The Functions and Dysfunctions
of Laughter

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ABSTRACT. Laughter is not usually included in the curriculum of medical schools, therefore it is often neglected by physicians. Laughter is a reflex located in the reticular system of the hindbrain. Rich connections explain its complexity. Laughter appears very early in life and each individual has a very characteristic and stable laughing style. Laughter and speech differentiate human beings from the animal kingdom. Laughter is a unique communication media, common to all human societies speaking different languages. Laughter has a mood elevating and relaxing effect. Laughter dysfunction consists of inappropriate response to the meaning or to the stimulus intensity. In the present study, laughter's dysfunction and disorders are presented. The purpose of this study was to extricate laughter from the diffuse obscurity of information into a new area of interest for scientific research.

LAUGHTER HAS A FUNCTION of release and purification through a complex discharge, and it is exclusively characteristic to humans. The reflex of laughter developed with the neocortex and appeared together with speech in humans (Darwin, 1890). Plato considered laughter a rational reaction, developed for hiding a lack of self-knowledge (Jowett, 1971). Rabelais (1982; a physician, priest, and writer) considered laughter an emotional reaction. He was captivated by the close association of laughter to well-being and humor. Philosophers were the first group interested in laughter and physicians were the last (Higel, 1959). Darwin (1890) in his evolutionist naturalist theory insisted on the precipitating role of satisfaction and stress resolution offered by laughter. Two evolutionist philosophers, Spencer and Bergson, considered laughter as a released energy (Bergson, 1911; Spencer, 1860). Freud (1905) adopted this concept and widely developed his psychological understanding of laughter as a released energy. Psychoanalysis understands laughter as a

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discharge of psychic energy, resulting from a longstanding inhibited tension that ends in satisfaction (Freud, 1905). The emotional eruption of laughter is the result of the contrasting effect of facing a naïve joke when expecting something very solemn and important (Keith-Spiegel, 1972). The stimulus intensity causing the tension has an important role. In infants only moderate intensity provokes curiosity and the exploration reflex, which are the precursor conditions for laughter (Rothbart, 1973). Kant (1952) had a similar opinion about the basic mechanism of laughter as the sudden transformation of a strained expectation into nothingness. Many other authors underlined the role of surprise in the laughter mechanism (Goodenough, 1931; Hayworth, 1928).

The aggressive function of laughter has been widely debated. In primitive people, laughter either enhanced or diverted aggression (Berlyne, 1972; Gordon, 1969; Lorenz, 1963). In the bible, the word laughter appears 29 times and on 13 occasions (45%) it is related to violence (Ziv, 1978). The baring of teeth or grinning during laughter has been related to an inherited trait derived from the threatening snarls of animals in an aggressive mood, from the savage shout of triumph, or the cruel mocking of a conquered or hated enemy by anthropological studies (Brown, 1925). Grinning is found in animals such as the yaffle or the green woodpecker (the laughing bird) and the laughing crow-goose, a variety of domestic pigeon called "the laughter" (Lockard, 1977).

The Social Substrate of Laughter

Bergson (1911) underlined the very general social substrata of the communicative function of laughter. Laughter is a primitive communication medium understood by all human societies in spite of their very different languages. No case of a human being who did not laugh once in his lifetime has been published in the literature. Age and sex influence the social substrate of laughter. Statistically, 94% of preschool children's laughter occurs in groups and only 6% when the child is alone (Kennerdine, 1931). Laughter frequency increases in direct proportion to social agglomeration. An obvious example is the collective laughter at parties (Freedman, 1979). The occurrence of epidemic laughter is a direct consequence of its social dependence (Bean, 1967; Izard, 1979). The group dependent laughter due to its role in communicative behavior has a universal cultural quality (Eibl-Eibesfeldt, 1972). The adaptive value in human social life of laughter becomes evident when compared with the destructive effect of inappropriate laughter in social contact. In their social relations humans try to make their partner laugh, ensuring a successful circulation of thoughts and emotions. Laughter may many times be a means of seeking approval, because it enhances self-confidence and dissipates the uncertainty in human contact, hence, some cultivate the technique of telling jokes. Moreover, when the laughter response is achieved the partner is in-
clined to reproduce it again and the mechanism is established afresh. The social attribute of laughter has its roots in tribal life, which bound them together (Wallis, 1922).

Development of Laughter

There are controversies concerning the period of laughter’s appearance. Some authors consider it to appear between the 6th and 8th week after birth (Gewirtz, 1965; Washburn, 1929). But laughter has been observed at the age of 5 weeks as a vocal response to a pat-a-cake game (Izard, 1977). Smiling is regarded as an innate human reaction developing gradually to laughter (Black, 1984). Jouvet and his group considered smiling as appearing in neonates from the first day (Challamel, Lahlou, Revol, & Jouvet, 1985). Smiling expresses a positive affect in connection with the functional role of Rapid Eye Movement (REM) sleep (Izard, 1977). Nevertheless, smiling and laughter were shown to have the same pattern in all human societies despite great economic and cultural differences (Gewirtz, 1965).

Age is the first important variable in laughter development. The aged people change the amplitude of their response to laughter stimuli. Maturation has an inhibitory role on laughter. Sex is the second important variable in laughter’s development. Hysterical laughter characterizes school girls more than females at any other age (Black, 1982).

Laughter Manifestation

Dearburn (1900) described laughter as the “repetitive contractions of the diaphragm, continuous contractions of the facial muscles, raising the corners of the eyes and eyebrows, flaring of the nostrils, elevation of the cheeks, retraction of the mandible and head, vasodilatation of the face, neck and hands, exophthalmia and tears.” This original description portrays laughter as a very general reflex, involving diaphragm, neck, and hand movements. Recently several authors described laughter as a mainly facial expression: “the upper lip is raised in smiling, it partially uncovers the teeth and also brings down a downward curving of the furrows, which extend from the wings or both nostrils to the corners of the mouth, this in turn, produces a puffing, or rounding out of the cheeks on the outer side of the furrows, creases also occur momentarily under the eye sockets, the eyes themselves undergo a general change which can best be described as becoming bright and sparkling’’ (Chevallier-Skolnikoff, 1973; Pollio, Mers, & Luchesi, 1972).

The true manifestation of laughter is a combination of both with mention that the major component of laughter was not underlined by both descriptions. It consists of the abrupt expiration due to a sudden contraction of the intercostal muscles (Sveback, 1975). This breathing motor activity is fol-
lowed by saccadic expiration-inspiration microcycles (Fry & Hader, 1977). As a result of the saccadic breathing, vocal cords add short and broken sounds. A varying number of agonistic synergistic and antagonistic skeletal muscles participate in the laughter reflex depending on its intensity. During excessive laughter the whole body is thrown backward. In infants the tendency to throw their body backward when sitting marks the start of laughter (Goodenough, 1931). Due to breathing motor changes, the cardiovascular system is also involved by means of head and face vasodilation, distended veins, and tachycardia. According to Stearns (1972), the reflex lacrimation produces a proportionate amount of moisture, based on the increased circulation in lacrimal glands (Figure 1).

**FIGURE 1.** Scheme of the facial expression of laughter.
The specific rhythm of skeletal muscle activity, breathing, cardiovascular changes, body carriage, lacrimation, and the specific sound determine a characteristic pattern, leading to an individual style of laughter (Fry & Hader, 1977). The laughing style is as stable a characteristic for the same individual as his fingerprints (Fry & Hader, 1977). In every individual's stable laughing style is a gradation scale. Darwin (1890) described a gradation scale from lower to excessive. Spencer (1860) noted the difference between smile and laughter to the intensity scale gradation. Pollio, Mers, and Luchesi (1972) described a four level scale from low, mild, moderate, to explosive. Kostler (1964) dedicated a whole study to the detailed analysis of laughter gradation. A genetic predisposition for wild laughing was found in the constitutional type of obese, logorrheic, and bulimic man displaying oral behavior.

**Mechanism of Laughter**

The rhythmic contractions of all skeletal muscles involved in laughter originates in a hypothetical pacemaker that coordinates these complex periodic cycles. It is suggested that the laughter pacemaker is located in the hindbrain and is responsible for the individual's very stable laughing style. The existence of this pacemaker has not yet been proven by direct excitation.

The assumption of a brainstem pacemaker of laughter leads to a reconsideration of the gradation concept. Until now it has been accepted that stimulus intensity determines different levels of neuronal recruitment as manifest by the gradation phenomenon of laughter (Darwin, 1890; Kostler, 1964; Pollio, Mers, & Luchesi, 1972; Spencer, 1860). The existence of the many various inhibitory circuits controlling laughter's pacemaker, however, may be responsible for the gradation phenomenon. Variables such as age, sex, education, culture, personality, and constitutional type may strengthen or weaken the inhibitory circuits, the result being a controlled, free, or wild outburst of the individual laughter style. The synchronized rhythmic sequences of breathing and laughing implies a neighborhood localization of respiratory centers and laughter pacemakers in the reticular system.

The reticular location makes possible short polysynaptic connections to all the motor cranial nerve nuclei, explaining the simultaneous facial expression, vocal sounds, lacrimation, and heart rhythmic changes during laughter. Experimental studies on cats showed connections in the periaqueductal gray matter between facial motor centers and vagus nuclei (Kelly, Beaton, & Magoun, 1946). The observation of two patients with a brainstem tumor in whom paroxysmal laughter was stimulated by eye pursuit movements and associated with urinary incontinence, indicated the reticular brainstem as a unique possible convergence locus of these mechanisms (Mackeith, 1959). Various other internuclear connections exist at brainstem level with inferior olivary nucleus and cerebellum (Hermann & Brown, 1957). One may con-
sider laughter as a mass response involving nervous, muscular, respiratory, and vascular systems controlled by a large area of the reticular brainstem. This large area belongs to a long and wide segment of the inferior brainstem tegmentum from the level of the 3rd to the level of the 12th cranial nerves.

The mechanism of laughter, however, cannot be explained only by this short polysynaptic interconnecting system without its very large spectrum of connections with the neocortex. The fact that jokes are specific laughter stimuli implies efferent connections from the perceptive verbal area of Wernicke to the brainstem pacemaker. Furthermore, the fact that visual caricature and doubled laughter are specific stimuli, implies efferents from occipital conicortex to the laughter pacemaker. The effect of hearing laughter provoking laughter, proves the existence of effferent connections from the auditory perceptive area to the hindbrainstem pacemaker. Capability of laughing artificially without effective content or “volitional laughter” proves motor cortex connections toward brainstem reticulum. The intrinsic relations between surprise, fun, triumph, joy, shyness, aggression, fear, and laughter, shows close relations with the limbic system of the brainstem pacemaker (Hayworth, 1928; Ironside, 1956; Papez, 1937).

During the REM sleep stage, the brain is activated through the reticular system and favors a low degree of laughter and smiling. Mostly related to pleasant dreams, sleep laughter (at a certain threshold of intensity) provokes an arousal due to the concomitant muscle contractions.

A wide spectrum of connections with subcortical formations, such as the thalamus (Von Bechterew, 1935), the subthalamus (Spencer, 1894), and basal ganglia (Davison & Kelman, 1939; Poeck & Pilleri, 1963; Wilson, 1923), was found to be involved in the motor organization of laughter. Diencephalo-hypothalamic connections achieve the cardiovascular and vegetative component of laughter (Davison & Kelman, 1939; Martin, 1950; Von Bechterew, 1935). The fact that laughter can be elicited by tickling protective body areas such as soles of the feet, armpits, ribs, and solar plexus shows the existence of connections between thalamo-parietal pathway and brainstem reticular area (Hayworth, 1928).

**Biochemistry of Laughter**

The mood elevative effect of laughter is assumed to be based on a biochemical mechanism involving various neurotransmitters. Due to the brief transient state, it is difficult to duplicate laughter in experimental conditions and because it is not found in animals, the biochemical changes are very difficult to study. During laughter the subject feels released from present cares and worries and a mood of joy prevails. For its duration, laughter inhibits a depressive preexisting mood, hence a norepinephrine elevation was suggested (Schildkraut, 1965) and serotoninergic involvement was affirmed (Maas, 1975). A
relationship between REM latency and the frequency of smile during sleep was also suggested together with a hypothesis that a rise in catecholamines and a fall in enkephalin levels is related to the antidepressant function of laughter (Hartman, 1968). The decrease of pathologic laughing obtained in 10 out of 25 severely demented patients treated with the antiparkinsonian drug, levodopa, proves once again the catecholaminergic involvement in the laughter mechanism (Udaka, Yamao, Nagata, Nakamura, & Kameyama, 1984). The inappropriate and disproportionate laughter characterizing psychotic patients may be due to a biochemical deficit at the dopaminergic receptor level. The suppression of depression by laughter may be worthy of further studies.

Laughter Dysfunctions and Disorders

Laughter dysfunctions and disorders were observed and described mainly in psychiatric, neurologic, pediatric, and internal medical practice studies (Challamel, Lahlou, Revol, & Jouvet, 1985; Freud, 1905; Hayworth, 1928; Kris, 1950; Pollio, Mers, & Luchesi, 1972). Psychotic laughter is the most frequent in the realm of laughter disorders. Encountered in schizophrenia (as out of a contextual meaning, Bleuler, 1951), Kraepelin named it “the silly laughter, unrestrained, appearing on all occasions without the least provocation and is altogether without emotional significance” (Defendorf, 1904). Bleuler (1950) grouped in parathymania the distorted expressions of unprovoked laughter frequently encountered in schizophrenia. They do not know why they are laughing, they feel that they are being compelled to laugh and they deny enjoyment or a feeling of happiness. Izard (1977) emphasized the contagious character of laughter in schizophrenics: “An entire ward may break out in hilarious laughter.” Black (1982) underlined the interchangeability of laughter-crying in schizophrenics. An exaggerated amount of laughter is encountered in mania and hypomania (Highet, 1959). “Laughing wild” was the name given by Gray to mania (Kolb, 1977). Shakespeare was the first to mention the lack of laughter in melancholy (Kolb, 1977). The depression is usually associated with suppression of laughter and decreased motor activity (Maddison & Duncan, 1965).

Hysterical laughing spells with no clear motivation are well known (Meissner, 1980). It is found following trauma, shock, and anxiety states (Geldman, 1959). Owing to its extracontextuality it is diagnosed early (Alexander, 1952; Wolfenstein, 1955). There is a contrasting feature between the moderation of the schizophrenic laughter and the screaming laughter of hysteria. The epidemic hysterical laughter described by Bean (1967) may be observed in a religious environment. He described it spreading among 1,000 school girls, affecting all of the surrounding schools, houses of the families, and culminating with whole villages as well. Some patients required hospi-
talization because of exhaustion (Bean, 1967). Sterns (1972) explained the convulsive epidemic laughter as a low socioeconomic infrastructure group of girls who, separated abruptly from tribal life, suffered anxiety and loss of identity which, in the imposed discipline of the religious school, erupted into epidemic hysterical laughter. A psychoneurotic forced laughter that is very difficult to distinguish from hysterical laughter was described in a head trauma (Dewan, 1958), Kuru disease (Gajdusek, 1963), frontal lobotomy (Kramer, 1954), and as an aura event of Grand Mal epilepsy (Anderson, 1936).

The second common laughter disorder is the spastic laughter associated with the lacunar syndrome. An apparent emotive lability provokes an explosive onset of spastic laughter that is out of proportion to the stimulus intensity. The sound is of a lamentation and not of happiness, whereas the facial expression is only slightly participating in the laughter. It may very easily turn by means of a slight suggestion into crying. A large spectrum of stimuli such as a visit, attempts to speak, or motor activity may initiate spastic laughter. It cannot be voluntarily controlled. In very severe cases spastic laughter may be caused by catastrophic news. The subsiding of spastic laughter may concur with the overall clinical remission and resolution of the last new microinfracst in lacunar syndrome. Its permanent presence imposed the diagnosis of pseudobulbar syndrome. Bilateral widespread lacunar lesions of the cortico-subcortical areas determined by atherosclerosis, multiple cerebral infarcts, or multiple sclerosis are the cause of pseudobulbar syndrome (Haymaker & Kuhlenbeck, 1980; Poeck, 1969). Unilateral lesions may also produce pseudobulbar laughter (Ironside, 1956). It may be present in spastic hemiplegia, in internal capsule hemorrhage, and in amyotrophic lateral sclerosis (Black, 1982; Poeck, 1969; Wilson, 1923).

Angelman's (1985) description of the puppet-like syndrome adds this disease to the group of spastic laughter. This syndrome consists of an association of gross mental retardation, microcephaly, epilepsy, ataxic gait, tongue protrusion, and speech absence with recurrent paroxysms of laughter (Bower & Yearyons, 1967; Dooley, Berg, Pakula, & Macgregor, 1981). Three clinical signs indicate the involvement of brainstem damage in this disorder: broad based ataxic gait, tongue protrusion, and bouts of inappropriate laughter.

All of these pathologic entities display a spastic laughter of the pyramidal type, but there is a spastic laughter of extrapyramidal type encountered in idiopathic Parkinson's disease (Haymaker & Kuhlenbeck, 1980; Poeck, 1969). The clinical difference between the pyramidal and extrapyramidal spastic laughter is that the latter is more monotonic, rigid, and of low amplitude rhythmic laughter than the former. Spastic extrapyramidal laughter may turn into pyramidal type following stereotaxic neurosurgery.

The third, and less common, laughter disorder consists of convulsive laughter. Saccadic episodes of uncontrolled laughter associated with or without tickling of certain areas of the body and with or without other focal epi-
FIGURE 2. Postural reflex convulsive laughter—Jacome’s patient (with author’s permission): (a) In the upper lefthand corner the precipitating convulsive position; (b) Patient resumes hyperextension with start of seizure; (c) Convulsive laughter occurs 4 s from the EEG start of the seizure.
leptic signs occurring up to 12–14 times a day in the presence of a certain cloudiness of consciousness constitute convulsive laughter. The seizures appeared with no precipitating factors and they are not associated with urine or fecal incontinence. Febrile convulsions in childhood may be present. Neurologic examination, laboratory investigations, CT scan, and interictal EEG are usually normal. Gascon and Lambrose (1971) required five criteria to designate convulsive laughter: (a) stereotyped recurrence, (b) absence of external precipitants, (c) other manifestations of epilepsy, (d) presence of ictal or interictal EEG epileptiform activity, and (e) absence of other causes of pathologic laughter.

According to Lennox and Lennox, convulsive laughter was first described by Dostoyevsky in his book *The Idiot* (Dostoyevsky, 1960). The author described these seizures as consisting of laughter associated with pleasure, harmony, grinning, giggling, and joyful weeping (Dostoyevsky, 1960). Another very early description of laughter seizures is attributed to Trouseau (1873). Daly and Mulder (1957) named this type of convulsive disorder gelastic epilepsy. Convulsive laughter goes back in history, when attempts were made to link this phenomenon with manifestations of religious experiences, both sacred and profane contributing to the connotation of epilepsy as *morbus sacer*.

From an epidemiological point of view, convulsive laughter is a rare epilepsy. Chen and Foster (1973) reviewed 5,000 children suffering from convulsive disorders and found 7 patients with gelastic seizures, representing 1.40% of epileptics. Less than 150 cases have been reported in the literature up until now (Matusik, Eisenberg, & Meyer, 1981). One may find 1–3% of laughter seizures among epileptics (Gascon & Lombrosco, 1971). It is much more prominent in children than in adults (Matusik, Eisenberg, & Meyer, 1981). Usually neurologists have the obsessive habit of localizing, categorizing, and classifying epileptic attacks. In this respect laughing epilepsy may appear in two forms, as a pure laughter seizure or associated with other automatisms (Daly & Mulder, 1957; Holmes, Dardick, & Russman, 1980). The majority of laughter epilepsy previously described in the literature belong to the associated type (Matusik, Eisenberg, & Meyer, 1981). The pure form consists of clouding of consciousness, saccadic laughter lasting approximately 30 s, usually followed by various degrees of amnesia (Daly & Mulder, 1957; Holmes, Dardick, & Russman, 1980; Loisseau, Cohanon, & Cohanon, 1971). In the associated type of laughter epilepsy, three groups of associations should be individualized: (a) with temporal lobe epilepsy; (b) with precocious puberty and mental retardation; and (c) miscellaneous brain disorders. In the group of “associated with temporal lobe epilepsy” running epilepsy is most frequently encountered, very rarely crying epilepsy (Quintarian or dacrocytic epilepsy), and extremely rare orgasmoepilepsy (Holmes, Dardick, & Russman, 1980; Jacome, McLain, & Fitzgerald, 1980; Sethi & Surya, 1976). Ja-
come et al. (1980) described a patient whose volitional hyperextension of the neck and trunk, precipitated laughter seizures associated with intense sexual feeling (orgasmolepsy). This postural reflex gelastic seizure was of temporal origin (Jacome, McLain, & Fitzgerald, 1980) (Figure 2). Often, Davidoff, Troost, and Richev (1976) described a patient suffering from laughter convulsions associated with weeping epilepsy and torticollis epilepsy due to a temporal lobe atrophy. Head turning was reported also in four cases of crying epilepsy (Offen, Davidoff, Troost, & Richev, 1976). In the associated form, the interictal EEG shows bilateral temporal slow waves on a generally flattened background (Druckman & Chao, 1957; Gascon & Lombroso, 1971; Kris, 1950; Lehtinen & Kivalo, 1965; Loisseau, Cohanon, & Cohanon, 1971). Wieser (1983) observed a 14-year-old boy suffering from fits of laughter accompanied by aggressive outbursts and aversive movements of the head due to a right mammillary body astrocytoma. Stereotaxic EEG depth electrodes (chronically implanted) recorded left periamygdaloid, left anterior temporal, and left frontocingulate cortex paroxysmal waves. Following neurosurgery the fits diminished (Figure 3).

The second group associated with precocious puberty and mental retardation is usually caused by a posterior hypothalamic pathology (Doff, 1938; Gascon & Lombroso, 1971; Matustik, Eisenberg, & Meyer, 1981; Yamada & Yoshida, 1977). These patients have a benign course in childhood. EEG shows frequent paroxysmal spikes and slow wave activity. The symptoms of precocious puberty are usually manifested as episodes of vaginal bleeding or external genital signs of maturity such as pubic hair or auxiliary leg hair by the age of 4. A clear cut Wechsler intelligence test for children reveals a mean of 10 to 58. This form of convulsive laughter lacks an affective component and is a type of laughter epilepsy without affect (Money & Hosta, 1967; Williams, Schmitt, & Savage, 1978).

The third group of association with miscellaneous brain disorders associated convulsive laughter with various cortical and subcortical lesions, such as frontal (Matustik, Eisenberg, & Meyer, 1981), limbic (Ironside, 1956; Loisseau, Cohanon, & Cohanon, 1971; Sethi & Surya, 1976), third ventricle (Foerster & Gagel, 1932), thalamic (List, Dowman, Bagehi, & Bebin, 1958; Poeck, 1969; Weil, Nosik, & Demmey, 1958), basal ganglia and the pituitary gland (Martin 1950), and mamillary bodies (Ironside, 1956).

The general etiology of the secondary laughter epilepsy is related to hemorrhages, tumors, encephalitis, trauma, and lipid storage disorders (Black, 1984; Holmes, Dardick, & Russman, 1980; Poeck, 1969; Trouvseau, 1873). Stress, fatigue, hyperventilation, and specific positioning of the head may also precipitate seizures (Jacome, 1980; Loisseau, Cohanon, & Cohanon, 1971; Sethi & Surya, 1976). A lateralization study linked left hemisphere to laughter epilepsy more readily than the right (Lipsey, Robinson, Pearlson, Rao, & Price, 1983).
The fourth laughter disorder in the order of frequency is gas induced laughter. In 1772, Priestley discovered nitrous gas \((\text{NO}_2)\), which he indulged in at revellers for social amusement because of the euphoria it produced. A reduced sensitivity to pain in such revellers was noted and it was suggested that it might be of use in surgery as an anaesthetic (Lyons & Petrucelli, 1978). The mechanism by which it produces laughter is still an enigma to science.

The fifth laughter disorder is laughter induced cataplexy. In this case a spell of laughter provokes cataplexy. This rare and intriguing association was stipulated since the first description of narcolepsy by Gelineau (1880) and further developed in Zarcone’s (1973) revision. Lachschlag syndrome consists of a cataplectic loss of muscle with or without irresistible attacks of sleep provoked by laughter. Cataplectic episodes may also be provoked by sudden fear or anger but laughter is much more common in precipitating attacks. The
suggested localization of the disturbance is the reticular system of the pontomesencephalon including the locus caeruleus (Hobsen, 1974). During laughing spells changes in the reticular formation may facilitate a decrease of muscle tone, favoring cataplectic attacks (Figure 4). Why all narcoleptics do not faint when laughing is still unexplained. Patients suffering from laughter epilepsy and presenting an associated hypotonia during gelastic attacks were also described and this association may offer a suggestion for the understanding of laughter induced cataplexy (Bower & Yearons, 1967; Lehtinen & Kivalo, 1965).

The last laughter disorder to be presented is Giggle incontinence or "I laughed until I wet myself." The causal relationship between laughing and uncontrolled urination has long been accepted. Mackeith (1959) coined the term giggle micturition in order to distinguish this form of leakage from stress incontinence induced by laughter. Another term in use is enuresis risoria (Glalin, 1979). One common strategy is to curb the sense of humor and maintain a straight face in order to avoid it. Urodynamic tests of these cases revealed no abnormality (De Jonge, 1975). Two common features that charac-

![FIGURE 4. Suggested reticular brainstem laughter pacemaker localization. Schematic representation of main cell groups of the medulla (section through mild-olivary region).](image-url)
terize all laughter disorders are reaction out of or contrary to contextual meaning and laughter not proportionate to the stimulus intensity or unrestrained.

**Laughter Therapy**

The cathartic effect of the laughter reflex consisting of a relaxation and a feeling of well-being indicates gelototherapy as a promising field for medicine. Recovery from ankylosing spondylitis by a regimen of self-prescribed laughter was reported (Cousins, 1976). This unique observation showed a lowering of erythrocyte sedimentation rate together with an immediate effect following laughter regimen. The use of laughter as a psychotherapeutic tool proved to be successful (Jackson & King, 1982; Potter & Goodman, 1983). The traditional date of April 1st for merriment may be interpreted as a preventive mass therapy, offered by human culture against stress and fear. The roots of this custom can be found in the Roman Empire period where every March 25th of the vernal equinox, Hilaria was celebrated.

Laughter is a neuropsychological field of inquiry, sui generis, belonging to the neglected medicine. By delineating this area of interest, scientific research will be possible.

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