also that mice responded less to methyleugenol. Thus, with 300 mg/kg they had about 20 min of tail insensibility and 38 min of righting reflex loss. Nevertheless, during this time period the mice could be submitted to surgical procedures in the abdomen; surgery in the brain was not tried.

Finally, table 2 gives the results employing methyleugenol suspended with cremophor EL in 2 surgical procedures. 2-month-old female reats were stereotaxically operated for unilateral destruction of substantia nigra through electrolysis. With sodium pentobarbital 7 out of 15 animals died and all of them presented bronchial secretion; it was difficult to find an optimal dose, as with 30 mg/kg most females still reacted to surgical manipulations whereas 40 mg/kg was too much for several of them. Conversely, no such problems occurred under 250 mg/kg of methyleugenol. All animals recovered well within 90–120 min after injection and no deaths or signs of sickness were noticed 1 week later. The 2nd surgical procedure, implantation of 7 electrodes for EEG and EMG recordings, took about 90 min to complete. 275 mg/kg of methyleugenol yielded a good degree of anesthesia although for a few animals a supplementary dose of 50 mg/kg was necessary after the 60–70th min. Even with this larger dose only 1 animal was lost during surgery; a 2nd animal died of infection 2 days later. It is worth mentioning, too, that soon after losing the righting reflex (about 2 min) the animal can be easily handled for the preparatory acts of surgery such as shaving the head and placement in the stereotaxic apparatus. This can save the time elapsing between loss of the postural reflex and the onset of insensitiveness (table 1).

In summary, our data show that methyleugenol can be an useful anesthetic agent for surgery in rats and mice. It is a cheap drug, well tolerated by the animals, it is easily available as its use is not restricted by law, and suspensions of it, which are stable for at least 2 weeks, can be prepared with the help of 2 easily available inert substances, cremophor EL and Tween-80.

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Effect of acetylsalicylic acid on iron absorption in the rat[1,2]

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Summary. The in vivo administration of ^{59}Fe to the rat accompanied by acetylsalicylic acid (ASA) enhanced significantly counts in blood, spleen,liver and femur without affecting those of the intestine. The results suggest that ASA augments iron absorption either via an inhibitory action on the synthesis of prostaglandins or by a purely chemical mechanism.

Several factors can influence intestinal iron absorption[3]. One, operative at the level of the intraluminal compartment, is related to the different forms of iron intake as well as with the simultaneity of food administration[4-8]. Another relevant influence is the intrinsic ability of the intestinal mucosa to absorb iron[9-11]. In addition the total body iron requirements are also important for the maintenance of a normal erythropoiesis[12,13]. In previous studies with isolated rat intestine we have documented that prostaglandins E_1 and E_2 can influence the mechanism of intestinal iron absorption, acting in a double fashion: a) on the uptake of iron by the villous cells of the intestine and b) on the processes by which the iron is transported at the serosal end of the intestinal barrier[14]. Based on these results it was decided to study the effect of acetylsalicylic acid, an inhibitor of prostaglandin synthesis, on iron absorption in vivo.

Methods. Male Wistar rats (200 ± 20 g) were bled by cardiac puncture 1.5 ml per 100 g b.wt during 2 consecutive days. 24 h later another bleeding of 0.75 ml per 100 g b.wt, was performed. After these 3 days of bleeding the animals developed an anemia with hemoglobin values below 10 g%. The rats were then starved during 24 h and forthwith divided in 2 groups: group I (control) receiving ^{59}Fe via a catheter placed in the stomach (0.5 μCi or equivalent to 600,000 cpm), together with an iron carrier of ferrous sulphate; group II (experimental) treated in the same way as group I but also receiving 10 mg of acetylsalicylic acid (ASA). The amount of ^{59}Fe was monitored in blood, liver,

Effect of acetylsalicylic acid (ASA) on the incorporation into several tissues of orally given ^{59}Fe

Variables	Control* (n = 8)	ASA** (n = 9)	Significance***
Body weight (g)	195 ± 10	194 ± 10	NS
Hemoglobin (g%)	9.6 ± 0.2	9.6 ± 0.4	NS
Hematocrit (%)	32 ± 1	32 ± 1	NS
Blood (cpm in 2 ml/100 g b.wt)	598 ± 150	1733 ± 437	$p < 0.02$
Spleen (cpm/100 mg w.wt)	192 ± 48	511 ± 130	$p < 0.02$
Liver (cpm/100 mg w.wt)	37 ± 3	55 ± 8	$p < 0.02$
Wet washed empty intestine (cpm/100 mg w.wt)	3051 ± 706	2234 ± 625	NS
Freshly removed intestine (cpm/100 mg w.wt)	278 ± 40	311 ± 82	NS
Dry intestine (cpm/100 mg w.wt)	1379 ± 227	1626 ± 479	NS
Femur	551 ± 119	984 ± 170	$p < 0.05$

* Determinations made of 180 min following ingestion (see 'methods' section). ** Means ± SEM. *** Student's t-test.

spleen, femur and intestine, the determinations being made at 180 min following the delivery of ^{59}Fe. In the case of intestine the ^{59}Fe uptake was determined immediately after removal as well as following washing the lumen with a known volume of saline or after dessication during 24 h at 80 °C. The dry and wet weights of intestine and the wet weights of spleen and liver were also obtained and results refered to cpm of ^{59}Fe per 100 mg of organ. Counts in blood and femur were expressed per 100 g b.wt and per femur, respectively. For statistical assessment the results were compared employing Student's t-test and differences between means were considered significant if $p=0.05$ or less.

Results. The table shows that at 180 min following the administration of ^{59}Fe accompanied by ASA there is a significant increase of counts, in comparison with the untreated controls, in blood, spleen, liver and femur. On the other hand, in freshly removed, in wet washed or in dry intestine of the ASA treated group the values were comparable to those found in controls.

Discussion. The foregoing results suggest that in the rat the action of ASA is to augment the absorption of iron. This influence is evident at 3 h following ingestion through a catheter placed in the stomach. Indeed an enhanced count of ^{59}Fe in the ASA-treated group was found in blood, spleen, liver and femur, whereas comparable iron counts were detected in the freshly removed, the wet washed as well as in the dry intestine. It is therefore plausible that an enhanced intestinal iron absorption in the ASA-treated group is followed by higher circulating levels of iron and by an enhancement in deposit organs. The influence of ASA on this phenomenon could probably be related to the action of ASA on the intestinal cycloxygenase system. However it must be noticed that another effect of the drug, independent of the mechanism of prostaglandin inhibition (e.g. purely chemical mechanisms such as are already known with ascorbic acid) cannot be discarded. Nevertheless the tentative hypothesis regarding the participation of prostaglandins is plausible in view of in vitro studies documenting that these compounds can alter iron fluxes through the intestine. Indeed, the present results are compatible with previous findings indicating that prostaglandins E_1 and E_2 diminished the passage of iron across the mucosa and serosa of the isolated rat intestine as well as with the report that indomethacin is able to enhance the same process in vitro[14].

1 This work was supported by Grant No. 6638 from CONICET (Argentina).
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Comparison of the responsiveness of isolated arteries and veins taken from the same dogs 17 days apart

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Summary. The present experiments demonstrate that isolated arteries and veins taken from the same dogs before and after an interval of 17 days show comparable responses to adrenergic nerve stimulation, exogenous norepinephrine and depolarizing solution. They provide an acceptable model for the in vitro study of chronic influences on canine vascular responsiveness.

The aim of the present experiments was to determine whether or not 2 sets of isolated blood vessels can be obtained from the same dogs after a sufficient time interval to allow the study of the effects of chronic surgical interventions or chronic administration of drugs on vascular responsiveness.

Pairs of dogs (mean weight 25.1 ± 2.4 kg) from the same litters ($n=6$) were studied on the same days. On day 1, the dogs were anesthetized with pentobarbital (30 mg/kg, i.v.); segments (3 cm) of the lateral saphenous vein and of the tibial artery of the left hindpaw were removed. After surgery, the dogs were allowed to recover. On day 17, the dogs were anesthetized again with pentobarbital; the lateral saphenous vein and the tibial artery of the right hindpaw were then removed.

From each segment of saphenous vein and tibial artery, 3 rings (4 mm width) were prepared and these were mounted in organ chambers filled with aerated Krebs-Ringer solution at 37 °C (m molar composition: KH_2PO_4, 1.2; NaCl, 118.3; KCl, 4.7; $MgSO_4$, 1.2; $CaCl_2$, 2.5; $NaHCO_3$, 25; Na_2Ca EDTA, 0.026; glucose, 11.1). The preparations were placed at the optimal point of their length-tension curve using a standard electrical stimulation[1,2]. After 30 min equilibration, either a frequency-response curve to electrical stimulation[3], a dose-response curve to norepinephrine, or a dose-response curve to increasing concentrations of K^+ in the presence of 10^{-5} M phentolamine[4,5], were obtained simultaneously in the 3 segments from each saphenous vein and tibial artery. For statistical analysis, Student's t-test for unpaired observations was used. The responses of both blood vessels of the same dog were compared on day 1 and day 17, while the comparison between the 2 dogs of the same litter was performed either on day 1 or day 17.

The optimal tension was comparable in the 4 groups of arteries and in the 4 groups of saphenous veins. In both the arteries and the veins similar frequency-response curves to electrical stimulation (0.5-64 Hz) were obtained on day 1 and day 17; no significant differences were noted between