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Alternating Dissociated Nystagmus with Palatal Myoclonus
— A Case Report —

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An extraordinary eye movement was seen in a vegetative patient. His eyeballs were exotropic in the primary position and showed dissociated nystagmus which appeared alternately in each eye every few seconds. He also had palatal myoclonus quite asynchronous with the nystagmus. To our knowledge, there has been no such nystagmus documented in the literature. We report the new nystagmus with his EOG and brain MRI.

Key Words : *Nystagmus, Dissociated*

INTRODUCTION

Many types of nystagmus have been described, and occasionally their recognition may help to localize the pathologic site. But for the most part, the exact mechanism of those nystagmus is still unknown. Because this case had widespread infarction in the brainstem and occipital cortex on MRI, the nystagmus had no precise localizing value. However, the nystagmus was so characteristic that a person can never forget it once he has seen a case, and so we report the case and refer to it as "alternating dissociated nystagmus".

CASE REPORT

A 60-year-old vegetative man was transferred to our hospital from another hospital where he had been managed for an acute state of sudden altered consciousness. He had been suffering from diabetes mellitus and hypertension for 10 and 5 years, respectively. Prior to this attack, he had experienced several episodes of mouth angle deviation and right-sided weakness, from which he recovered after a few weeks with some residual weakness. At the end of August 1989 he complained of dizziness and visual dimness, and on September 2nd, he was found in a state of stupor and brought to the hospital where he was treated under the diagnosis of brainstem infarct.

His condition was worsened to coma and quadriplegia without brainstem reflexes.

After 2 months of hospitalization, he was transferred to our hospital in a vegetative state. On examination at transfer, he was unresponsive to verbal or visual stimuli except some extension of his right limbs. His eyes were closed bilaterally, and showed 3mm unreactive isocoric pupils. Eyeballs revealed bilateral exotropia and internuclear ophthalmoplegia, with spontaneous jerky abducting nystagmus on the right side. Oculocephalic and corneal reflexes were absent. His respiration was irregular with intermittent apnea, and he showed hypoactive DTRs with bilateral extensor plantar reflexes. He did not show a definite sleep-wakefulness cycle. Two months after the transfer, without any

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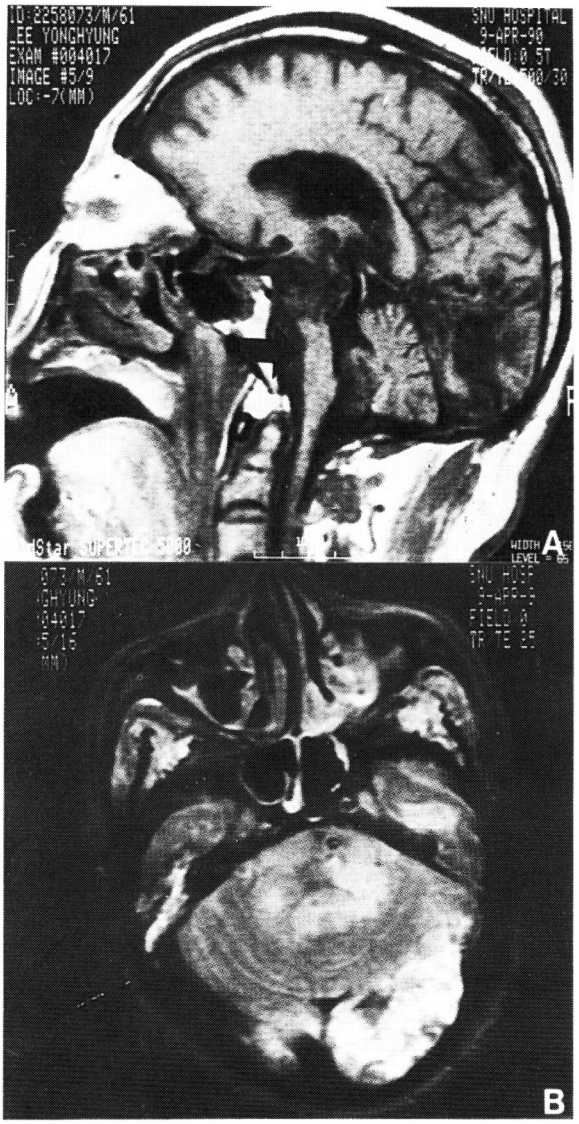

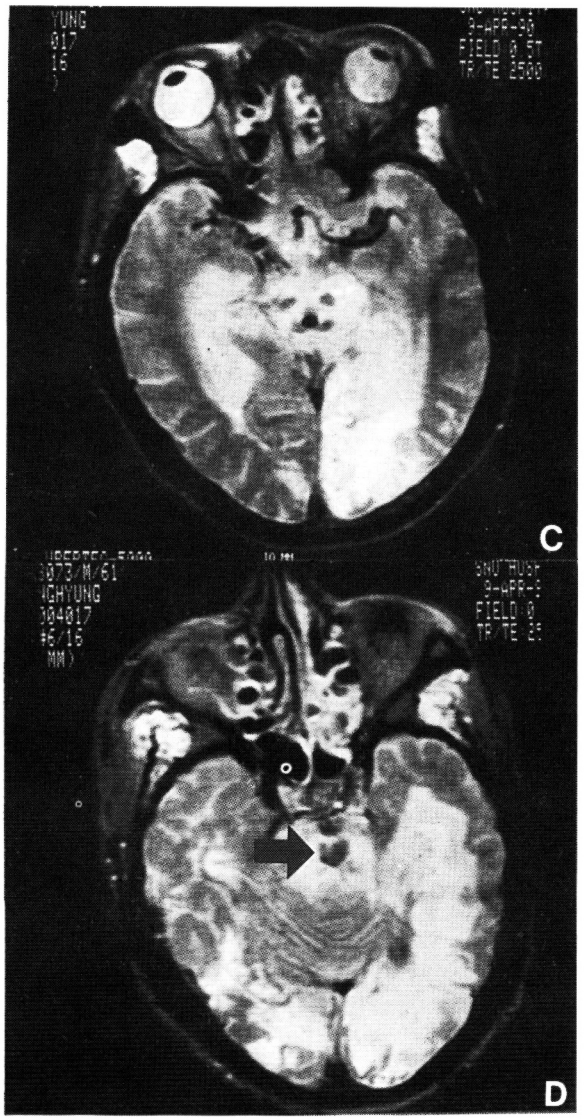

Fig. 1. Brain MR imaging of the patient(A, B, C, and D)
A : T1-weighted, mid-sagittal view : Severe destructive change of midbrain due to ischemic infarct and atrophic pons(arrow).
B : T2-wighted axial view, medullary level : Bilateral cerebellar infarction.
C : T2-weighted axial view, midbrain level : Massive infarction in the midbrain sparing red nucleus and in both occiputs. No visible basilar artery flow signal void.
D : T2-weighted axial view, pons level : Some portion of anterior tegmentum seems to be intact(arrow).

remarkable neurologic changes, we found that jerky nystagmus developed alternately in each eye at every one to a few seconds with a brief null period ; caloric test was negative. At that time we also found that palatal myoclonus continued the whole day without cessation. Brain MRIs, done twice at the transfer and 3 months later, showed multiple areas of infarction in the pons, midbrain, cerebellum, and temporo-occipital lobes without remarkable interval change(Fig. 1). The electro-oculography(EOG), simultaneously with EMG on the soft palate, EEG, and EKG, were recorded, which showed the jerky nys-

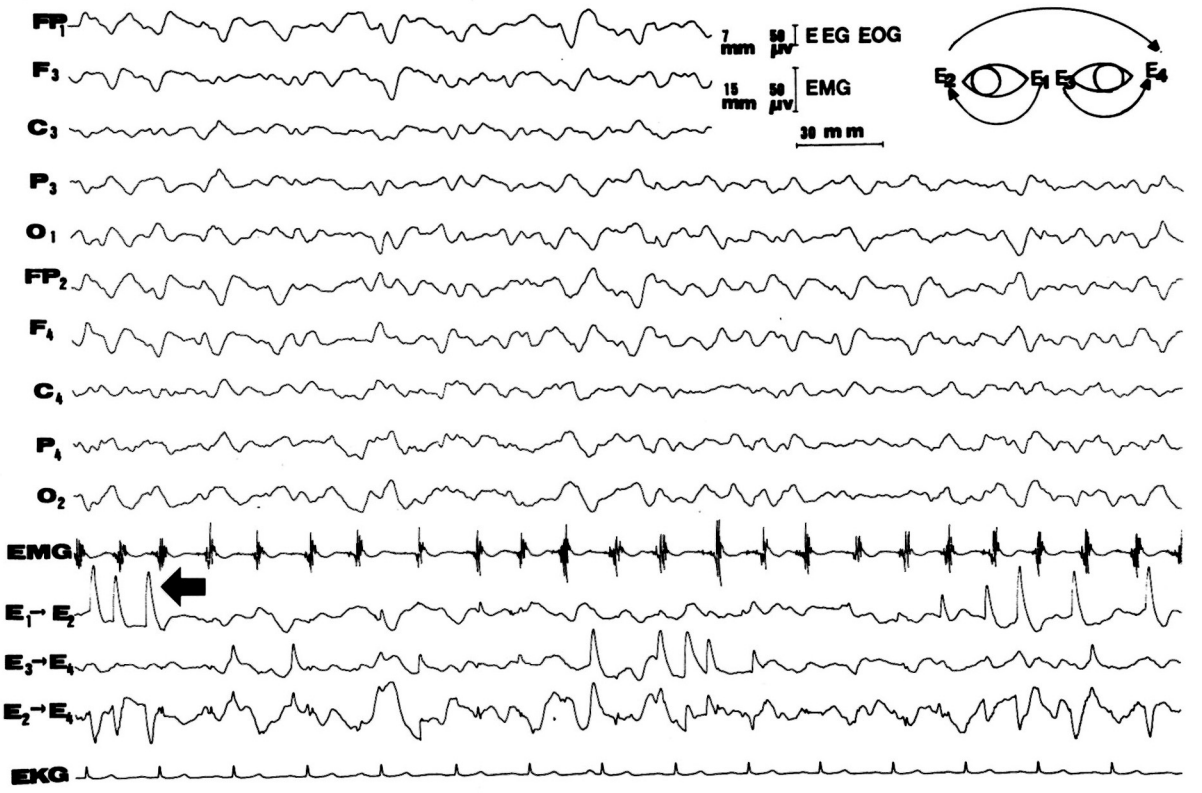

Fig. 2. EOG with EEG, and EKG

EOG : When right eyeball developes nystagmus(arrow) to the right side, no nystagmus is seen in the left side and vice versa. The alternating pattern has no regularity. The individual waveform shows the slow phase of an exponentially decaying course.

EMG : Palatal myoclonus is clearly seen with a relatively regular frequency of about 2.5/sec.

EEG : Referential electrodes with average potential. Generalized slow wave without spike or other epileptiform discharge

* Paper speed is 30mm/sec. Time constant of EOG is 0.1. Time constant of EMG is 0.001. This recording was made from a DC-coupled device.

tagmus alternately occuring in each eye without regularity(Fig. 2). The EMG revealed a relatively regular rhythmic contraction of the soft palate muscle at about 2.5Hz intervals, which was quite asynchronous with the nystagmus on the EOG. The EEG showed generalized cerebral dysfunction without any spike or sharp epileptiform discharge. We also made video recordings of the characteristic ocular nystagmus and palatal myoclonus.

DISCUSSION

It is generally believed that comatose patients do not evidence jerky nystagmus which may involve cortical visual processing (Leigh and Zee, 1983), but contrary to this belief, our patient apparently developed jerky nystagmus as manifested in the EOG. Though the mechanism is still obscure, we postulated some possible explanations regarding the origin of the nystagmus.

This patient had a bilateral anterior internuclear ophthalmoplegia(INO) with exotropia, which could have been due to bilateral medial rectus subnuclei dysfunction, and the term "Webino"(Wall-Eyed Bilateral INO) syndrome, i. e., bilateral INO associated with exotropia, might apply to this case(Inocencio and Ball-

ecer, 1985). The nystagmus in our case also had an abducting and dissociated pattern, similar to the nystagmus in INO. In addition, the slow phases of the nystagmus had a waveform of an exponentially decaying course as seen in the EOG , which reflects an unsustained eye position signal caused by an impaired neural integrator and can be shown in a gaze-evoked nystagmus including that of INO(Baloh et al., 1987). The abducting dissociated nystagmus in INO has been relatively well studied, but its origin still has not been fully understood(Bender and Weinstein, 1939 ; Stroud et al., 1974 ; Pola and Robinson, 1976 ; Zee et al., 1987).

From the point of view that our patient had a bilateral INO and that the pattern of the nystagmus in this case had many similarities with the nystagmus seen in the INO, the generation of this "alternating dissociated nystagmus" might be attributed to the INO. But in all nystagmus studied in INO, the patients were conscious and could focus their eyes on command. However, our patient was comatose. Furthermore, the most striking feature in this case was the alternating nature of the nystagmus. So it might be concluded that this patient spontaneously focused his eyes to the right and then to the left, in other words, a "ping-pong" gaze(Fisher, 1967). Another explanation is the possibility of roving eye movements in this patient considering that the alternating pattern was not regular. Each eye sign mentioned above is postulated to be an effect of a brainstem pacemaker when cortical influences are released due to bilateral cerebral dysfunction, though there are still varying arguments about the origin(Senelick, 1976 ; Stewart et al., 1979 ; Larmande et al., 1987). Our patient had a widespread brainstem infarct, and the main lesion was thought to be the midbrain. In the MRI finding, a small portion of the pontine tegmentum appeared relatively intact, and that portion might have been the generator of the alternating movement.

There have been some reports of palatal myoclonus combined with ocular motor disorder just as observed in this case(Bender et al., 1952 ; Chokroverty and Barron, 1969 ; Tahmoush et al., 1972 ; Jacobs and Bender, 1976). But in other cases the ocular motor abnormality had been synchronous with the palatal myoclonus, and the nystagmus was usually pendular so the mechanism of the nystagmus in this case should be considered separately from that of the palatal myoclonus, which is now believed to be the result of dentato-olivary pathway interruption (Lapresle and hamida, 1970 ; Koeppen et al., 1980) as shown in the MRI finding in this case.

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