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.

1 N.E. Anden, A. Rubenson, K. Fuxe and T. Hökfelt, J. Pharm. Pharmac. *19*, 627 (1967).
2 A.R. Cools and J.M. Rossum, Archs. int. Pharmacodyn. Thér. *187*, 163 (1970).
3 B. Costall and R.J. Naylor, Eur. J. Pharmac. *24*, 8 (1973).
4 S. Lal and C.E. de la Vega, J. Neurol. Neurosurg. Psychiat. *38*, 722 (1975).
5 U. Ungerstedt, Acta Physiol. Sc. suppl. *367* (1971).
6 O. Lindvall and A. Björklund, Acta physiol. scand. suppl. *412* (1974).
7 C. von Essen and B.E. Ross, Acta Pharmac. Toxicol. *35*, 433 (1974).
8 L.I. Goldberg, Pharmac. Rev. *24*, 1 (1972).
9 C. von Essen, Acta Neurol. scand. *50*, 39 (1974).
10 G. Teasdale and T. McCulloch, in: Cerebral Function Metabolism and Circulation, p.98. Ed. Munksgaard, Copenhagen 1977.
11 N.T. Zervas, M.H. Lavyne and M. Negoro, New Engl. J. Med. *293*, 812 (1975).
12 K. Auckland, Circulation Res. *14*, 164 (1964).
13 O.U. Scremin, A.A. Rovere, A.C. Raynald and A. Giardini, Stroke *4*, 232 (1973).
14 J.K. Merlis, Am. J. Physiol. *131*, 67 (1940).
15 A.A. Rovere, A.C. Raynald and O.U. Scremin, Experientia *33*, 1461 (1977).
16 O.U. Scremin, E.H. Rubinstein and R.R. Sonnenschein, Stroke *9*, 160 (1978).

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

Rina Singer[1], M. Sagiv, B. Landau, D. Allalouf, E. Segenreich and C. Servadio

Male Fertility Unit, Rogoff-Wellcome Medical Research Institutes, Beilinson and Hasharon Hospitals, Petah Tikva (Israel), 2 July 1979

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

Table 1. Motility and vitality of spermatozoa from human semen differing in sperm density, at various time intervals more distant groups

Sperms/ml ($\times 10^6$)	No. of samples	Motility %	Grade	Vitality Eosin	Eosin-nigrosin	Motility %	Grade	Vitality Eosin	Eosin-nigrosin	Motility %	Grade	Vitality Eosin	Eosin-nigrosin	Vitality Eosin	Eosin-nigrosin
		1 h	1 h	1 h	1 h	3 h	3 h	3 h	3 h	5 h	5 h	5 h	5 h	24 h	24 h
Group 1 0.5–10	34	40.1±3.0	1.7±0.1	56.3±2.7	53.2±2.7	33.0±2.7	1.3±0.07	46.5±2.8	43.5±2.7	25.1±2.3	1.1±0.05	40.2±2.5	37.5±2.5	10.9±1.8	13.1±2.4
Group 2 10.5–40	20	50.5±2.2	2.2±0.1	60.9±2.5	60.6±2.7	40.4±2.8	1.8±0.1	49.2±2.7	51.5±2.7	36.1±2.7	1.5±0.1	46.4±2.0	47.0±2.3	15.4±3.2	16.4±3.6
Group 3 40.5–200	23	58.5±3.1	2.4±0.1	64.5±2.4	62.6±1.9	48.4±2.6	1.9±0.1	51.9±2.4	52.4±1.7	44.8±2.2	1.7±0.1	49.9±1.8	46.7±1.5	19.1±2.1	19.4±2.4

Values are mean ± SE. The differences between 2 successive groups and time intervals within the same group were not always statistically significant. The comparison between (1 and 3) or longer time intervals (1 and 5 h) were usually significant ($p \leq 0.005$).

Table 2. Percentile decreases in motility and vitality 5 h after ejaculation in comparison to values recorded after 1 h

Sperms/ml ($\times 10^6$)	No. of samples	Motility %	Grade	Vitality Eosin	Eosin-nigrosin
		5 h	5 h	5 h	5 h
Group 1 0.5–10	34	37.5*	35.3*	28.6*	29.6*
Group 2 10.5–40	20	28.6	31.9	23.8	22.5
Group 3 40.5–200	23	16.3	29.2	22.7	25.0

* The numbers are percentages of decrease.

tests), and motility grades, were found to increase gradually with increasing sperm counts and to decrease with time intervals (table 1). The differences between 2 successive groups or time intervals were not always statistically significant. In such cases comparison between more distant groups (1 and 3) or longer time intervals (1 and 5 h), were usually significant ($p \leq 0.005$).

Percentile decrease in motility (percentage and grade) and vitality 5 h after ejaculation (table 2) were more significant in specimens with low sperm densities (group 1) than in the less oligospermic and normospermic. Increased percentages of active and viable spermatozoa with increase in sperm-count, and their decrease with time-intervals and higher proportions of vital rather than motile cells (table 1) are known.

The observed changes in activity and viability of spermatozoa 5 h after ejaculation (table 2) show that oligospermia is characterized by a decreased span of activity in addition to the usually higher proportion of spermatozoa exhibiting lower activities (as evidenced by the enlarged populations of immobile cells and relatively poor motility grades of the mobile ones), and the lowered proportion of vital cells. These behavioural patterns could be due to inherent metabolic errors possibly responsible for poor resources of intracellular energy and/or impaired availability of energy, resulting in a more rapid exhaustion of the sperm. The enhanced deterioration of the parameters examined in specimens with low sperm density is in line with our previous observations[9,12,13] suggesting a correlation between oligospermia and intracellular defects.

1 To whom reprint requests should be addressed.
2 J. Macleod, Fert. Steril. *20,* 545 (1969).
3 R.J. Lightfoot and B.J. Restall, J. Reprod. Fert. *26,* 1 (1971).
4 R. Eliasson, Andrologie *3,* 49 (1971).
5 E.S.E. Hafez, in: Human Semen and Fertility Regulation in Men, p.83. Ed. E.S.E. Hafez. The C.V. Mosby Company, St. Louis 1976.
6 D. Mortimer, J. Reprod. Fert. *51,* 99 (1977).
7 B. Fredricsson and G. Bjork, Fert. Steril. *28,* 841 (1977).
8 E.S.E. Hafez, in: Techniques of Human Andrology, p.153. Ed. E.S.E. Hafez. North-Holland, Amsterdam 1977.
9 R. Singer, M. Sagiv, M. Barnet, E. Segenreich, D. Allalouf, B. Landau and C. Servadio, Andrologia (in press).
10 A.T.K. Cockett, I.C.V. Netto, K.A. Dougherty and R.L. Urry, J. Urol. *114,* 560 (1975).
11 K.A. Dougherty, R.L. Urry and A.T.K. Cockett, J. Urol. *118,* 1008 (1977).
12 R. Singer, M. Barnet, D. Allalouf and I. Chowers, Int. J. Fert. *24,* 53 (1979).
13 R. Singer, M. Sagiv, M. Barnet, D. Allalouf, B. Landau, E. Segenreich and C. Servadio, Int. J. Fert., in press (1980).