

Ocular flutter, an exceptional complication of amphetamine misuse

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Abstract

We present the first case of ocular flutter related with amphetamine intoxication and discuss the physiopathology of this interesting syndrome and the biochemical differences between OF and OC.

A-15-years-old Caucasian male with not previous medical history experienced a rapid onset of dizziness. On neurological admission the patient was nervous, there were no cognitive deficit. Clinical eye movements examination revealed frequent horizontal and symmetric involuntary burst of large to-and fro. There was a bilateral pupil mydriasis but no motor or sensory deficit, no tremor and no limb ataxia and deep tendon reflexes were all presents. Blood pressure was 145/90 mm Hg and pulse 105 beats/min CT performed on admission was normal. CSF contained 0 lymphocytes/mm³, 45 mg/dl and normal glucose levels. Urine drugs analysis was positive for amphetamine. All symptoms progressively resolved within 6 hours. An MRI performed was normal. On follow up the patient remained asymptomatic six months later.

Amphetamine produces a delayed and sustained increase in glutamate levels (6, 7). The amphetamine increase will induce an stimulation in common oculomotor integrator and in IPBN, and secondary to the IPBN and OP cells inhibition. The result of these neurotransmitters changes is a burst of involuntary saccadic movement. The horizontal predominance of burst movement could be explained because horizontal IPBN neuron seems to be glyceric and vertical-IPBN neurons gabaergic. Although al lesion of OPN could produce OF, a dysfunction in other neuron of the saccadic system could explain this syndrome. Amphetamine intoxication should be consider in the differential diagnosis of OF.

Keywords: Ocular Flutter, Pathophysiology, amphetamine

Introduction

Ocular flutter (OF) is one of the two types of eye movement abnormalities lack the intersaccadic interval [1]. Typically presents burst of horizontal movements that use to have quite small amplitude and very high frequency. In the other hand opsoclonus (OC) presents multidirectional eye movements [1,2]. These types of eye movements have been described with different situations as paraneoplastic, postinfection, hyperosmolar coma, toxic chemicals and drugs intoxication [1-4]. We present

the first case of ocular flutter related with amphetamine intoxication and discuss the physiopathology of this interesting syndrome and the biochemical differences between OF and OC.

Clinical case

A-15-years-old Caucasian male with not previous medical history experienced a rapid onset of dizziness and blurred vision while he was playing soccer. There were no other complaints

such as weakness, headache or jumping vision. There were not familiar or personal past medical history of migraine epilepsy or other neurological diseases. On neurological admission the patient was nervous, there were no cognitive deficit. Clinical eye movements examination revealed frequent horizontal and symmetric involuntary conjugated burst of large to-and fro. There was a bilateral pupil mydriasis but no motor or sensory deficit, no tremor and no limb ataxia and deep tendon reflexes were all presents. Blood pressure was 145/90 mm Hg and pulse 105 beats/min. An electrocardiogram revealed a supra-ventricular tachycardia. Blood count and routine biochemistry were normal. CT performed on admission was normal. CSF contained 0 lymphocytes/mm³, 45 mg/dl and normal glucose levels. Urine drugs analysis was positive for amphetamine. All symptoms progressively resolved within 6 hours. An MRI performed 48 hours after admission was normal. On follow up the patient remained asymptomatic six months later.

Discussion

The horizontal to-and-fro saccadic oscillations observed in this patient correspond to the definition of ocular flutter. In our

opinion the most interesting aspects of this case are: 1) that amphetamine could induce OF and 2) that the relationship between OF and amphetamine should have biochemical and pathophysiological implications.

OF is a burst of involuntary saccadic horizontal oscillations without an intersaccadic interval [1, 2]. According to physiological models (fig 1) excitatory premotor burst neurons (EPBN) are inhibit by omnipause (OPN) neurons secreting in the synaptic cleft glycine; on the other hand, EPBN stimulated the inhibitory premotor burst neurons (IPBN) using glutamate and finally this neurons inhibit OPN [5]. Amphetamine produces a delayed and sustained increase in glutamate levels [6, 7]. The amphetamine increase will induce an stimulation in common oculomotor integrator and in IPBN, and secondary to the IPBN and OP cells inhibition. The result of these neurotransmitters changes is a burst of involuntary saccadic movement.

On the other hand ¿why are these burst movement in horizontal directions? This interesting question could be explained because horizontal IPBN neuron seems to be gycergic and vertical-IPBN neurons gabaergic [8, 9]. This neurotransmitter difference could explain the question.

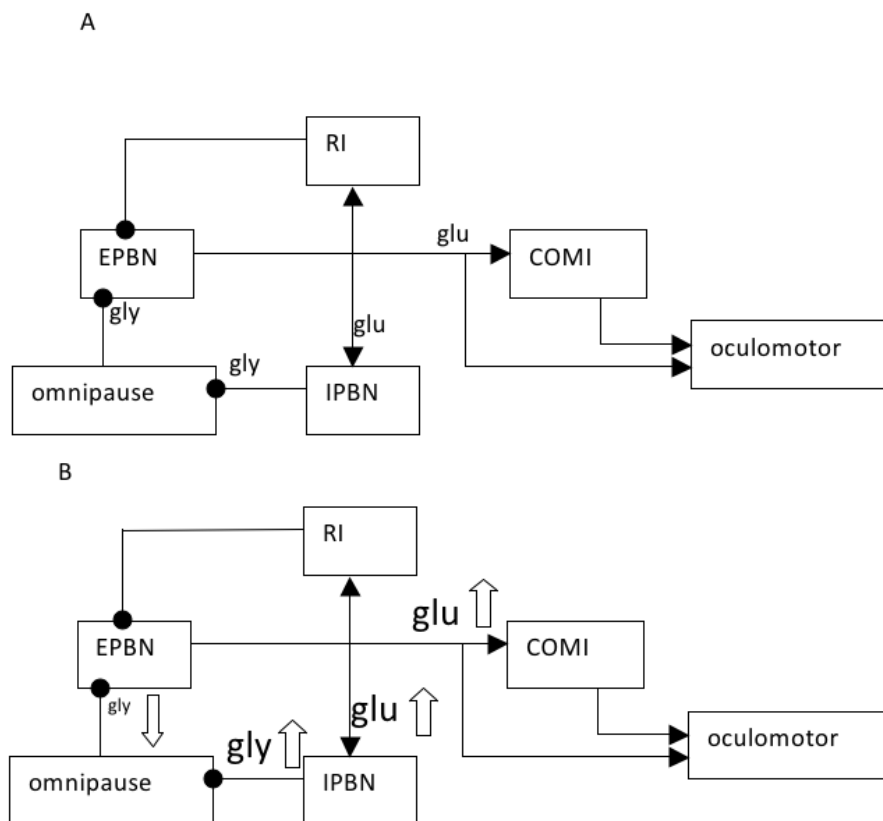


FIGURE 1A. Model of saccadic movement (modified from Ramat 2007 reference 5). 1B effects of amphetamine in saccadic ocular movement system. COMI common oculomotor integrator, EPB excitatory premotor burst neurons, IPBM inhibitory premotor burst neurons, RI re-settable integrator. Gly: glycine, Glu: glutamate

Although a lesion of OPN could produce OF, a dysfunction in other neuron of the saccadic system could explain this syndrome. The horizontal predominance of burst movement could be explained because of neurotransmitter differences in horizontal and vertical IPBN neurons. Amphetamine intoxication should be considered in the differential diagnosis of OF.

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