Results

Findings in DS Strain

In DS animals, 4 weeks of HS diet (Table 1) significantly increased the arterial pressures, but had no significant effect on the heart rate. After 4 weeks of diets, heart weights (Table 1) and heart-to-body weight ratios were higher in the HS group. The HO-1 immunostaining of the left coronary artery (Fig. 1) was enhanced in vascular smooth muscle and in the endothelium of DS animals placed on 4 weeks of HS diet. The HO-2 immunostaining was apparent both in the smooth muscle and in the endothelium, but was not appreciably affected by the HS diet.

Hearts extracted (Fig. 2) from HS diet animals exhibited a higher pressure and calculated coronary resistance when perfused at a constant flow of 4.0 ± 0.1 mL/min (P < .05 LS v HS). Treatment with the HO inhibitor, 15 μmol/L CrMP, increased the perfusion pressures in LS animal hearts (102 ± 2 and 116 ± 5 mm Hg, before and 45 min after; n = 5; P < .05), but the CrMP response was exaggerated (P < .05 HS v LS) in hearts taken from the HS diet animals (115 ± 6 and 187 ± 8 mm Hg, before and 45 min after; n = 7; P < .05). Similarly, treatment with CrMP increased calculated coronary resistances in LS animal hearts (26 ± 3 and 33 ± 4 mm Hg/mL min), before and 45 min after; n = 5; P < .05), but the CrMP response was exaggerated (P < .05 HS v LS) in those taken from the HS diet animals (33 ± 6 and 51 ± 9 mm Hg/mL min), before and 45 min after; n = 7; P < .05). The CrMP-induced increases in resistance were accompanied by a faster decrease in cardiac contractility (Fig. 3) in the HS (756 ± 180; 621 ± 115, and 642 ± 132 mm Hg/sec, before, 10, and 45 min after; n = 7; P < .05) than in the LS groups (807 ± 68; 703 ± 40, and 652 ± 34 mm Hg/sec, before, 10, and 45 min after; n = 5; P < .05).


Findings in DR Strain

In complementary experiments using DR animals, 4 weeks of HS diet (Table 1) had no effect on arterial pressures or heart rates. After 4 weeks of diets, neither the heart weights (Table 1) nor the heart-to-body weight ratios were different. In addition, LS and HS animals showed no differences in either HO-1 or HO-2 immunostaining of the left coronary artery (images not shown). In contrast to our findings with the DS animals, DR hearts required a flow of 14.0 ± 1.0 mL/min to maintain a stable preparation. The LS (n = 5) and HS (n = 6) hearts extracted from the DR animals (Fig. 2) yielded similar perfusion pressures (128 ± 3 and 126 ± 6 mm Hg, LS and HS), and calculated coronary resistances (9.3 ± 0.5 and 8.7 ± 1.0 mm Hg/mL min, LS and HS). Treatment with the HO inhibitor, 15 μmol/L CrMP, similarly increased the perfusion pressures in LS and HS animal hearts (135 ± 4 and 144 ± 6 mmHg, respectively) and corresponding increases in the calculated coronary resistances (9.9 ± 0.5 and 9.5 ± 0.1 mm Hg/mL min) at 45 min. In LS hearts, CrMP-induced increases in resistance were accompanied by decreases (P < .05) in cardiac contractility (1906 ± 115, 1683 ± 259, and 1642 ± 233 mm Hg/sec; before, 10, and 45 min after; P < .05), which were similar to those measured in the HS group (2119 ± 213, 1782 ± 311, and 1759 ± 271 mm Hg/sec; before, 10, and 45 min after).