Paracellular route. The small intestinal epithelium is not a 'tight' epithelium[15,16] and therefore a backdiffusion of already absorbed substances through the tight junctions occurs. This back diffusion could explain the behaviour of net secretion of Na^+ in case of perfusion solutions with a Na^+ concentration below the serum concentration.

Cell loss. Increased cell loss from the villus tops could be the reason for a reduced glucose absorption, but from the investigations of CLARKE[17], such mucosal cell loss seems unlikely.

There seems to be the following mechanisms by which vincristin may influence sugar transport: First, by reduced engergy production due to inhibition of metabolic pathways. Secondly it is known that microtubuli influence membrane characteristics[18]. Therefore membrane transport of glucose (input or output) may be decreased by vincristine. Another membrane located transport system may be influenced – the sodium transport system. Sugar transport shows a sodium dependency in vivo also[19,20], and there is a correlation between net transport of sodium and net sugar transport[21,22]. Two findings cast some doubts on the theory that the reversed net transport of sodium is the reason for the decrease in glucose transport: the impairment of net sodium transport is detectable even in the first sampling period and there seems to be a dependency of the direction of net sodium transport upon the luminal sodium concentration.

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Changes in the Distensibility of the Cat Aortic Arch Induced by Noradrenaline

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Summary. In the isolated preparation of the aortic arch of the cat, noradrenaline (NA) reduced at low pre-loads, and increased at high pre-loads, the arterial wall distensibility. For each dose of NA, the changes were directly related to the pressure level in the system.

Sympathetic fibres originating in the stellate ganglion of cats, rabbits and mice are known to be incorporated into the aortic nerve[1-3], and to terminate in the smooth muscle of the aortic wall. Electron microscopy of the wall of the aortic arch in cats revealed the presence of nerve endings which contained vesicles[4]. When observed in the vicinity of the smooth muscle fibres of the adventitia, they resembled very closely those described in other vascular areas, generally considered to have an effector function[5-9]. They persisted after degeneration of the aortic nerve induced by section at the neck level[4]. The functional significance of these fibres is still unknown, but since they appear to be effector in nature, it seems reasonable to suggest that an increase in this activity would modify the contractile state of the smooth muscle, thereby resulting in a change in the elastic properties of the aortic arch. The characteristics of these fibres make noradrenaline the most likely candidate for the neurotransmitter. Therefore, an in vitro study was undertaken to analyze the effects of NA on the aortic arch distensibility through changes induced on the pressure-volume diagram. Since the arterial distensibility is reduced as the mean blood pressure increases[10], one could not dissociate in an intact preparation the distensibility changes induced directly by NA on the arterial wall from those secondary to the hypertensive effect of the amine.

Material and methods. Segments of aortic arch, about 30 mm long, were removed from anesthetized cats and isolated from surrounding tissue. All branching arteries were ligated. Both ends of the segment were fitted to 2 metalic cannulae, one of which was closed and the other connected to the system of the volume injection. The whole assembly was rigidly attached to 2 upright blocks, able to slip on a horizontal bar when displaced by a force of 1 g or less. The assembly was mounted in a tissue bath maintained at 36–36.5 °C and filled with Hank's solution (pH 7.3–7.4) equilibrated with 5% CO_2 + 95% O_2. One of the 2 upright blocks was firmly attached to a bar, whereas the other was connected to a pulley system to which longitudinal loads of 7.5, 27.5 and 52.5 g were applied. Each load was used in a group of 7 preparations. A period of at least 30 min elapsed from the pre-loading until the start of the experiment.

The segments were inflated with Hank's solution by means of an infusion delivered by a pump at the constant rate of 5 ml/min. Total volume inside the segment could be calculated at any time by adding to the initial volume of the segment the volume computed from the time scale of an oscilloscope. The initial volume was calculated from the formula $V = h\pi r^2$. h and r were measured at the end of each experiment after performing a longitudinal excision of the wall and extending the segment on a flat surface. It was previously observed that pre-loading the segment did not modify the V value, because the increase in length compensated for the reduction of the

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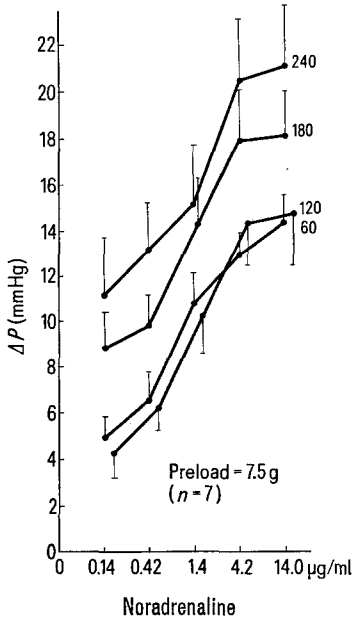

Fig. 1. Pressure changes induced in the isolated cat aortic arch by increasing doses of noradrenaline. Each curve represents the changes obtained at a pre-determined control pressure value (60, 120, 180 and 240 mm Hg). Pre-load: 7.5 g. Vertical bars refer to SEM.

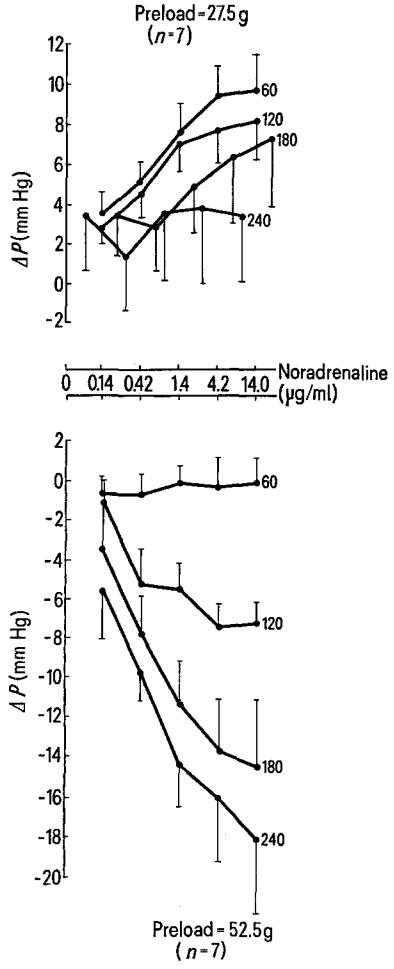

Fig. 2. Same as in Figure 1. Pre-loads: 27.5 and 52.5 g.

radius. The pressure was measured with a Statham P23Db transducer. Pressure-volume (*P-V*) diagrams were directly displayed on an oscilloscope (Tektronix 5013). Infusion and onset of sweep were synchronized so that the time base of the oscilloscope gave injection rate (ml/min). Measurements were obtained from photographs performed with a Tektronix C59 camera. When successive control *P-V* diagrams became identical, increasing doses of noradrenaline bitartrate (Schering) were added cumulatively to the bath until the peak effect was attained. The concentration ranged from 0.14 up to 14 µg/ml.

Results. *P-V* diagrams showed a typical S-shaped curve with 2 inflections at the pressure levels of 60–100 and 140–180 mm Hg, respectively. As the longitudinal pre-load was increased, both inflections were displaced toward lower pressure values, so that in group 3 the first inflection was consistently attained at a level below 60 mm Hg. 4 reference points were previously selected on the X-axis (volume) of the control *P-V* diagram, corresponding to the pressure values of 60, 120, 180 and 140 mm Hg. For each dose of NA, the pressure change (ΔP) corresponding to each reference point was measured. A dose-response curve was thus obtained for every reference point by plotting pressure changes vs dose of NA.

In group 1 (pre-load: 7.5 g, Figure 1) NA induced a dose-dependent increase in *P* at each reference point. The increments became more elevated at the higher reference points. This means that NA induced a reduction of the arterial distensibility, and that the reduction was more evident as the artery became more deformed by the infusion. In group 2 (pre-load: 27.5 g, Figure 2, upper half) NA again induced a dose-dependent increase in *P*. However, NA was only able to increase dose-dependently the *P* in the lower part of the *P-V* diagram, but it failed to modify the pressure in the higher part of the diagram. At the pre-load of 52.5 g (group 3, Figure 2, lower half) NA displaced the *P-V* diagram to the right, so that for every reference point the amine reduced the pressure (negative ΔP) in a dose-dependent manner. Furthermore, for each dose of NA, the magnitude of the reduction in pressure was directly related to the pressure level of the system. The curves seem to indicate that, as the pre-load in the aortic arch increases and at pressure levels above 60–100 mm Hg, NA increases the distensibility of the arterial wall.

Discussion. Our results obtained in the aortic arch partially agree with those obtained by several authors in other segments of aorta. In the abdominal aorta of intact dogs, ALEXANDER[11] observed that adrenaline reduced the diameter of the segment and increased the distensibility in a pressure range of 0–150 mm Hg. Similar results were obtained in the dog aorta by WIGGERS and WEGRIA[12]. Working on data reported by GOLDENBERG et al.[13], COPE[14] concluded that NA was able to increase the aortic distensibility in humans, whereas adrenaline induced inconsistent effects. The aortic archs of our group 3, probably subjected to a more 'physiologic' longitudinal tension, responded similarly to NA with an increase in the distensibility of the wall. It is evident that, at least in the aortic arch, the degree of longitudinal tension be-

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comes a critical factor, determining the response of the wall as a whole to NA. Although the special architecture of the aortic arch makes it difficult to determine in situ the exact longitudinal tension of the wall, the present results seem to indicate that the response of the aortic arch to NA does not differ considerably from that observed in other aortic segments, if we accept that the longitudinal tension applied to the archs of group 3 (52.5 g) was the most closely related to the actual values. It is interesting to point out that a longitudinal tension of 55 g was applied to the descending thoracic aorta in order to restore in situ the full length of previously excised segments[15].

The increase in distensibility has been explained on the basis of structural peculiarities[16], whereby the NA-induced contraction of the smooth muscle fibres would result in a reduction of the tension of the more fibrous, less distensible component of the vascular wall, so that the smooth muscle, which is more distensible, would determine the shape of the P-V curve.

Since the baroreceptors can be stimulated in a different way depending on the distensibility of the aortic wall, it is conceivable that changes in distensibility induced by the NA release of efferent fibres in the aortic arch could provide a mechanism for efferent control of the receptors activity. Alternatively, they could provide a system which would adapt the arterial tree impedance to variable cardiac output and heart rate during sympathetic stimulation[17].

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Influence of Gonadal Hormones on the Developing Amphibian Brain: Changes in Ribonucleic Acid, Protein and Activity Levels of Acetylcholinesterase on in vivo Administration of Progesterone

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Summary. The levels of RNA and protein and activity levels of acetylcholinesterase decreased in the brain of developing tadpoles of 13–15-day-old *Bufo melanostictus* on in vivo administration of progesterone (200 µg/0.1 ml refined peanut oil). These changes suggest decelaration in the activity of the protein-synthetic machinery in progesterone administered animals.

There are a large number of reports showing changes in the content and/or composition of RNA of the neural tissue of vertebrates under a wide variety of conditions[2–7]. For instance, the cytoplasmic RNA amount increases after excitation of nervous activity and decreases after inhibition of nervous activity, intoxication, and pathogenic action in mammals[2]. Increase of neuronal and glial RNA has been described in diseased areas associated with Parkinsons disease in rats[3].

Evidence is accumulating that various hormones affect the process of protein synthesis in the CNS of vertebrates at the translation level. For instance, steroid hormones are known to influence the protein synthesis by acting on the DNA – guided formation of RNA in the brain of rats[8, 9].

However, information is lacking on steroid hormone regulation of protein synthesis. Since gonadal steroids have an organizing influence on CNS centres during critical periods of development in vertebrates[2], the present study was proposed. The paper presents information about the effects of in vivo administration of progesterone on the levels of RNA and protein in the brain of a developing toad, *Bufo melanostictus*. Since manipulation of the hormonal environment in the early neonatal period affects the brain excitability, the activity levels of acetylcholinesterase as a function of progesterone administration were determined in the CNS of developing tadpoles.

Materials and methods. About 2-week-old tadpoles of *B. melanostictus* were collected locally and maintained in the laboratory at 24 ± 1 °C in glass aquaria. They were fed on water plants for a period of 3 days before they were utilized for experimentation. From this stock,

Changes in the levels of RNA, protein and activity level of acetylcholinesterase (AchE) in the brain of developing *Bufo melanostictus* on in vivo injection of progesterone

Constituent	Controls (normal active animals)	Experimentals injected with progesterone	Incidence of change
RNA (mg/g wet wt.)	20.7 ± 3.5[a] (3)	7.5 ± 1.3[a] (4)	− 64.6
Protein (mg/g wet wt.)	120 ± 6.1[a] (3)	96.0 ± 8.5[a] (3)	− 20.0
AchE µmol Ach (mg/min)	5.45 ± 38[a] (4)	3.6 ± .44[a] (4)	− 34.6

Figures in parentheses indicate the number of pools-observations Values are mean ± SD. For each observation tissue from 16–18 animals was pooled. Sign (−) indicating decrease over controls. [a] Significant $p < 0.01$.

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