Disordered emotional expression characterized by outburst of involuntary, uncontrollable laughing and crying has been well recognized since the late nineteenth century (Adams and Victor, 1989). This pathological laughing or crying results from lesions of bilateral internal capsule also involving the basal ganglia, lesions of substantia nigra, cerebral peduncles, hypothalamus and from pronounced involvement of corticobulbar fibres. The best example are provided by lacunar vascular disease and less often by amyotrophic lateral sclerosis and multiple sclerosis. It may also result from widespread lesions of hypoxic-hypotensive encephalopathy, cerebral trauma or encephalitis (Adams and Victor, 1989).

Unilateral cerebral lesions have also been reported to be associated with mood alteration. The emotional state resulting from the left and right sided cerebral damage has been different. Insult to the left side of brain is likely to produce a dysphoric mood state (Sackeim et al., 1987), whereas the right sided insult is associated with euphoric state (Sackeim et al., 1982; Starkstein et al., 1988; Agarwal et al., 1987). One patient who developed pathological laughter after right cerebral infarction is being reported. To the best of our knowledge it has not been reported in Indian literature.

Case Report: Mrs. G. 45 years old, was brought by her husband with complaints of repeated spontaneous laughter and right sided weakness. She was alright about a month ago when she developed sub acutely a mild weakness of her left side. It was not associated with headache, nausea or vomiting, vertigo, diplopia, seizure or any alteration in conscious level. The weakness improved from second day onwards. After one week she undertook a short journey by train. While travelling she had a few episodes of spontaneous laughter. When she got down from the train, it was noticed that her left side paresis had increased to complete paralysis. Since then she had recurrent episode of spontaneous laughter for no apparent reason throughout the day. It occurred on the slightest provocation such as during an attempt to answer a question. Her episodes of hilarious laughter lasted for 1 to 12 minutes.

On examination her pulse was 76/min and BP was 130/90 mm of Hg. General examination was normal. Mental status examination couldn't be done as any attempt by her to answer question resulted in spontaneous laughter. She had left supranuclear facial weakness with left hemiplegia. Her deep tendon reflexes were brisk and extensor planter response was noted on left side. All her routine biochemical tests were within normal limits. Cranial CT Scan showed right frontoparietal infarction (Fig. 1). EEG done after one month of weakness was normal. Her motor power recovered gradually to

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1. Asstt. Professor, 2. Professor and Head, 3. Senior Resident, Department of Neurology, K.G.'s Medical College, Lucknow - 226 003.
grade III in six weeks time. Her episodes of pathological laughter were reduced after about two months. She conveyed verbally her feelings of embarrassment during these laughing spells and started having short weeping spells without hilarity. Mild laughing episodes on provocation are persisting in a two year follow up.

**DISCUSSION**

The lady described had a clear cut change in her emotional behaviour following left hemiparesis. She showed an enduring spontaneous, uncontrollable display of emotion in the form of repeated laughter which was unrelated to any precipitating factor in her surroundings. Her concurrent mood was unrelated to the displayed laughter and later on she started having associated weeping episodes as well. The laughter followed ischaemia of the right cerebral cortex. Cerebrovascular disease is the most common cause of pathological laughing (and crying) although it has also been described in a variety of neurological conditions such as tumours (Achari and Colover, 1976; Black, 1982), trauma, infections, degenerative, inflammatory and demyelinating disease (Adams and Victor, 1989; Sackeim et al., 1982), after hemispherectomy (Sackeim et al., 1982) and frontal lobotomy (Black, 1982).

Pathological laughing and crying has been usually associated with bilateral involvement of corticobulbar fibers. But now it has been convincingly shown that it may result from unilateral lesions as well. It has been demonstrated that right hemispheric damage is associated with euphoric reaction characterized by minimizations of symptoms, joking, elation, emotional placidity or social disinhibition (Agarwal et al., 1987; Gainotti, 1972). Sackeim et al. (1982) has reported significant association of outbursts of laughing with right hemispheric insult. Reports of patients who broke into peals laughter after right intracarotid amobarbital sodium injection have been recorded (Terzian, 1964). Administration of unilateral electroconvulsive therapy to the right side of brain has been associated with a better therapeutic response than left sided administration (Cohen et al., 1974).
Under normal circumstances emotional displays are subject to inhibitory control. Pathological laughing and crying is a result of release from cortical inhibitory fibers as a result of brain damage (Black, 1982). It appears that left side of brain subserves positive emotional states which is inhibited by right side. If the right sided inhibition is removed due to any pathological condition, positive emotional states eg. laughter may result. Outbursts of laughing are also observed as ictal phenomenon (gelastic epilepsy) (Daly and Mulder, 1957). They most frequently occur during the course of seizure but may also occur as component of aura or with termination of seizures. In contrast to the finding of pathological laughing to be associated with predominantly right sided destructive lesions, foci in the cases of gelastic epilepsy were more frequently left sided (Sackeim et al., 1982). Outbursts of laughing in this situation probably result from excitation within the left side of brain.

The functional-anatomic organization of the affective components of language in the right hemisphere has been described (Askenasy, 1987). The laughing outburst in this case is consistent with the available information about hemispheric lateralisation in prosing and expression of emotion. Why some patients develop outburst of emotion like laughing and crying while others develop frank mania or depression is not quite clear. It could be because of quantity changes in availability of concentration and responses to certain neurotransmitters. Neurotransmitter imbalance in emotional disturbance and lateralisation in human cortical catecholamine pathways is known (Mandell and Knapp, 1979).

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