Nystagmus Evoked by Intermittent Photic Stimulation of the Rabbit's Eye

In the course of our recent investigations it has become evident that 'central nystagmus', originally evoked in the rabbit by stimulation of the 'meso-diencephalic nystagmogenic area'[1], is in fact due to excitation of the optic pathways, e.g. the optic nerve[2] or the superior colliculus[3]. Similarly, electrical stimulation of the retina via corneal electrodes produces eye movements with the same characteristics as central nystagmus. In the rabbit, where practically complete decussation takes place in the optic chiasm, stimulation of, say, the right optic nerve and the left superior colliculus produces eye beats to the right. The direction of the movement is identical with that of optokinetic nystagmus elicited by moving an object from the lateral towards the medial border of the field of vision of the right eye[4]. Since both electrical and optokinetic excitation produce repetitive potentials along the optic pathway, one might expect that any other form of stimulation which gives rise to rhythmic discharges in the optic pathway, should equally evoke nystagmus. However, previous experiments with intermittent photic stimulation yielded negative results[2].

Systematic re-examination of the problem has now shown that movements, termed here 'flash nystagmus', can indeed be elicited by monocular flashing, if the contralateral eye is protected from light (Figure 1). The response appears only within a narrow frequency range (10–40 flashes/sec), the optimal effect being obtained at about 30/sec (Figure 2). The susceptibility of the rabbits to this stimulus is variable: about $^3/_4$ of the animals tested gave a positive reaction, but only 10% showed a strong response, such as in Figure 1, while in the majority of tests 1–5 eye beats/min were observed.

Previous failure to evoke 'flash nystagmus' must be ascribed to the insufficient number of rabbits tested and to the use of non-optimal frequencies.

Flash nystagmus has several properties in common with central nystagmus: (1) *Direction of the response.* Flashing of the left eye elicits nystagmus to the left as does electrical excitation of the left optic nerve or of the right superior colliculus. Likewise, the afternystagmus points in the same direction as the eye movements during photic stimulation. (2) *Photic inhibition.* Flash nystagmus is strongly inhibited by stationary illumination of the other eye (Table a), as was shown for central nystagmus[5]. Similar inhibition takes place with the eye exposed to the

Inhibitory effect of light on flash nystagmus

(a) *Effect of illumination of the contralateral eye.* The right eye was exposed for 60 sec to intensity I of a Strobotest No. II photo-stimulator, placed at a distance of 20 cm from the centre of the cornea. The left eye was protected by a small black hood, underneath which a 3 W bulb was fixed.

Rabbit No.	Flashes/sec	Condition of left eye	Nystagmus response
4	30	Darkness	15
		Illumination	5
17	30	Darkness	9
		Illumination	1
19	25	Darkness	40 + 2[a]
		Illumination	6

[a] These figures read as follows: 40 beats during photo-stimulation; afternystagmus 2 beats.

(b) *Effect of flash intensity.* The left eye was stimulated with 25 flashes/sec; the right eye was kept in darkness throughout the experiment. Intensity I corresponds to 10^5 lux, II to $7.5 \cdot 10^6$, and III to $1.5 \cdot 10^8$ lux.

Flash intensity	Nystagmus response
I	73 + 8
II	21 + 1
III	16

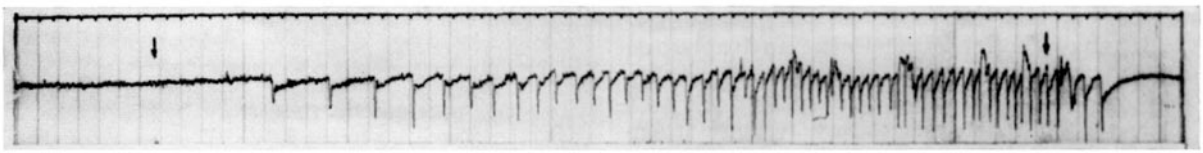

Fig. 1. Nystagmogram of flash nystagmus, elicited by 60 sec exposure of right eye to 25 flashes/sec, delivered from a Strobotest No. II photo-stimulator. Eye beats to right marked as downward excursions. Note latency of 8 sec and increasing rate of eye movements. Afternystagmus points in the same direction as eye beats during stimulation.

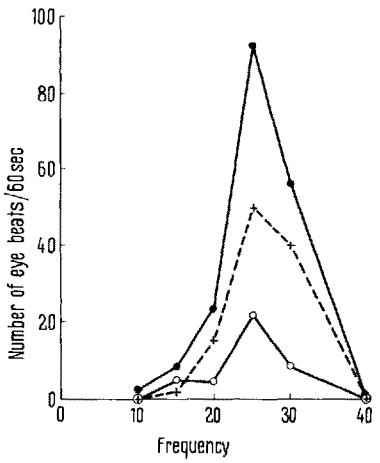

Fig. 2. Number of eye beats as function of rate of flashing +---+, control; ●——●, 90 min after intravenous administration of 1 mg/kg chlorpromazine; o——o, 180 min after the injection. Note that the optimal response occurs always at the same flash frequency.

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flashes, as can be shown by varying flash intensity (Table b). (3) *Biphasic effects of chlorpromazine.* Flash nystagmus like central nystagmus is enhanced by chlorpromazine[6] (Figure 2). Application of the drug makes it possible to evoke flash nystagmus in all animals, even if they had been refractory before. The reinforcement of the eye movements persists for several hours and is followed by a depressory phase. The period of enhancement of flash nystagmus is much longer (4–6 h) than that observed for central nystagmus.

Flash nystagmus differs from central nystagmus in the limited frequency range of stimulation (Figure 2) and in the position of the optimum, which is 40–60 c/s for excitation of the optic nerve[2]. This divergence is under closer study.

Quantitative differences are also apparent between optokinetic and flash nystagmus. The optokinetic stimulus is much more effective; all animals give a positive response and in the same animal the optokinetic reaction is much more intense than flash nystagmus.

Zusammenfassung. Monokuläre Reizung des Kaninchens durch Lichtblitze ruft einen Nystagmus hervor, dessen Richtung identisch ist mit der Richtung der Augenbewegungen, die durch elektrische Stimulierung des ipsilateralen Nervus opticus verursacht werden. Die anhaltende Beleuchtung des kontralateralen Auges hemmt die Nystagmusreaktion.

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PRO EXPERIMENTIS

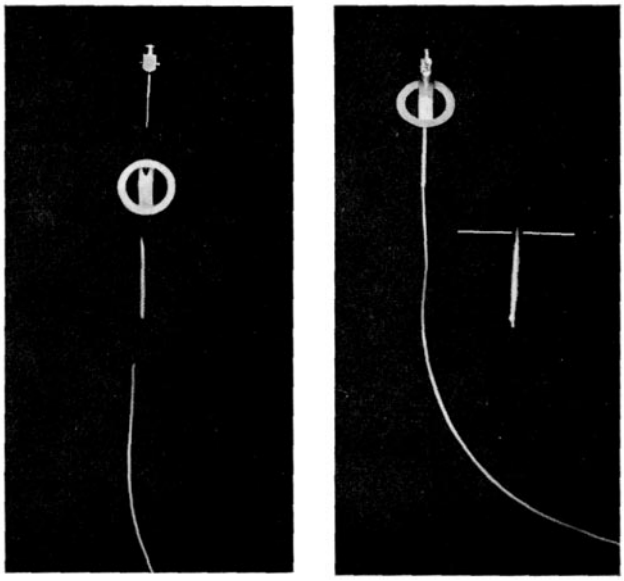
A Technique for Repetitive Long-Term Measurement of Aortic Pressure and Cardiac Output in the Unanaesthetized Dog Using an Implanted Catheter

Techniques for studying circulatory dynamics in unanaesthetized animals are gaining increasing interest due to the realization that anaesthetics cause considerable changes in cardiovascular function and may greatly influence reactions to drugs. In the course of cardiovascular studies in unanaesthetized dogs we have developed a new method, allowing continuous or repeated recording of aortic pressure pulses and cardiac output over periods of many months. Because visitors to our laboratories have shown considerable interest in this method we present its details in this report.

Principle of the method. A Teflon® catheter is inserted into the abdominal aorta from the femoral artery. The catheter is connected to a syringe needle with a stopcock-type valve embedded in a polyamide plate which is held in place by subfascial implantation.

Assembly of catheter and materials used. The Teflon® catheter (internal diameter 1.0 mm, external diameter 1.5 mm) is connected to a syringe needle with a valve of the stopcock type. The needle, which is made of stainless steel, is embedded and fixed by Araldite® in a polyamide plate consisting of a flat ring and a crossbar; it passes through the crossbar at an angle of approximately 25° to the plane of the ring itself. In order to prevent kinking the first 4 cm of the catheter beyond the ring are reinforced by a second Teflon® catheter (internal diameter 1.6 mm) with a spiralled surface contour. Details are given in Figures 1 and 2.

Operating procedure. Mongrel dogs weighing 12–20 kg are anaesthetized with pentobarbital (30 mg/kg i.v.). Under strictly aseptic conditions an incision of about 3 cm is made over and parallel to the distal part of the femoral artery. After splitting the fascia the artery is dissected free from surrounding tissue and ligated. The Teflon® catheter is then introduced into the femoral artery and threaded up to the point at which the ring itself prevents further progress. Thus the part of the needle extending beyond the crossbar and protruding into the Teflon® catheter lies within the artery. This proximal part of the catheter is anchored to the artery by ligatures which themselves are fixed to the polyamide plate. The plate is



a b

Fig. 1. (a) Single parts of the catheter (before assembly). From top to bottom: syringe needle with valve of the stopcock-type, polyamide ring with crossbar, Teflon® catheter with spiraled surface (reinforcing catheter), aortic Teflon® catheter. (b) Catheter assembled and ready for implantation. At the right, key to close and open the valve.