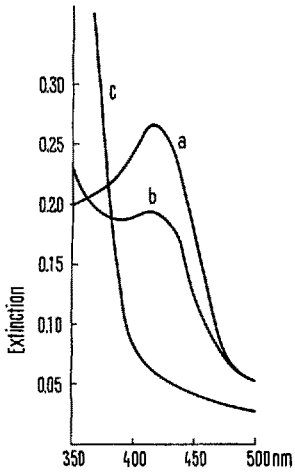
smaller amounts. There is a partial loss of the yellow sepiapterins in the eyes of both sexes, as shown in the Figure; correspondingly the probable precursor biopterin accumulates chiefly in the bodies. Visual examination of the pellets from homogenates of *se* heads has shown that in either sex the colour of the pellets relative to inhibited flies is lighter than in control flies. Resuspending the pellets in 3% HCl in methanol, freshly saturated with SO_2 gas, red hydroxanthommatin is extracted, the colour intensity of which appears to be markedly less in the inhibited flies than in the control ones[6,8].



Light-absorption curves of AEA extracts of the heads of the mutant *sepia* (10 heads/1 ml): (a) for control flies; (b) for flies grown on HPP 0.06%; (c) for control flies, the extract of which has been irradiated 24 h under UV-light. The maximum at 420 nm present in the curves (a) and (b) corresponds to that of the sepiapterins.

Riassunto. Quando ceppi dei mutanti *se* e *cl* di *D. melanogaster* sono fatti crescere su terreni contenenti l'inibitore della xantino-deidrogenasi 4-idrossipirazolo (3,4 d) pirimidina, si osserva una perdita parziale dei pigmenti dell'occhio: tale perdita riguarda non soltanto i pigmenti pteridinici rossi (drosopterine), fenomeno questo già descritto, bensì anche i pigmenti pteridinici gialli (sepiapterine) nonchè i pigmenti ommocromici dell'occhio.

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[6] Notwithstanding that the diminution of the ommochromes visually observed is a very clearcut one, we were not able to perform a quantitative determination of the pigment in the form of hydroxanthommatin according to Butenandt et al.[7], because the colour intensities obtained after extraction of the pellets with 2*N* HCl and butanol were unstable and the absorption maximum frequently shifted to the yellow.

[7] A. Butenandt, E. Biekert, H. Kübler and B. Linzen, Hoppe-Seyler's Z. physiol. Chem. *319*, 238 (1960).

[8] This research was supported by the Consiglio Nazionale delle Ricerche, Rome (Italy).

Two Different Hemodynamic Patterns Underlying Hypotension during Desynchronized Sleep[1]

We have previously reported detailed studies of arterial pressure changes during natural sleep in the cat, showing that blood pressure falls more markedly during sleep with a desynchronized electroencephalogram and rapid eye movements than during sleep with synchronized electroencephalographic patterns[2]; that the hypotensive effect of desynchronized sleep is strikingly exaggerated by sino-aortic deafferentation[2]; and that this exaggeration is caused by abolition of the buffering action of chemoceptive impulses from the carotid and aortic bodies[3].

The experiments summarized below have been devised with the aim of further clarifying the hemodynamics of sleep, with particular reference to desynchronized sleep (DS). Most of the operating, recording and statistical techniques were as previously reported[2,3]. All cats were studied in a sound-attenuating cage. Systolic and diastolic pressure was recorded throughout the wakefulness-sleep cycle from a cannulated femoral artery; electroencephalogram, cervical electromyogram, and eye movements were also monitored on a Grass P7 polygraph. Details of other procedures are mentioned below.

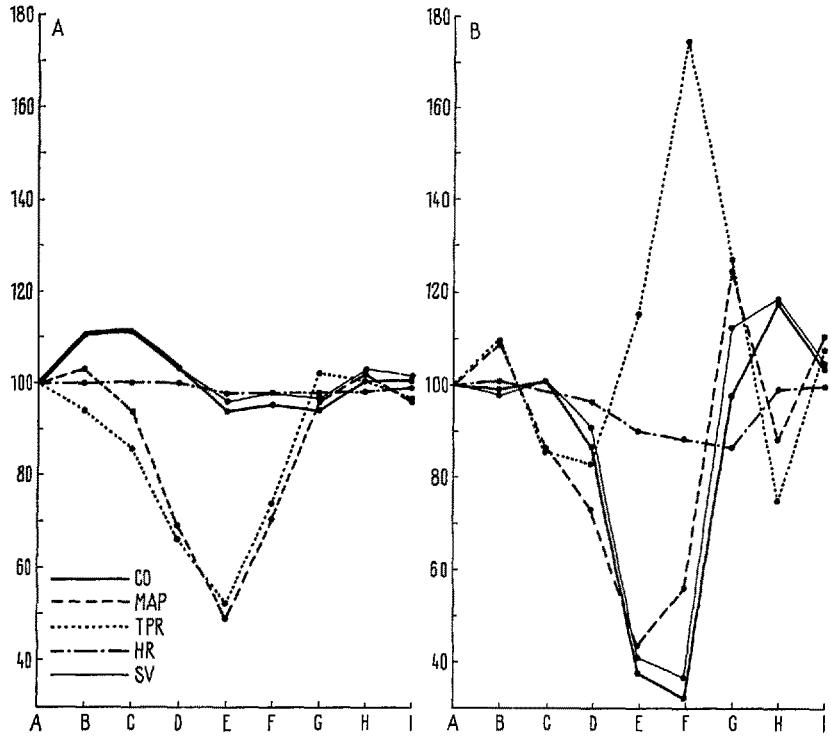
In a first group of cats, most of them with previous sino-aortic deafferentation, arterial pressure changes during sleep have been compared before and after various kinds of heart denervation (bilateral stellatectomy, bilateral cervical vagotomy, and combined bilateral stellatectomy and vagotomy). In a few cats blockade of cardioinhibitory vagal fibres was obtained by large doses (1 mg/kg i.v.) of methylatropine. Statistical analysis showed that during sleep arterial pressure undergoes changes of the same type and of corresponding size both when the heart innervation is intact and when it is destroyed. This means that the neural control of the heart is not basically involved in the blood pressure fall occurring in sleep.

These experiments, however, though implying that hypotension during sleep is mainly dependent on peripheral vascular phenomena, do not indicate whether the fall in blood pressure is basically due to a decreased vascular resistance or to a reduced venous or pulmonary return resulting in a lower cardiac output. This problem has been the object of a second series of experiments, in which, besides arterial pressure, aortic flow has been continuously monitored in sleeping cats by means of an electromagnetic flowmeter probe (Statham 4000A flowmeter, chronic K-probes) chronically implanted around the ascending aorta. The probe was calibrated with saline or blood before and after the experiment. Electronic integration of the aortic flow curve (Grass 7P10 integrator

[1] This research has been sponsored by the Air Force Office of Scientific Research, through the European Office of Aerospace Research (OAR), United States Air Force, under Grant No. AF EOAR 66-47, and by Consiglio Nazionale delle Ricerche (Gruppo Nazionale di Medicina Sperimentale). Dr. Kumazawa was a visiting investigator from Nagoya University, Nagoya, Japan, under a IBRO-Unesco fellowship.

[2] M. Guazzi and A. Zanchetti, Science *148*, 397 (1965); Archs. ital. Biol. *103*, 789 (1965).

[3] M. Guazzi, G. Baccelli and A. Zanchetti, Science *153*, 206 (1966).



Two hemodynamic patterns (A and B) underlying hypotension during desynchronized sleep (DS). CO, cardiac output; MAP, mean arterial pressure; TPR, calculated total peripheral resistance; HR, heart rate; SV, stroke volume. On the abscissas, measurements taken at, (A) synchronized sleep before DS; (B) onset of DS; (C) during DS when blood pressure starts to fall; (D) later on during DS; (E) during DS when blood pressure is lowest; (F) toward the end of DS; (G) arousal from DS; (H) after DS; (I) later after DS. On the ordinates all hemodynamic changes are expressed as percentage of measurements taken in (A). Cat with sino-aortic deafferentation: vagal activity blocked by methylatropine (1 mg/kg i.v.).

with automatic reset) yielded stroke volume short of coronary blood flow. Cardiac output was calculated from stroke volume and heart rate (recorded by Grass 7P4 cardiotachometer). Total peripheral resistances were calculated in arbitrary units by dividing mean arterial pressure (in mmHg) by cardiac output (in l/min).

During DS, besides the known changes in arterial pressure, there was also a variable decrease in cardiac output. In all episodes studied in cats with intact sino-aortic reflexes, as well as in the great majority (154 out of 178) of the episodes recorded in cats with sino-aortic deafferentation, the decrease in cardiac output was usually slight as compared with that in mean arterial pressure (Figure A). Therefore, the most typical hemodynamic pattern during DS consisted in a blood pressure fall which almost entirely, or largely resulted from a decreased peripheral resistance. Sino-aortic deafferentation simply exaggerated the pattern already occurring before abolition of the reflexes. No substantial qualitative nor quantitative change was observed after heart denervation.

A quite different hemodynamic pattern was also observed, which was equally independent of cardiac innervation. It was evident only in a small proportion of DS episodes in sino-aortic deafferented cats (24 out of 178). These were the episodes characterized, as previously reported[2], by blood pressure falls to such low levels that transient cerebral ischemia occurs. In these instances, hemodynamics at the beginning of DS followed the usual pattern, with a pressure fall mainly due to decreased peripheral resistance. Suddenly, however, arterial pressure started falling more rapidly, while cardiac output decreased with such steepness as to exceed the rate of decrease in blood pressure. Lowest values of cardiac output and highest resistance were attained when overt signs of cerebral ischemia occurred. Hemodynamics came back to normal when brain ischemia aroused the animal (Figure B).

These experiments show that hypotension during DS is independent of cardiac innervation, and usually results from a decreased peripheral resistance. This suggests vasodilatation brought about by decreased sympathetic activity, a suggestion that is in agreement with electrophysiological evidence[4]. A different hemodynamic pattern occurring in the few episodes of cerebral ischemia observed in sino-aortic deafferented cats is difficult to interpret. The paradoxical increase in calculated resistance cannot safely be taken to mean peripheral vasoconstriction, because of the possible changes in blood viscosity due to the extreme fall in cardiac output. Nor can it be said whether this second hemodynamic pattern is a simple exaggeration of the first more usual one beyond some turning point at which other reactions are added, or whether it results from different neural or non-neural mechanisms.

Riassunto. L'ipotensione presente durante il sonno desincronizzato del gatto, con riflessi seno-aortici intatti o aboliti, è dovuta soprattutto a diminuzione delle resistenze periferiche e in ben piccola parte a riduzione della gettata cardiaca. Solo negli episodi di sonno desincronizzato, osservati talora nel gatto con deafferentazione seno-aortica, in cui l'ipotensione è così profonda da produrre ischemia cerebrale, la gettata cardiaca diminuisce marcatamente e l'indice di resistenza paradossalmente aumenta.

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[4] Y. IWAMURA, Y. UCHINO, S. OZAWA and S. TORII, Proc. Japan Acad. *42*, 837 (1966); W. BAUST, personal communication.