

Clinical Summary



Dementia pugilistica

By

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ICD codes

ICD-9:

Postconcussion syndrome: 310.2

Organic brain syndrome (chronic): 294.9

Contusion of unspecified sites: 924.9

ICD-10:

Postconcussional syndrome: F07.2

Organic brain syndrome (chronic): F06.9

Contusion of unspecified sites: T14.0

Synonyms

Boxers dementia; Punch-drunk syndrome

Historical note and nomenclature

Boxing has been a human activity since antiquity (Corsellis 1989). The safety of boxers became an issue in the early 19th century when the use of spikes and other brutal methods was outlawed in Great Britain. In 1866 the Marquis of Queensbury Rules established the use of boxing gloves, weight categories, and the 10-second knockout. However, these measures did not eliminate either the acute or the chronic neurologic effects of boxing. In 1928, Martland recognized the syndrome of dementia pugilistica when he recognized the "punch drunk" or "slug nutty" syndrome in professional boxers (Martland 1928). In recent years, investigators have clarified the role of repeated subconcussive head injuries in the development of dementia pugilistica (Unterharnscheidt 1995). These concerns over brain damage from boxing have led to frequent calls to ban the sport (Council on Scientific Affairs 1983; Cowart 1989). The incidence of boxing related chronic traumatic brain injury, however, has decreased during the last decades because of the reduction in exposure to repetitive head trauma and increasing medical monitoring of boxers. Medical and neuroimaging assessments have also contributed to the detection of early and potentially pre-symptomatic cases of chronic traumatic brain injury (Clausen et al 2005).

Clinical manifestations

Dementia pugilistica is a syndrome characterized by motor, psychiatric, and cognitive symptoms (Morrison 1986; Guterman and Smith 1987; Jordan 1987; Mendez 1995; Erlanger et al 1999; Jordan 2000; Blennow et al 2005; Toth et al 2005). Dementia pugilistica is the progressive, end-stage of the chronic traumatic encephalopathy of boxers. Chronic traumatic encephalopathy does not have to be progressive and constitutes a continuum from mild signs and symptoms of neurologic dysfunction to the

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complete syndrome of dementia pugilistica. Chronic traumatic encephalopathy in boxers evolves gradually or after a significant bout, and usually begins near the end of the boxing career or shortly after retirement. Boxers progress to dementia pugilistica 7 to 35 years after the initiation of boxing, often many years after the last boxing match (Roberts 1969). In general, dementia pugilistica occurs in professional boxers who have had multiple bouts and who are known for "taking a punch" (Critchley 1957; Roberts 1969; Ryan 1998). Compared to professional boxers, amateur boxers show milder neurophysiological and neuroimaging evidence of chronic traumatic encephalopathy (Haglund et al 1990; Haglund and Eriksson 1993; Stewart et al 1994; Kemp et al 1995).

The first symptoms of chronic traumatic encephalopathy in boxers are motor, affective, and personality changes. Motor symptoms include an intention tremor, **dysarthria**, and mild incoordination, especially in the nondominant hand. Occasionally, parkinsonian or pyramidal tract signs occur in mild chronic traumatic encephalopathy. The affective changes are characterized by emotional lability, euphoria, and hypomania. Conversely, some boxers become withdrawn and apathetic or manifest a reactive depression. Other personality changes include psychomotor retardation, exacerbation of premorbid personality traits, aggression, suspiciousness, childishness, loquacity, and restlessness (Brooks et al 1987).

Chronic traumatic encephalopathy in boxers may progress to a second stage distinguished by parkinsonism and an accentuation of the psychiatric symptoms. In addition to the **tremors** and dysarthria, **rigidity**, **bradykinesia**, and other **extrapyramidal** signs develop. Psychiatrically, there is prominent difficulty with impulse control, disinhibition, irritability, inappropriateness, excessive sexual demands, and explosive outbursts of anger and aggression. There may be morbid jealousy and a suspicion of spousal infidelity, and a pathological sensitivity to the effects of alcohol. Memory difficulty begins in this stage, usually as a mild retrieval deficit that is worst for information of little personal significance (Johnson 1969).

As chronic traumatic encephalopathy progresses to dementia pugilistica, there is a decrease in cognitive and daily functions, often accompanied by signs of pyramidal tract disease and **ataxia** (Johnson 1969). In dementia pugilistica, there is prominent slowness of thought and speech, and impairments of memory, attention, and frontal-executive behaviors (Johnson 1969; Brooks 1976; Bond 1986). Dementia pugilistica patients frequently have a cheerful or silly affect and diminished insight despite uninhibited violent behavior and paranoia (Critchley 1957; Spillane 1962; Johnson 1969; Roberts 1969). Occasionally, they exhibit other, specific behaviors, such as hyperorality and hypersexuality as elements of the Klüver-Bucy syndrome from injury to the tips of the temporal lobes (Johnson 1969).

Neuropsychological studies of former and active boxers, although not specific for dementia pugilistica, illustrate the pattern of cognitive deficits that herald this dementia. Eight neuropsychological studies of professional boxers (n=274) found difficulties in memory, information processing and speed, finger tapping speed, attention and concentration, sequencing abilities, and **frontal-executive functions** such as judgment, abstraction, reasoning, planning, and organization (Johnson 1969; Roberts 1969; Kaste et al 1982; Sironi et al 1982; Casson et al 1984; Drew et al 1986; Levin et al 1987; Ross et al 1987). In one study, 18 former and active primarily professional boxers had abnormal results on at least one neuropsychological test (Casson et al 1984). In another study, 15 out of 19 boxers scored in the impaired range of the Reitan Impairment Index, as compared to 2 of 10 matched basketball and baseball players (Drew et al 1986).

Neuropsychological abnormalities correlated significantly with the number of professional bouts, the presence of abnormal CT scans, or both (Roberts

1969; Casson et al 1984; Drew et al 1986; Ryan 1998). In contrast, lower verbal ability in boxers may be a premorbid condition (Roberts 1969; Thomassen et al 1979), and neuropsychological tests can be normal in young, active professional boxers (Levin et al 1987).

Thirteen studies that included amateur boxers (n=856) suggest the presence of mild neuropsychological changes (Thomassen et al 1979; Kaste et al 1982; Casson et al 1984; Brooks et al 1987; Levin et al 1987; McLatchie et al 1987; Ross et al 1987; Heilbronner et al 1991; Murelius and Haglund 1991; Butler et al 1993; Haglund and Eriksson 1993; Stewart et al 1994; Kemp et al 1995). Recent studies show a decline in memory and in visuoconstructive performance (Stewart et al 1994; Kemp et al 1995;). Two studies found lower finger tapping speed, especially in the left hand of amateur boxers who participated in a large number of bouts (Thomassen et al 1979; Murelius and Haglund 1991). In other studies, decreases in learning and slower reaction times did not differ between amateur boxers and other athletes (Brooks et al 1987; McLatchie et al 1987; Butler 1994). No neuropsychological dysfunction emerged when amateur boxers were compared to water polo and rugby players (Butler et al 1993), former or active soccer players (Thomassen et al 1979; Murelius and Haglund 1991), track and field athletes (Murelius and Haglund 1991), and amateur athletes or boxers in training (Brooks et al 1987; Levin et al 1987). In addition, there were no neuropsychological differences when former amateur boxers with a long career (25 to 180 bouts) were compared with those with a short career (0 to 15 bouts) (Murelius and Haglund 1991) or when amateur boxers and controls were matched on the basis of the lower educational status of boxers (Thomassen et al 1979). Finally, although Heilbronner and associates found impairments in verbal and incidental memory after a fight (Heilbronner et al 1991), another study failed to confirm pre- versus post-boxing differences in amateur boxers (Butler et al 1993).

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Clinical vignette

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Etiology

The cause of the brain injury in chronic traumatic encephalopathy and dementia pugilistica is the cumulative effect of repetitive subconcussive blows (Critchley 1957; Guterman and Smith 1987; Jordan 1987). Although serious head injuries are less common in boxing than in horse racing, sky diving, football, mountaineering, and other sports, head injuries are particularly repetitive in boxing (Morrison 1986). The cumulative effect of mild head injuries may cause permanent sequelae (Carlsson et al 1987), and even very mild degrees of brain trauma can produce neuropathological changes similar to severe trauma (Povlishock and Coburn 1989). Experimental studies in animals confirm the presence of **severe head injury** after repeated applications of low intensity blows at short interblow intervals of 5 to 20 seconds (Unterharnscheidt 1975). In addition, a blow delivered by a professional boxer can have a force of 0.63 tons delivered in 100 ms, equivalent to being struck by a 13 pound mallet at a velocity of 20 mph (Atha et al 1985).

The damage from individual blows to the head is due to a mixture of different types of forces. There may be linear acceleration forces due to a straight-on blow to the head through the center of gravity. There is no loss of consciousness, but there may be coup-contrecoup contusions, gliding contusions in the parasagittal regions, and subfrontal contusions where the inner skull provides a hard surface. There is also an acute deceleration injury when a boxer's head strikes the mat. The most important force is that of rotational acceleration due to the inertial lagging of the brain relative

to the skull. Because the brain is suspended and partly tethered, oblique blows, such as hooks or uppercuts, result in revolution of the brain within the skull with loss of consciousness, [diffuse axonal injury](#), and even subdural hematomas from torn bridging veins. In addition, head blows can produce a "groggy state" or [delirium](#) in which boxers are poorly responsive but remain on their feet. During the groggy state, boxers are at greater risk for rotational injuries because of a loss of defensive reflexes, particularly the inability to stabilize their neck muscles. Zhang and colleagues performed MR and diffusion-weighted imaging in 24 boxers and in 14 age- and sex-matched control subjects with no history of head trauma ([Zhang et al 2003](#)). They found that the measured diffusion values in the boxer group were significantly higher than those measured in the control group. The most common MR finding in the boxer group was volume loss, [cavum septum pellucidum](#), subcortical and periventricular white matter disease.

Other factors may play an etiological role in dementia pugilistica. First, an important recent study shows that boxers with the [apolipoprotein E epsilon4 allele](#) are particularly susceptible to chronic neurologic deficits ([Jordan et al 1997](#)). The apolipoprotein E epsilon4 allele is a major susceptibility gene for [Alzheimer disease](#). Second, again similar to Alzheimer disease, dementia pugilistica patients have neuropathology in the nucleus basalis of Meynert, the source of most [CNS](#) cholinergic neurons, and a compromise of the acetylcholine system ([Uhl et al 1982](#)). Third, there is compromise of the [blood-brain barrier](#) as revealed by increases in creatine kinase isoenzyme BB from astrocytes during the first 9 minutes after a boxing match ([Brayne et al 1982](#)), and as suggested by increased [CSF](#) protein ([Mawdsley and Ferguson 1963](#)). Fourth, boxing may accelerate age-associated cognitive changes, which are further accentuated by a lower level of premorbid education, verbal abilities, and neuropsychological functioning in many boxers ([Johnson 1969](#); [Thomassen et al 1979](#); [Haglund and Eriksson 1993](#)). Finally, [chronic subdural hematomas](#), obstructive [hydrocephalus](#), prior cerebral hypoxia, vascular injury, disseminated intravascular coagulation, and systemic fat [emboli](#) may contribute to brain damage in individual boxers.

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Pathogenesis and pathophysiology

The cognitive deficits of dementia pugilistica reflect the location of the brain damage. Linear and rotational movement damages the frontotemporal regions where they abut on non-smooth bony surfaces. These contusions result in disinhibition and executive disturbances from orbitofrontal injury, and memory impairment from temporal-hippocampal damage. Small cortical scars with specific cortical deficits result from petechiae, microcirculatory disruption, and direct contusions in gray matter ([Payne 1968](#); [Adams and Bruton 1989](#); [White and Likavec 1992](#)). Furthermore, atrophic changes, scarring, and Purkinje cell loss in the base of the cerebellum probably follow transient tonsillar herniation and contribute to [dysarthria](#), incoordination, and [ataxia](#) ([Corsellis et al 1973](#)).

Diffuse axonal injury is presumably the source of much of the cerebral atrophy and secondary ventricular enlargement in dementia pugilistica, as well as much of the attentional deficit. Rapid rotational acceleration-deceleration results in [diffuse axonal injury](#) from shearing of nerve fibers, particularly the long-tract axons originating in the brain stem ([Strich and Oxon 1961](#); [Adams et al 1977](#)). Diffuse axonal injury is characterized by thinning and atrophic changes in the dorsolateral midbrain, [basal ganglia](#), periventricular white matter, and [corpus callosum](#) ([Pearce 1984](#); [Bodensteiner and Schaefer 1997](#)).

Geddes and colleagues ([Geddes et al 1999](#)) examined the brains of 4 young men age range 23 to 28 years, all of whom suffered mild chronic

head injury. Pathological findings cases were of neocortical neurofibrillary tangles and neuropil threads, with groups of tangles consistently situated around blood vessels in the worst affected regions. It appeared that repetitive head injury in young adults may be initially associated with neocortical neurofibrillary tangle formation in the absence of amyloid beta-peptide deposition. The distribution of the tau pathology suggested that the pathogenesis of cytoskeletal abnormalities may involve damage to blood vessels or perivascular elements.

The rotational injury further damages other midline structures. Injury to the superior cerebellar peduncle-red nucleus contributes to [dysarthria](#) and tremor. There is a loss of pigment and neurons in the lateral aspect of the [substantia nigra](#) contributing to the development of parkinsonism ([Unterharnscheidt 1995](#)). Boxers have flattening and detachment of the fornix, and a greater incidence of a [cavum septum pellucidum](#) from fenestration and rupture into the lateral ventricles ([Corsellis et al 1973](#); [Bodensteiner and Schaefer 1997](#)). Twelve of 13 autopsied cases of dementia pugilistica had a cavum septum pellucidum compared to less than 2% of all adult autopsies ([Corsellis et al 1973](#)). The cavum of boxers differs from the normal variant in that there are more and larger fenestrations.

There are diffuse neurofibrillary tangles in the cerebral cortex as well as in the temporal horn areas ([Unterharnscheidt 1995](#)). In the hippocampi and amygdalae, abundant neurofibrillary tangles accompany extensive neuronal loss ([Lampert and Hardman 1984](#)). These tangles may occur early throughout the neocortex and before other neuropathological changes ([Geddes et al 1996](#)). Similar to the neurofibrillary tangles of [Alzheimer disease](#), the neurofibrillary tangles of dementia pugilistica are ubiquitinated ([Dale et al 1991](#)), have tau-immunoreactivity ([Tokuda et al 1991](#)), respond to the same antisera ([Roberts 1988](#)), and have high levels of aluminum and iron ([Bouras et al 1997](#)). Unlike Alzheimer disease, the neurofibrillary tangles of dementia pugilistica are located in superficial layers of the neocortex rather than deep layers ([Hof et al 1992](#)), and occur without neuritic plaques; however, there are beta protein immunoreactive deposits (early plaques) throughout the neocortex ([Roberts et al 1990](#); [Tokuda et al 1991](#)). In addition, dementia pugilistica patients have cerebrovascular amyloid deposits ([Tokuda et al 1991](#)). Boxing and repeated head injuries have been also associated with [hypopituitarism](#), and a high prevalence of neuroendocrine abnormalities ([Kelestimur et al 2004](#)).

Schmidt and colleagues ([Schmidt et al 2001](#)) compared the molecular profile of tau pathologies in dementia pugilistica with those in Alzheimer disease and showed that the same tau epitopes map to filamentous tau inclusions in Alzheimer disease and dementia pugilistica brains, whereas the abnormal tau proteins isolated from dementia pugilistica brains are indistinguishable from the 6 abnormally phosphorylated brain tau isoforms in Alzheimer disease brains. Thus, these data suggest that recurrent brain injury may cause dementia pugilistica by activating pathological mechanisms similar to those that cause brain degeneration due to accumulations of filamentous tau lesions in Alzheimer disease, and similar, albeit attenuated, activation of these processes by a single brain injury may increase susceptibility to sporadic Alzheimer disease decades after the event. Pathologically, chronic [traumatic brain injury](#) shares many characteristics with Alzheimer disease (ie, neurofibrillary triangles, diffuse amyloid plaques, acetylcholine deficiency, or abnormal tau accumulation immunoreactivity) ([Jordan 2000](#); [Blennow et al 2005](#)).

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Epidemiology

In modern times, the prevalence of chronic traumatic encephalopathy and dementia pugilistica in professional boxers has diminished due to increased

safety precautions and better monitoring of the sport. Mild or "subclinical" brain damage is much more common than the complete dementia pugilistica syndrome (Stiller and Weinberger 1985). In the only true epidemiological study, Roberts randomly selected 224 former professional boxers from the British boxing registry. One half of these had evidence of neurologic or neuropsychiatric abnormalities. Chronic traumatic encephalopathy was clearly present in 17% overall and in 28% of those over the age of 50 (Roberts 1969); however, among the remaining boxers, 40% had **disequilibrium**, **dysarthria**, or alcohol intolerance. Johnson characterized 17 former, mostly professional fighters with chronic traumatic encephalopathy. Eleven of these boxers had a chronic amnesic state, 3 had a progressive dementia, 5 had morbid jealousy syndrome often in the face of impotence, 4 had rage reactions often with pathological intoxication, and 2 had a persistent psychosis (Johnson 1969). Zyzryn and colleagues studied a total of 107 injuries were in 427 fight participations, corresponding to an injury rate of 250.6 injuries per 1000 fight participations (Zyzryn et al 2003). The most commonly injured body region was the head/neck/face (89.8%). Specifically, injuries to the eye region (45.8%) and **concussion** (15.9%) were the most frequent.

The incidence and severity of dementia pugilistica correlates with men who began fighting in their teens and had had several hundred professional fights (Johnson 1969; Roberts 1969), had a 10-year or longer boxing career (Johnson 1969; Roberts 1969; Casson et al 1984; Jordan et al 1997), and were nonscientific boxers known for taking a punch (Critchley 1957; Johnson 1969; Roberts 1969; Corsellis et al 1973). With some exceptions (Sironi et al 1982; Jordan et al 1992), most studies show a greater relationship of chronic traumatic encephalopathy and dementia pugilistica to greater than 20 professional bouts than to the number of knockouts (Roberts 1969; Casson et al 1984; Drew et al 1986; Ross et al 1987; Ryan 1998). Furthermore, dementia pugilistica may be associated with the total number and type of head blows, particularly if the angle of impact or failure to stabilize the head results in rotational head movements.

Parenthetically, several reports have shown a higher-than-average incidence of head injury in the history of patients with **Alzheimer disease** (Rudelli et al 1982; Mortimer et al 1985; Sullivan et al 1987), suggesting that head trauma plays a provocative or permissive role in the development of the dementia of Alzheimer disease.

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Prevention

The predominant aim of boxing is to cause physical damage to the opponent. Protective measures during boxing do not entirely eliminate the risk of developing dementia pugilistica, and a large medical consensus supports the banning of boxing, especially professional boxing (Council on Scientific Affairs 1983; Cowart 1989). An alternative approach would be to reduce the number and severity of blows allowed to the head.

Barring these controversial solutions, there are other techniques to minimize the risk of head injury during boxing. First, strict regulations must cover boxing, preferably through a single boxing authority. For example, amateurs are better protected because of regulations covering the monitoring of bouts, limiting the number of rounds, improving the padding of gloves, etc. Second, there could be better mechanisms to identify and suspend boxers with early signs of dementia pugilistica. This includes mandatory medical records and better pre- and post-fight screening with neurologic examinations, neuropsychological testing, EEGs, CTs, or MRIs (Ross et al 1987; Charnas and Pyeritz 1990). Third, ringside physicians under state control could have greater powers to intervene early and to impose longer suspensions. Finally, educational efforts should stress

informed consent about the risk of brain damage and emphasize reducing the risks of head injury. Unfortunately, although the wearing of headgear decreases the force of impact, it does not prevent rotational injury and could add to the risk because of the greater surface area available for impact. Moreover, the wearing of gloves, which was introduced to protect the fists and not the brain, may accentuate the force of the blow as the gloves become wet and heavy from sweating.

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Differential diagnosis

The differential diagnosis of dementia pugilistica includes [Alzheimer disease](#), frontotemporal [dementia](#), [vascular dementia](#), and neurodegenerative conditions, such as [Parkinson disease](#) with dementia, [diffuse Lewy body disease](#), olivopontocerebellar atrophy, and others. The possibility of contributions from alcoholic dementia and from consequent nutritional deficiencies are particularly pertinent among this population. Dementia pugilistica may be distinguished from other dementing illnesses by the characteristic course and involvement of motor, psychiatric, and cognitive symptoms. Furthermore, neuroimaging helps rule out coincident neurologic changes, such as [chronic subdural hematomas](#).

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Diagnostic workup

In addition to a basic neurologic workup, the diagnostic evaluation emphasizes the predictive value of neuroimaging procedures for the development of the chronic traumatic encephalopathy of boxers and dementia pugilistica. Chronic [traumatic brain injury](#) associated with boxing occurs in approximately 20% of professional boxers ([Jordan 2000](#)). CT scans in professional boxers show atrophy of the cerebrum and cerebellum with ventricular enlargement in 50% to 60% and a [cavum septum pellucidum](#) in 10% to 23% ([Casson et al 1982](#); [Kaste et al 1982](#); [Jordan et al 1992](#); [Bodensteiner and Schaefer 1997](#)). Neuroimaging also shows evidence of previous perivascular hemorrhage, contusions, porencephalic cysts, and [chronic subdural hematomas](#). The frequency of a cavum septum pellucidum in boxers is higher than the 0.73% to 5.4% incidence among consecutive adult CT scans ([Macpherson and Teasdale 1988](#); [Bogdanoff and Natter 1989](#)). The cerebral atrophy correlates with the presence of a cavum septum pellucidum. Cerebral atrophy on CT occurred in 27% of boxers without a cavum septum pellucidum, 44% with a small cavum, and 67% with a large cavum ([Jordan et al 1992](#)). Although not found by Jordan and colleagues ([Jordan et al 1992](#)), other studies show a correlation of cerebral atrophy on CT scans with the number of bouts fought ([Ross et al 1983](#); [Casson et al 1984](#)), with impairment on memory and other neuropsychological tests ([Casson et al 1984](#); [Ross et al 1987](#)), and with a history of knockouts ([Casson et al 1982](#); [Sironi et al 1982](#); [Jordan et al 1992](#)). MRI scans may not detect any acute changes in boxers ([Levin et al 1987](#)); however, MRI is superior to CT in the long-term evaluation of professional boxers and can detect abnormalities not seen on CT scans ([Jordan and Zimmerman 1990](#)). Moreover, Cabanis and associates noted cerebral atrophy on MRI among boxers who could "stand punishment" versus those who avoided punches ([Cabanis et al 1986](#)). Finally, in contrast to professional boxers, amateur boxers, even those suspended due to knockouts or excessive head blows, lack definite changes on CT or MRI scans ([Jordan and Zimmerman 1988](#); [Haglund and Bergstrand 1990](#); [Haglund and Persson 1990](#); [Holzgraefe et al 1992](#); [Haglund and Eriksson 1993](#)).

Neurophysiological studies show variable results. EEG abnormalities may occur in one third to one half of professional boxers, and consist of diffuse slowing or flat, low-voltage records ([Johnson 1969](#); [Sironi et al 1982](#); [Ross et al 1983](#)). EEG changes correlate with the number of bouts fought and

with a history of knockouts (Sironi et al 1982; Ross et al 1983). Even among amateurs, Haglund and Persson found a higher incidence of mild EEG abnormalities compared to soccer players and track and field athletes (Haglund and Persson 1990). Brain electric activity mapping, brainstem auditory evoked potentials, and P300 evoked responses are not abnormal in boxers (Breton et al 1991; Haglund and Eriksson 1993). In a single metabolic study, flow values significantly below normal were observed in both hemispheres of professional boxers with xenon-133 inhalation (Rodriguez et al 1983). In one recent study, cerebral perfusion scanning showed more aberrations among amateur boxers than among other sportsmen (Kemp et al 1995).

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Prognosis and complications

There is no clear method of evaluating whether patients with mild chronic traumatic encephalopathy will progress to dementia pugilistica. Mild, static symptoms of chronic traumatic encephalopathy are probably much more common than the full syndrome of dementia pugilistica. Risk factors associated with dementia pugilistica include increased exposure (ie, duration of career, age of retirement, total number of bouts), poor performance, increased sparring, and [apolipoprotein](#) genotype (Jordan 2000). Moreover, early abnormalities on neuropsychological measures and cerebral atrophy or a [cavum septum pellucidum](#) on neuroimaging may presage the development of chronic traumatic encephalopathy. The width of the third ventricle on neuroimaging may be a particularly useful prognostic index, and indicates diencephalic atrophy from [diffuse axonal injury](#).

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Management

The management of chronic traumatic encephalopathy and dementia pugilistica emphasizes the avoidance of continued head injuries, even if it means ending the boxing career. Speech, physical, and occupational therapy may help in minimizing the deficits from dementia pugilistica. Active rehabilitation can result in modest gains in neuropsychological functions and in interpersonal skills. Antipsychotics, lithium, antidepressants, sedatives, anxiolytics, anticonvulsants, opiate antagonists, and beta-blockers are potentially useful to treat aggression, violence, and poor impulse control among boxers. Olanzapine and other new [neuroleptic](#) drugs can be useful in the treatment of aggressive and psychotic behavior, while minimizing the amount of drug-induced [extrapyramidal](#) syndrome; however, clozapine increases the risk of seizures in these patients (Michals et al 1993). Anti-Parkinsonian medications for the extrapyramidal symptoms are another consideration. Finally, cognitive rehabilitation strategies include teaching the patient new strategies to compensate for lost or impaired functions. It has not been established whether cholinesterase inhibitors are useful to improve symptoms of [memory loss](#) and cognitive dysfunction in this population.

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Pregnancy

Pregnancy is not a factor.

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Anesthesia

Anesthesia is not a factor.

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Associated disorders

[Alzheimer disease](#)

Chronic traumatic encephalopathy of boxers

[Huntington disease](#)

[Pick disease](#)

Posttraumatic dementia

Posttraumatic parkinsonism
[Subcortical dementia](#)

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Differential diagnosis

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frontotemporal dementia
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neurodegenerative conditions
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[diffuse Lewy body disease](#)
dementia associated with olivopontocerebellar atrophy
alcohol/drug-induced dementias
dementia due to subdural hematoma

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Demographics

For more specific demographic information, see the Epidemiology, Etiology, and Pathogenesis and pathophysiology sections of this clinical summary.

Age

19-44 years
45-64 years
65+ years

Population

None selectively affected.

Occupation

Boxers

Sex

male>female, >2:1
male>female, >1:1

Family history

None

Heredity

None

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