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Title: PARTIAL ILEAL BYPASS FOR HYPERCHOLESTEROLEMIA: 5-YEAR LIPID RESULTS OF THE PROGRAM ON THE SURGICAL CONTROL OF THE HYPERLIPIDEMIAS

The Program on the Surgical Control of the Hyperlipidemias is a prospective, randomized trial evaluating the effect of lipid modification by partial ileal bypass on overall mortality and the course of coronary artery disease following a single myocardial infarction. Complete, 5-year lipid results are available for the first 330 diet-treated control and 330 surgery patients in this study. Partial ileal bypass, initially described by our group in 1963, consists of bypass of the distal one-third or the distal 200 cm of small intestine, whichever length is greater:

| | | C | LDL | HDL | VLDL | TG |
|---|---|---|---|---|---|---|
| Control | B | 249±31 | 177±34 | 40±10 | 31±23 | 204±145 |
| (n=330) | 5-Yr | 237±34 | 166±34 | 40±10 | 31±26 | 192±146 |
| Surgery | B | 251±34 | 179±36 | 40± 9 | 31±22 | 201±122 |
| (n=330) | 5-Yr | 179±33 | 100±27 | 42±10 | 36±30 | 229±161 |

B=Baseline. C=Total Cholesterol. TG=Triglycerides. All values as mean±SD in mg/dl.

Partial ileal bypass reduced total cholesterol 24%, reduced LDL 39%, raised HDL 7%, raised VLDL 17%, and raised triglycerides 24% compared to dietary therapy. The HDL/total cholesterol ratio was 47% higher, and the HDL/LDL ratio was 91% higher in the surgery patients. In a subgroup of these patients, apo A-I was 15% higher, apo B-100 was 27% lower, HDL-2 was 33% higher, and HDL-3 was unchanged 5 years following surgery. These diet-controlled, 5-year lipid results demonstrate that partial ileal bypass leads to significant, sustained lipoprotein normalization in hypercholesterolemic patients. These lipid chan,es compare favorably with results obtained from dietary or multiple agent pharmacologic therapy.

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Title:

EFECTS OF CYCLOSPORIN A, ALLOPURINOL AND WHOLE BODY RADIATION ON EXPERIMENTAL ACUTE INTESTINAL ISCHAEMIA

INTRODUCTION.- Preliminar studies performed in our laboratory suggested the possible participation of immune system in the fisiopathology of experimental intestinal ischemia in rats. The present work was designed in order to study the mechanism of action of different therapies which were suppose to affect survival after acute intestinal ischemia.

MATERIAL AND METHODS.- Four groups of 25 female Sprague Dawley rats (200 g. weight) were considered: control (Ctrl), CsA, allopurinol (Allo), whole body radiation (WBR). After a fasting period of 12 h., animals were anesthetized with penthobarbital sodium (30 mg/kg) and superior mesenteric artery was clamped for 2 h. The extent, macroscopic and microscopic appearance of the ischemic lesions were studied in five rats from each experimental group sacrified 30 minutes after releasing the intestinal clamp. Mortality and surviving period rates were quantified in the remainder.

RESULTS.- Mortality rates 48 h. after revascularization were as follows: Ctrl (75%), CsA (35%), Allo (35%), WBR (40%). Diminution of mortality rates accounted by all three treatments, was statistically significant ($p < 0.05$). Mean survival time among death animals was: Ctrl (11.86 ± 4.7 h.; n=5), CsA (17.42 ± 9.2 h.; n=7), Allo (13.71 ± 10.6 h.; n=7), WBR (23.75 ± 9.2 h.; n=8). Increase of mean survival time accounted by CsA and WBR was statistically sifnificant (U=20, $p < 0.05$; U=14, $p < 0.01$). Percentage of ischemic intestine was: Ctrl (44.4 % ± 3.7), CsA (41.4 % ± 5.7), Allo (35.2 % ± 3.7), WBR (42 % ± 3.1). Allo-treated was the only group showing statstically significant decrease ($p < 0.01$). Microscopic studies revealed different degree of ischemic damage in experimental series: Ctrl (3.92 ± 0.5), CsA (5.1 ± 0.6), Allo (4.82 ± 0.49), WBR (4.18 ± 0.76), but only CsA and Allo resulted statistically significant (U=1.5, $p < 0.05$).

CONCLUSION.- The three experimental series improved survival after intestinal ischemia. Differences in the other parameters studied suggest that this treatments do not share the same mechanism of action. Results obtained with CsA and WBR are in accordance with our hypothesis of a possible role of the immune system in the fisiopathology of this syndrome.