primarily in the anteroventricular cochlear nucleus (AVCN)[3-5]. Other transmitters discussed in this area of the brain are: acetyl choline, which has been located primarily to the granular layer of the cochleus nucleus (GCN)[6], and γ-aminobutyric acid, which has been located predominaltly to the AVCN[7]. Although our aim in this study has mainly been to develop a system where transmitter release could be demonstrated, given physiological stimuli, it is certainly of interest in future studies to localize more precisely those terminals releasing ^{3}H-glutamate, and to confirm that exogenous ^{3}H-glutamate could be used as a marker for the endogenous transmitter.

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Dopamine and cerebral cortical blood flow in the rat

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Summary. Dopamine topically applied to the cerebral cortex (1–20 µg/ml) or administered i.v. (0.5–64 µg/kg/min) has no effects on cerebral cortical blood flow in the rat.

Dopamine agonists induce complex central nervous effects[1-4]. The possible role of cerebral vascular receptors in those actions has been suggested, since nerve terminals containing dopamine have been demonstrated in cerebral arteries[5-7] and dopamine has been shown to modify blood flow in several organs[8]. Several authors have reported increases in cerebral blood flow following intravenous administration of dopamine agonists[9,10]. The effects of topical application of dopamine to cerebral blood vessels in vivo have not been evaluated however, and the possibility exists that the above mentioned effects of intravenous dopamine agonists might be mediated indirectly, particularly in view of the fact that dopamine constricts (rather than dilates) cerebral vessels in vitro[11]. To evaluate a possible direct cerebral vascular action of dopamine, this drug was topically applied to the exposed cerebral cortex while local blood flow was being measured at the same site by the hydrogen clearance technique. In another series of experiments, cerebral cortical blood flow (CoBF) was measured in the same way while dopamine was given intravenously.

98 albino rats were used. The animals were anesthetized by intraperitoneal administration of 1.5 g/kg urethane. A femoral artery was cannulated and the mean arterial pressure (MAP) was continuously monitored. The parietal cortex was exposed on one side and a platinum electrode (30 µm in diameter) was inserted 0.5 mm into the cortex in order to record the tissue hydrogen (H_2) concentration. H_2 was given by inhalation until a steady state concentration was attained in the tissue. The rate of tissue H_2 desaturation after interruption of the H_2 inhalation was used to calculate blood flow. Details of the technique can be found elsewhere[12,13]. Dopamine, dissolved in synthetic cerebrospinal fluid[14] at concentrations from 1–20 µg/ml was topically applied to the cortex surrounding the H_2 sensing electrode. In another set of experiments, dopamine was infused i.v. at rates from 0.5 to 64 µg/kg/min. The concentration of the solutions was adjusted to deliver the same infusion rate of fluid (6.8 µl/min) in all experimental groups. CoBF was measured immediately before and at 30 min intervals after the commencement of the dopamine application or infusion. In a number of animals, only 0.9% NaCl was infused, or artificial CSF was applied as a control.

Results and discussion. Topical application of dopamine to the cerebral cortex did not change CoBF significantly (table 1). During continuous intravenous infusions, a progressive increase in CoBF was observed for all concentrations of the drug as well as in animals infused with 0.9% NaCl alone. MAP showed a tendency to decrease during the infusion for all concentrations tested. It is concluded that dopamine has no effect on CoBF when administered topically or i.v. to rats.

The increase in CoBF during prolonged infusions, both in controls and in treated animals, is probably related to a slow dissipation of the anesthetic (which was given in a single injection at the beginning of the experiments) since CoBF has been shown to be related to the level of urethane

Table 1. Cortical blood flow (CoBF) before and after topical application of dopamine to the cerebral cortex

	Mean CoBF before application of test solution (± SE)	Mean change (± SE)* in CoBF after application of test solution	n
Synthetic CSF (no drug)	43.26 ± 13.52	0 ± 2.73	15
Dopamine 1 µg/ml	51.88 ± 5.71	5.50 ± 4.83	8
Dopamine 5 µg/ml	31.75 ± 2.74	2.17 ± 2.70	12
Dopamine 10 µg/ml	35.20 ± 6.74	3.50 ± 2.89	10
Dopamine 20 µg/ml	27.29 ± 2.22	2.07 ± 2.10	14

CoBF is given in ml/100 g/min. All differences were insignificant with the Student t-test for paired samples. * Mean ± SE of the difference between the first determination of CoBF after, and the last determination before, application of test solution. n = number of animals.

Table 2. Cortical blood flow (CoBF) and mean arterial pressure (MAP) before and during continuous intravenous infusion of 0.9% NaCl or dopamine at various rates

Experimental condition	Before infusion	Time from onset of infusion 15 min	45 min	75 min	105 min
0.9% NaCl (11)	CoBF 30.09±5.96	32.10± 5.39	36.00± 6.01	48.27±12.57	50.80±13.07
	MAP 71.73±2.77	65.00± 4.21	61.09± 4.97	60.00± 5.28	55.90± 6.93
Dopamine (6)	CoBF 43.17±9.14	51.33±10.88	51.83±10.90	63.33±15.85	62.50±14.71
0.5 µg/kg/min	MAP 58.50±2.88	61.83± 3.01	61.00± 4.93	53.17± 6.47	45.20± 8.09
Dopamine (6)	CoBF 25.17±5.15	31.17± 5.66	37.00± 9.47	42.33±11.55	34.16± 8.86
2 µg/kg/min	MAP 73.50±4.29	66.66± 8.33	57.50± 7.07	52.83± 6.94	52.00± 7.60
Dopamine (8)	CoBF 27.50±2.65	30.87± 3.41	34.50± 2.32	46.50± 8.40	42.37± 6.71
4 µg/kg/min	MAP 68.75±4.52	71.25± 4.59	77.00± 4.63	73.50± 3.36	69.38± 5.25
Dopamine (8)	CoBF 22.62±3.09	30.62± 6.35	32.87± 5.64	33.37± 5.55	40.50± 9.12
64 µg/kg/min	MAP 71.00±5.21	57.38± 4.89	52.63± 5.57	50.63± 5.55	46.63± 6.09

CoBF is given in ml/100 g/min. MAP in mmHg. Values are mean ± SE. Number of animals used indicated in parentheses.

anesthesia in the rat[13]. These results are in line with our previous observations of a very limited effect of adrenergic agents on cortical blood vessels[15] contrasting with the important dilator effects of cholinergic agents[13-16].
The central effects of dopamine agonists and antagonists do not seem to be related to dopamine vascular receptors.

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Motility and vitality of human spermatozoa at various time intervals after ejaculation

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Summary. Motility and vitality of spermatozoa from semen differing in sperm density were assessed at various intervals after ejaculation. Percentile decreases in both parameters were found to be higher in oligospermic specimens than in those with higher sperm densities.

The importance of the motility and survival of sperm has been accepted to be crucial for penetration through the cervical mucus, uterotubal junction and ovum membranes in the process of conception[2-8].
The present experiments were designed to examine the decreases in both motility and viability of spermatozoa at various time intervals after ejaculation in specimens differing in sperm density. Our purpose was to confirm a previous observation[9], according to which spermatozoa originating from oligospermic semen behave differently from those of the normospermic semen, with respect to motility and viability.
77 semen samples, with counts ranging from 0.5 to 200 million spermatozoa/ml were obtained after 4 days of abstinence from men attending our clinic. The samples were divided into groups (1-3) according to sperm counts (table 1). After being kept at 37 °C, each specimen was routinely evaluated for the percentage of motile cells, and the motility grade[10] at 1, 3 and 5 h following ejaculation.
The proportion of vital cells was assessed using 3 techniques, namely the eosin test (mixture of a drop of semen and a drop of 2% aqueous eosin blue), 1% eosin y-nigrosin, and 5% eosin y-nigrosin[11], at the same time intervals and additionally 24 h later.
Mean percentages of motility and vitality were calculated and the differences between 2 successive groups and between time intervals within the same group were compared using the Student t-test. The proportions of vital sperm obtained with 5% eosin y-nigrosin were lower than those with 1% eosin y-nigrosin, as reported by Dougherty et al.[11]. The differences between the 2 methods, however, were not statistically significant. The values presented in table 1 refer to the 5% eosin-nigrosin test.
Percentage activity and viability (eosin and eosin-nigrosin