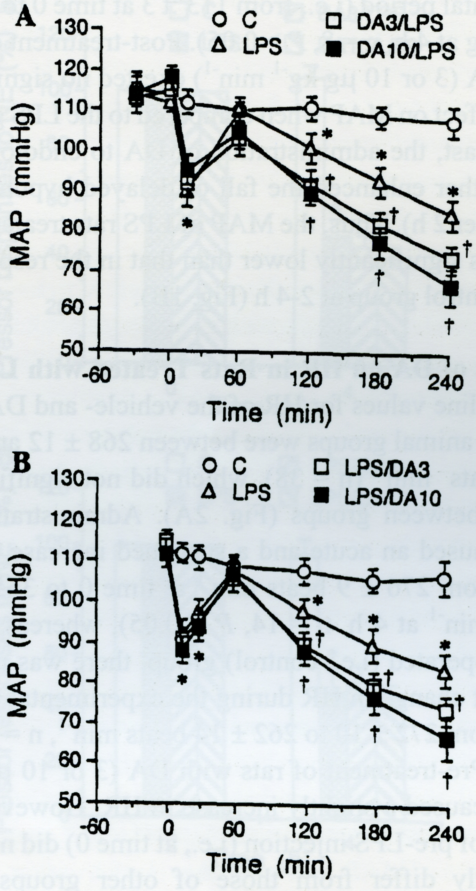

Fig. 1. Effects of (A) pre-treatment and (B) post-treatment of dopamine (DA) on mean arterial pressure (MAP) in rats treated with endotoxin. Depicted are the changes in MAP during the experimental period in different groups of animals which received injections of vehicle (C; n = 8), vehicle plus lipopolysaccharide (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.