NYSTAGMUS AND ATAXIA INDUCED BY BLOCKADE OF AFFERENT INFORMATION FROM THE NECK

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Longet (1845) described cervical ataxia in animals but it was not until 1939 that Biemond (1939) wrote for the first time about cervical nystagmus. We have found that the cause of these phenomena is stimulation or blockade of the flow of afferent information from cervical joint and muscle receptors. Ataxia and nystagmus due to changes in the vertebral-basilar circulation are quite a different entity. First we will describe what happens in normal rabbits, cats, monkeys and men when we diminish the flow of afferent activity by injections of procaine or lidocaine hydrochloride in the neck. Subsequently you will see the results if prior to these injections lesions have been made in the vestibular apparatus, the cerebellum, the spinal cord or the cervical dorsal roots.

Injection of 1.5 to 2 cc 2% procaine hydrochloride on one side in the neck of normal rabbits resulted in falling and rolling to the side of the injection, lateropulsion and hemihypotonia lasting up to 3 hours (Biemond & de Jong 1969). The concomitant nystagmus was of a slightly shorter duration. In

Fig. 1. (A) Monkey shielding right half of body after right cervical block with 2% lidocaine. (B) Deviation of the body axis toward the side of cervical block on the right.
healthy cats injections led in 26 cases to nystagmus with a duration of 4 to 60 minutes, maximum frequency 3 per second, but in 60 cases to hypotonia and ataxia (de Jong et al., 1977).

The ataxia was even stronger in normal monkeys (fig. 1). They propped the more ataxic half of the body against the walls of the cage and steadied themselves by grasping the overhead bars with the better – coordinated hand. The nystagmus lasted only 1 to 5 minutes and could be seen on the registration in 17 of the 40 times that neck injections were given (fig. 2). Optokinetic nystagmus, after nystagmus and caloric nystagmus were hardly influenced by these neck injections.

![Diagram](image)

**Fig. 2.** Nystagmus induced by unilateral neck injection in a monkey. Each trace is a horizontal EOG. (A) Eye movements before injection in light and darkness. After left neck injection (B) there was nystagmus to the left. In another monkey, nystagmus to the left was induced after a right neck injection (C) and to the right after a left neck injection (D). Calibration of 10 degrees for each of the traces is shown next to C.

In two men neck injections led to a strong sensation that they were drawn towards the injected side ‘Like a bar of iron by a strong magnet’. There was a positive plumbline Romberg sign, with deviation and past-pointing toward the side of the injection, but no dysarthria, intention tremor, dysmetria, dysdiadochokinesis or disturbance in kinesthesia. When supine they had the feeling that the couch was slowly toppling over toward the injected side. In an hour these symptoms gradually disappeared. Neither with Frenzel glasses nor on the registration could nystagmus be seen. The men were, however, injected at the level of the vertebrae C₃ - C₄. In subsequent experiments on monkeys, nystagmus was found to occur only if injections were made at the
occipito-cervical junction. In the rabbit no cervical nystagmus could be elicited by procaine injections when both labyrinths were destroyed or when all cervical nerves and roots from C₁ to C₄ were cut (Biemond & de Jong 1969). Interruption of both vertebral neuro-vascular bundles, however, did not abolish cervical nystagmus in the rabbit or the cat. After an unilateral labyrinthectomy procaine injections in either side of the neck of rabbits and cats elicited neck-afferent nystagmus. This points to a bilateral mediation of activity from the neck, responsible for the nystagmus. When in these animals with one intact labyrinth the cervical nerves were cut on one side, a severe ataxia and a long-lasting nystagmus were seen, lasting up to 6 weeks in rabbits and 4 days in cats (fig. 3). In cats the maximum frequency of this nystagmus varied from 0.4 to 1.5 cps, the maximum velocity of the slow phase from 5 to 40 degrees per second and the average duration of the slow phase of 30 consecutive beats from 0.2 to 1 second.

Fig. 3. Cervical nystagmus after section of left dorsal roots C₁ and in a cat 9 months after a rightsided labyrinthectomy. Trace A right vertical, B right horizontal, C left vertical, D left horizontal EOG.
In rabbits, cervical nystagmus could still be elicited after cerebellectomy (4). This was also true for cats that had undergone a hemisection at the C1 level of the dorsal spinal cord (fig. 4) or a midline myelotomy between C1 and C3 (fig. 4). The hemisection of the dorsal cord produced nystagmus, lasting up to 8 weeks.

These lesion studies suggest that the cervico-oculomotor pathway runs at least partially ipsilateral through the ventral portions of the spinal cord and through the vestibular system.

![Fig. 4. Nystagmus induced by cervical injection in a cat I. after dorsal hemisection of the spinal cord and II. after midline myelotomy in another cat. Legend: see Fig. 3.](image)

When we look at these data we see that unilateral neck anaesthesia has a predominantly ipsilateral effect and is followed by ataxia, past-pointing, hypotonia and unwillingness to move. In man, there is no intention tremor, dysarthria, dysmetria of finger movements or impaired limb kinesthesia. Neck afferent nystagmus was stronger and lasted longer in the rabbit, was weaker in the cat and least in the monkey.

Tonic cervico ocular reflexes are stronger in the rabbit and weaker in the monkey and man; this is probably the explanation for the quantitative differences in cervical nystagmus. In monkeys, as in cats, this nystagmus was predominantly horizontal and present in the upright position. In the rabbit horizontal and vertical nystagmus were induced by dorsal root section, both in the upright position as on positional testing.

Dichgans (1973) found that cervico-ocular pathways become much more potent in monkeys after labyrinthectomy. In such cases short compensatory eye movements during head movements were based primarily on sensations that originated in the cervical muscle and joint receptors.

This conversion of a latent to an overt mechanism after labyrinthectomy implies that in some circumstances cervical abnormalities may be responsible
for causing disorders of equilibrium and possibly nystagmus.

The recurrence of nystagmus and ataxia in the labyrinthectomized cat and rabbit after unilateral dorsal root section seems relevant in this regard.

In conclusion we can say that neck afferent nystagmus and ataxia together with dizziness and vertigo can arise from disorders affecting the afferent flow of impulses from deep tissues of the neck. Since these impulses are mediated through the vestibular nuclei, it is not surprising that, to date, the clinical differential diagnosis between vestibular and neck afferent vertigo and ataxia can not be made with certainty.

REFERENCES


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Question by Krejčová (Prague): The finding of Dr. de Jong is interesting from the clinical point of view, because the infiltration of neck muscles with anaesthetics is often performed in patients suffering from a cervical syndrome. This treatment usually brings benefit to most of them, but some complain of slight dizziness and weakness.

This used to be explained by transient decrease of blood pressure following the infiltration of anaesthetics. De Jong's paper presents another explanation supported by the results of animal experiments.

Answer by De Jong: I agree, but I have never heard up till now that the procaine infiltrations were used as a routine clinical procedure.