TRANSIENT RELEASE REFLEXES IN CATATONIC SCHIZOPHRENIA

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This case report describes the presence of grasp reflex and palmomental reflex as a state dependent phenomenon in a 23 year old patient with catatonic schizophrenia. A transitory disturbance of frontal lobe function is proposed as the probable mechanism. The need to study the release reflexes as an effort to delineate a neurological "sub group" of schizophrenia is suggested.

Key words: transient release reflex, frontal lobe, catatonia.

INTRODUCTION

Hughling Jackson (Anonymous, 1987) introduced the concept of levels in the nervous system associated with loss of recently evolved function in progressive brain diseases, so that "primitive reflexes" (release reflexes) present in infants but absent in normal adults might reappear in old age or in cerebral diseases. Primitive reflexes have been traditionally studied as a part of soft neurological signs in schizophrenia which has been variously described as fleeting, transient or non-localizing (Buchanan & Heinrichs, 1989).

The presence of grasp reflex (GR) and palmomental reflex (PMR) are thought to indicate diffuse cerebral pathology (Tweedy et al, 1982) and more specifically of frontal lobe diseases (Swash & Mason, 1984). In most conditions the reflex is persistent. Many neurologists believe that in certain reversible dementias the release reflexes may be state dependant (Lohr, 1985).

The presence of release reflexes in schizophrenia have been considered to be a marker for cognitive deficits as well as a diffuse cerebral dysfunction of uncertain origin (Youssef & Waddington, 1988; Keshavan & Yeragami, 1987; Whittle & Douglas-Miller, 1987). However, Nizamie et al (1989) opine that the degree of brain damage in order to release these reflexes is lacking in schizophrenia, whereas Lohr (1985) had reported a case of catatonic schizophrenia in which transient grasp reflexes were present only in the catatonic stage. He suggested that there might occur a transitory frontal lobe dysfunction in schizophrenia, which could account for this phenomenon.

We herein report a patient with catatonic schizophrenia who exhibited both grasp as well as palmomental reflexes while he was in the catatonic stage. These reflexes disappeared completely after recovery.

CASE REPORT

Mr. A, a 23 year old unmarried male college student with no past or family history of any psychiatric illness presented with a three month history of remaining aloof, smiling and laughing to self, hearing voices and alleging that people were talking about him. He had stopped talking and eating for about fifteen days prior to admission, and maintained a stopped posture most of the time. There was no history of any nervous system infection, seizure disorder or any alcohol or drug abuse in the patient.

The patient was admitted in the ward where he remained completely mute and exhibited passive negativism. He maintained odd postures with minimal bodily movements. He was diagnosed to have catatonic schizophrenia and all laboratory investigation was within normal limits. Patient was put on parenteral nutrition due to an inability to maintain adequate oral intake. Neurological examination was within normal limits (detailed sensory and higher cognitive function was not done as he was not fully co-operative) except for the presence of bilateral grasp and palmomental reflexes; no other release reflex was elicited.

He was treated with intramuscular trifluperine 4mg daily and Electroconvulsive therapy (bilateral sine wave unmodified) thrice weekly. Release reflexes were assessed daily. The grasp and palmomental reflexers persisted for four days within which he received two sessions of electroconvulsive therapy. He showed remarkable improvement in his catatonic symptoms. From the fifth day onwards, no release reflex was present and he spoke relevantly and coherently. He gave a retrospective account of auditory hallucinations and ideas of reference. He remained in the hospital for another four weeks and was completely symptom free at the time of discharge. He has been keeping well on subsequent follow up.
DISCUSSION

Primitive reflexes are present in infants but are usually lost in the process of unhindered neuronal maturation and myelination; these have been recognized to reappear with senescence or brain diseases. Grasp reflexes and palmomental reflexes have been traditionally studied as a part of soft neurological signs in schizophrenia (Buchanan & Heinrichs, 1989). Though a significant proportion of normal persons are found to have palmomental reflexes, none have been found to have a grasp reflex and it is more specific for an organic etiology (Keshavan et al., 1979). In our patient, both these reflexes were present only as long as the catatonic symptoms persisted and these transient reflexes had a state dependant quality.

It may be possible to speculate that a transitory frontal lobe disturbance may be simultaneously operating in the catatonic stage in schizophrenia (Lohr, 1985). Moreover, catatonic symptoms have also been reported in frontal lobe dysfunction (Ruff & Russakoff, 1980; Cummings, 1985). This “transitory frontal lobe disturbance” may be further studied by functional imaging techniques. Keshavan & Yeragami (1987) suggest that the primitive reflexes can identify a “neurological” subgroup among patients with schizophrenia. The excess release of these primitive reflexes may also result from anomalies in brain development (Youssef & Waddington, 1988) and a neurodevelopmental origin of schizophrenia is currently gaining importance (Murray & Lewis, 1987).

It may be worthwhile to follow up such patients longitudinally along with other neurological parameters so that these release reflexes may act as markers of a subtle cerebral dysfunction of uncertain etiology. These forgotten signs should not be neglected due to lack of stability since many of the neurobehavioural symptoms may be transient, but if observed seriously, can provide great insights into the study of functional psychoses (Keshavan & Yeragami, 1987).

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