Abstract. We hypothesized that smooth-pursuit eye movement is related to higher brain functions and that its impairment (iSPEM) could be useful in diagnosing neurological dysfunctions. We prospectively examined 305 patients and studied the relations between iSPEM and five items that reflect intellectual and/or frontal lobe function (dementia, sucking, snouting, hand grasping, elbow flexion response). We divided these patients into four subgroups: group A, patients with cerebellar ataxia as the presenting manifestation and with main lesions in the cerebellum; group B, patients with main lesions in brain regions other than the cerebellum; group C, patients with main lesions in the spinal cord, peripheral nerves, and muscles; and group D, patients with non-organic functional disorders, such as paroxysmal attacks and physical pain. Consequently, iSPEM was significantly (p<0.01) related to impairment in intellectual and frontal lobe functions in patients with cerebral lesions, and it also can be regarded as being equivalent to primitive reflexes.

In patients with intellectual or frontal lobe dysfunction, handling (rapid and slow manipulation) of the forearm during daily neurological observations has been found to induce a spontaneous, involuntary, and gradual flexion of the elbow, which we termed as ‘elbow flexion response’ (EFR), speculating that it could be used as another primitive reflex for diagnosing neurological dysfunction (1). On the basis of clinical observations, we speculated that impairment in smooth-pursuit eye movement (iSPEM) is similarly related to higher brain function and considered that it may also be similar to the primitive reflexes. Here, we tested this hypothesis by prospectively studying the patients who participated in our daily rehabilitation program to identify the relationship between iSPEM and other items reflecting intellectual and/or frontal lobe function.

Patients and Methods

We studied 305 patients who were admitted to the Department of Neurology, Sapporo City General Hospital, and completed the routine rehabilitation program between April 2004 and March 2007. We divided these patients into four subgroups according to the presence and location of organic neurological lesions: group A comprised 20 patients who presented with cerebellar ataxia with main lesions in the cerebellum; group B comprised 159 patients with main lesions in brain regions other than the cerebellum; group C comprised 58 patients with main lesions in the spinal cord, peripheral nerves, and muscles; and group D comprised patients with non-organic functional dysfunction, including paroxysmal attacks and physical pain (Table I). The profile of each subgroup is summarized in Table II.

As registered neurologists, we evaluated SPEM in routine clinical settings for neurological disease. The examiner sat in front of the patient and smoothly moved a finger horizontally and vertically; the patient was asked to gaze at and closely follow the examiner’s finger. On the basis of our clinical experience, we judged whether the patient’s eye movement was smooth [iSPEM (–)] or defective [iSPEM (+)]. If smooth pursuit was saccadic, we judged it to be defective. A registered occupational therapist judged the patient’s intellectual ability or the presence or absence of dementia according to the revised Hasegawa dementia scale (HDS-R; dementia (+): score ≤20; dementia (–): score >20) (2). The patients were administered a battery of four tests for evaluating frontal lobe function: three for evaluating primitive reflexes (sucking, snout, and hand grasp) and one for evaluating the elbow flexion response (EFR); the results of these tests were classified as ‘positive’ or ‘negative’ on the basis of our clinical experience. We then tested the relationship between iSPEM and the five items (intellectual impairment, three primitive reflexes, and EFR) by using the Fisher’s exact probability test (3).

Results

The proportion of patients in each subgroup who tested positive for iSPEM and dementia and exhibited the three primitive reflexes (sucking, snout, and hand grasp) and EFR...
In this table, significant relations between iSPEM and the five items are denoted by asterisks. In groups A and C, we did not observe any significant relations between iSPEM and any of the five items; however, in group B, we observed significantly strong relations between iSPEM and all five items. In group D, we observed significant relations between iSPEM and four out of the five items, namely all items other than the snout reflex.

Discussion

The mechanism underlying smooth-pursuit eye movement is still being investigated, but research suggests that the frontal lobe plays an important role (4-6). iSPEM is caused by damage to area 7 (which is situated in the posterior parietal lobe) (7, 8) or the middle temporal visual area (whose presence in humans has not yet been confirmed) (9-11), which sends fibers to the frontal eye motor area (5, 12, 13). In the efferent pathway, the cerebellar dentate nucleus sends fibers via the superior cerebellar peduncle to the contralateral thalamus and an area adjacent to the eye motor area; therefore, cerebellar lesions are also reported to cause iSPEM (14-16).

Because SPEM should have been strongly influenced by cerebellar dysfunction in group A, the influence of the frontal lobe or diffuse brain function will be relatively weak in this group. This is why we distinguished cerebellar lesions (group A) from other brain lesions (group B). Consistent with our assumption, the results for group A did not show any significant relations between iSPEM and the five items.
Because these items represent involuntary and spontaneous movements of the peripheral organs (eyes, face, and hands), they are more directly controlled by the functions of the muscles, peripheral nerves, and spinal cord, all of which mediate the control by the brain. Therefore, in group C, the association of iSPEM with the five items, which are originally related to the control by the brain, would be absent, and consequently iSPEM and these items would be independent of each other. This finding may also explain the absence of a significant relation between iSPEM and these items in group C. In group D, where the dysfunction was not organic but functional, iSPEM shows significant relations with items other than the snout reflex, although the level of significance was weaker than that observed in group B. As no significant organic damage was observed in group D, brain dysfunction due to aging may be the strongest factor causing iSPEM, and such dysfunction may have also influenced the five items studied and contributed to the significant relations between iSPEM and four of the five items.

As mentioned above, iSPEM was related to impairment in intellectual and frontal lobe function, except in the patients with cerebellar lesions and neurological lesions distal to the brain. Therefore, iSPEM may also be regarded as a primitive reflex like EFR. Smooth-pursuit eye movement is an examination that can be easily carried out in routine clinical settings; therefore, like EFR, iSPEM can be used for identifying brain dysfunction. We also propose that all clinicians should perform this maneuver during routine neurological examination.

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References