Skeletal and Dental Fractures Associated With Electroconvulsive Therapy

Chittaranjan Andrade, MD

Each month in his online column, Dr Andrade considers theoretical and practical ideas in clinical psychopharmacology with a view to update the knowledge and skills of medical practitioners who treat patients with psychiatric conditions.

Department of Clinical Psychopharmacology and Neurotoxicology, National Institute of Mental Health and Neurosciences, Bangalore, India (candrade@psychiatrist.com).

ABSTRACT

During the motor seizure associated with electroconvulsive therapy (ECT), the muscles of the trunk and limbs contract forcefully and repetitively, predisposing to injuries to muscles, joints, teeth, and bones. This motor seizure is irrelevant to the therapeutic action of the treatment. It is therefore modified by the administration of an intravenous muscle relaxant, such as succinylcholine, after the administration of the anesthesia in the ECT premedication. Well-modified ECT is associated with markedly diminished skeletal muscle contractions and hence with minimal skeletal and dental risks. In this context, anecdotal reports across a range of skeletal disorders testify to the safety of well-modified ECT in ultrahigh-risk patients. Population-based data suggest that the fracture risk with modified ECT is 2 events per 100,000 ECTs; if only recent data are examined, the risk may be as low as 0.36 events per 100,000 ECTs. Population-based data also suggest that the dental fracture risk with modified ECT is 0.02% per ECT and 0.17% per ECT course. The overall magnitude of skeletal and dental fracture risk depends on patient factors and on how well the ECT procedure is performed. Preexisting bone and dental disease increase the risk; good seizure modification, proper use of bite blocks, and effective jaw immobilization during ECT reduce the risk. Careful assessment of preexisting risk and good ECT practice can minimize the risk of skeletal and dental complications during ECT.

J Clin Psychiatry 2023;84(1):23f14797

Electroconvulsive therapy (ECT) is perhaps the most effective treatment available for major mental illness. ECT is associated with adverse effects related to (*a*) the administration of a short-acting anesthetic agent, (*b*) the administration of a muscle relaxant, (*c*) the administration of an anticholinergic drug (if indicated), and (*d*) the delivery of an electrical stimulus to the brain.[1]

The electrical stimulus delivered during ECT results in a central and peripheral seizure. The central seizure comprises the generalized synchronous bursts of neuronal discharge that can be observed in the electroencephalogram, and the peripheral or motor seizure comprises the generalized tonic-clonic convulsion that is observed in the trunk and limbs. The central seizure is essential for the efficacy of ECT and is also responsible for much of the variance in the cognitive adverse effects of the treatment. The motor seizure has no therapeutic value, is cosmetically displeasing, and may rarely be associated with peripheral adverse effects affecting muscles, joints, teeth, and bones. The motor seizure is therefore attenuated or "modified" through the use of an intravenous muscle relaxant in the ECT premedication; this is why ECT, as presently practiced, is known as modified ECT. How effectively the musculoskeletal and dental adverse effects are minimized depends on how well the motor seizure is modified.

Musculoskeletal and dental injuries result from events such as stretching, twisting, compression, or direct injury. In the context of ECT and the motor seizure, the sudden jerk associated with tonic contraction of muscles and the repeated jerks associated with each clonic contraction comprise the musculoskeletal events that can result in musculoskeletal or dental injury. Skeletal and dental fractures are examples of such injury.

A large number of population-based studies have used national medical registers, health insurance databases, and other health care databases to examine the association between medication exposure and health outcomes. Similar studies examining ECT have only recently begun to be published. A previous article in this column examined the risk of bone fractures occurring after exposure to prolactin-raising and prolactin-sparing antipsychotic drugs.[2] This article examines the risk of skeletal and dental fractures occurring in the context of ECT.

Unmodified ECT and Skeletal Fractures

In the era before routine seizure modification, ECT was associated with a 20%–40% risk of skeletal fractures; a few studies reported lower risks, and others, higher risks. The risks were greater in men than in women, in patients with greater muscularity, and in those who had osteoporosis. The fractures were mostly compression fractures of the vertebral bodies in the thoracic spine. The fractures were generally subclinical, were not necessarily associated with backache, and were detected through radiologic screening of the spine. Fractures elsewhere, such as of long bones, were rare. No long-term follow-up of affected patients was available.[3]

Chittaranjan Andrade

Unmodified ECT continues to be reported.[4] Two prospective studies in the current era examined the occurrence of vertebral fractures in patients treated with unmodified ECT. In the first study,[5] conducted at a center at which anesthesiological facilities were unavailable, 50 consecutive patients each received a course of 6 bilateral sinusoidal wave ECTs. X-rays of the thoracolumbar spine were obtained in all patients. Only 1 patient (2%) was found to have experienced a vertebral fracture during the ECT course. In the second study,[6] 56 consecutive patients received a mean of 2.9 (total, 162) bilateral brief-pulse ECTs before anesthesiological support could be obtained. X-rays of the thoracolumbar spine were obtained in all patients. No patient experienced vertebral fracture. Both studies had been conducted with the specific intent of demonstrating the musculoskeletal hazards of unmodified ECT with a view to discourage its practice. The findings of both studies were therefore unexpected and puzzling. It was hypothesized that the use of intravenous benzodiazepines (usually, 10 mg of diazepam) as premedication, in lieu of anesthesia and succinylcholine, may have attenuated the intensity of the motor seizure through their muscle-relaxant action.[6] These findings notwithstanding, for many clinically important reasons, the use of unmodified ECT is strongly discouraged.[3]

ECT and Risk of Fractures:
The Benefits of Seizure Modification

Guidance has been provided for the administration of ECT to patients with or at high risk of fractures[7]; the emphasis lies on good seizure modification. As earlier stated, the skeletal risks associated with ECT arise from the tonic-clonic contractions of the muscles of the trunk and limbs, and these contractions are attenuated by the use of succinylcholine or other muscle relaxants in the ECT premedication. Succinylcholine is commonly dosed at 0.5–1.0 mg/kg because, at this dose, the motor seizure is reasonably well modified for most patients; about 5% of patients, however, may require doses > 1.5 mg/kg.[8] Because of wide interpersonal variation, a neurostimulator may need to be used to identify the ideal dose for an individual patient. However, if succinylcholine is dosed at 1–2 mg/kg for patients at high risk of orthopedic complications, muscle relaxation during ECT could be expected to be reasonably complete; the tonic phase of the convulsion would be limited to the period (commonly, <1 s) for which electricity is passed and perhaps for a few seconds thereafter, and the clonic phase would be limited to a few weak twitches of the trunk and limbs.[9–11]

Because of the infrequency of cases and ethical difficulties in conducting randomized clinical trials in such patients, the orthopedic safety of modified ECT in ultrahigh-risk patients must be evaluated through anecdotal literature. Whereas fractures have been reported under unusual circumstances in patients receiving modified ECT,[12–17] many reports testify to the safety of the treatment in the context of ultrahigh-risk patients. These include patients with severe osteoporosis,[18] metastatic bone disease,[19] osteogenesis imperfecta,[20] Ehlers-Danlos syndrome,[21] and Harrington rod implants.[22,23] These also include patients with recent long bone fractures,[24] multiple bone fractures,[25] surgical repair of hip fracture,[26–28] vertebroplasty,[29] and maxillofacial repair.[30]

Fracture Risk With ECT: Population-Based Data

In a study of reports of ECT-related adverse events in the Veterans Affairs National Center for Patient Safety database for the years 1999–2010, Watts et al[31] identified 1 case of fracture across an estimated total of 73,440 ECT treatments. The fracture was of the ulna, and it had resulted from the forearm striking the bedrail during the convulsion; the ECT team had failed to ensure adequate neuromuscular paralysis. Thus, the treating team was at fault and not the treatment.

Two other studies provided population-based estimates of skeletal fracture associated with ECT. In one study, Blumberger et al[32] described a population-based cohort study of morbidity and mortality associated with ECT in Ontario, Canada. The data were obtained from health administrative records for 2003–2011. The sample comprised 8,810 adults. The median age of the sample was 52 years; 26% of patients were > 65 years old. The sample was 61% female. These patients received a total of 135,831 ECTs. There were 88 cases of hip fracture recorded, equivalent to a rate of 65 fractures per 100,000 ECTs.

It is hard to know what to make of this finding because the authors[32] examined not fracture during ECT but fracture within 7 days of ECT. This means that, in the study, fractures would have been associated with ECT even had they occurred outside the ECT suite, on days in between ECTs, and in the week after the end of the ECT course. The fractures could have been because of falls related to ECT, concurrent medications, medical comorbidities, or other factors. In order to better interpret their findings, the authors should have included a comparison cohort comprising patients with severe depression who did not receive ECT.

In the other study, Luccarelli et al[33] obtained data from US states that mandated reporting of ECT treatments; these states were California, Colorado, Illinois, Texas, and Vermont. Illinois contributed the least data in terms of years of coverage (8 years) and Texas contributed the most data (25 years). Vermont contributed the least data in terms of numbers of patients and treatments (1,994 patients, 27,821 ECTs) and Texas, again, the most (41,212 patients, 293,946 ECTs). Overall, there were 15 incidents of fracture in 111,424 patients across 737,477 ECTs. This corresponds to 2.0 fracture events per 100,000 ECTs.

For the most recent years of the study,[33] 2013–2017, data were available for all 5 states. During this period, only 1 fracture event was reported in 41,989 patients across 280,894 ECTs. This corresponds to a fracture event rate of 0.36 per 100,000 ECTs. Luccarelli et al[33] suggested that improved anesthesia practice may have been responsible for the lower fracture rate during 2013–2017. They observed that the fracture risk with ECT is lower than the risk of colon

perforation during colonoscopy or the risk of mortality during general anesthesia.

An important limitation of this study[33] is that only fractures apparent to the treating physicians would have been reported; subclinical fractures would have remained unnoticed. However, to detect subclinical fractures, routine spinal x-rays would have been necessary, and, because of the likelihood of low yield, performing this investigation routinely would not be cost-effective.

Dental Fracture With ECT: Mechanisms

Oral health is poor in patients with major mental illness for reasons related to poor nutrition, self-neglect, decreased salivation due to anticholinergic effects of medications, and others.[34] Therefore, patients with major mental illness who receive ECT may be at increased risk of dental adverse events during ECT. These events occur because the muscles of the jaw contract forcefully during the motor seizure, exerting greater sudden impact and subsequent sustained pressure on the teeth than during normal biting and chewing; the incisors are particularly at risk of loosening or breaking because they are slightly inclined forward and because they are not as structurally strong as the premolars and molars. A further problem is that, because ECT is administered in repeated sessions, injury to teeth may cumulate across the ECT course. The risks could be expected to be greater with unmodified than with modified ECT, though this has not been formally proven.[3]

Dental Fracture With ECT: Risks

Older studies reported widely different rates for dental fracture with ECT, ranging from none across more than 200,000 ECT sessions[35] to 3 in 242 patients.[36] A more recent but very small study[37] identified 1 tooth fracture in 30 patients who received a total of 68 ECTs; this sample may have been atypical because, before ECT, the mean number of sound teeth was only 15 in subjects who were dentate, and because 10 patients already had at least 1 broken tooth.

Dental Fracture With ECT: Population-Based Data

In a study of reports of ECT-related adverse events in the Veterans Affairs National Center for Patient Safety database for the years 1999–2010, Watts et al[31] identified 5 cases of tooth injury, all related to non-use or incorrect use of the bite block, across an estimated total of 73,440 ECT treatments.

Göterfelt et al[38] described what may be the first and (so far) only population-based study that specifically examined dental fracture associated with ECT. In this study, dental fracture was defined as loss of a part or whole of 1 or more teeth. The study authors extracted data for 16,681 patients from Swedish national registers for the years 2012–2019. The mean age of the sample was 52 (range, 13–99) years. The sample was 60% female. The patients had received 254,906 ECTs across 32,862 courses of treatment. Depression was the commonest indication for ECT, and 66% of ECTs had been administered with unilateral electrode placement.

There were 56 dental fractures reported; that is, 0.02% per ECT, 0.17% per ECT course, and 0.34% per patient (the average patient had received 2 courses of treatment). Dental fracture rates did not differ significantly between men and women, across different age and diagnosis groups, and across different electrode placement groups; because dental fracture was rare, these analyses may have been underpowered. However, the mean number of ECTs received was greater in patients who had experienced dental fracture than in those who had not (29.9 vs 15.2, respectively). No dental complication more serious than dental fracture was reported. The study[38] also noted that, during 2011–2018, there were 342 ECT-related malpractice claims among which 35 were related to dental fracture.

An important but unavoidable limitation of this study[38] is that the findings are only as good as the quality of the data reported; for example, instances of loosening of teeth may have been underreported if treating teams did not look for this adverse outcome. Another limitation is that the register information could not differentiate between chipped and lost teeth. Finally, the standard of ECT practice may vary across centers and oral health and dental care may vary across countries; so, the findings of this study may not generalize well to all ECT practices and to all environments.

Reducing ECT-Related Dental Risks

Dental fractures with ECT are almost always due to poor ECT technique. The risk of dental fractures during ECT can be minimized by examining and attending to dental problems, if any, before the ECT procedure; removal of dentures before ECT; insertion of a properly designed bite block that provides adequate cushioning and is appropriately sized to the patient's mouth; and application of adequate upward pressure on the mandible, starting before the passage of current and continuing to the end of the convulsion in order to minimize tonic-clonic jaw movements that can injure the teeth. Guidelines for dental risk management are available (Minneman 1995,[39] Morris et al 2002,[40] Paparone et al 2019[41]).

Concluding Notes

Forceful and repetitive skeletal muscle contractions during ECT are associated with a very small but definite risk of skeletal and dental fractures. The magnitude of risk depends on patient factors and on factors related to the practice of ECT. Preexisting bone and dental disease are examples of patient factors that increase the risk; good seizure modification, proper use of bite blocks, and effective jaw immobilization are procedural factors that reduce the risk. Careful screening for and assessment of preexisting risks and meticulous attention to good ECT practice will markedly reduce the risk of skeletal or dental complications during ECT. The onus for minimization of risk therefore lies with the ECT team.

A higher absolute stimulus dose and a longer stimulus duration could increase the intensity and duration of

Chittaranjan Andrade

stimulus-related skeletal muscle contraction during ECT. The effects of these stimulus variables on skeletal and dental risks with modified ECT therefore merit evaluation in future research.

Whereas skeletal complications that are clinically significant are likely to be symptomatic and hence detected, dental complications that are clinically significant are hidden in the mouth and may not be detected if not asked about.

Standard operating procedures for post-ECT evaluation should therefore include inquiries about and examination of teeth. Finally, more population-based studies in the field are required; however, formal prospective study of skeletal and dental outcomes could yield more trustworthy results because routinely reported information in health care and insurance databases may not include all clinically significant adverse events.

REFERENCES

1. Andrade C, Arumugham SS, Thirthalli J. Adverse effects of electroconvulsive therapy. *Psychiatr Clin North Am.* 2016;39(3):513–530.
2. Andrade C. Prolactin-raising and prolactin-sparing antipsychotic drugs and the risk of fracture and fragility fracture in patients with schizophrenia, dementia, and other disorders. *J Clin Psychiatry.* 2023;84(1):23f14790.
3. Andrade C, Shah N, Tharyan P, et al. Position statement and guidelines on unmodified electroconvulsive therapy. *Indian J Psychiatry.* 2012;54(2):119–133.
4. Chanpattana W, Kramer BA, Kunigiri G, et al. A survey of the practice of electroconvulsive therapy in Asia. *J ECT.* 2010;26(1):5–10.
5. Andrade C, Rele K, Sutharshan R, et al. Musculoskeletal morbidity with unmodified ECT may be less than earlier believed. *Indian J Psychiatry.* 2000;42(2):156–162.
6. Shah N, Mahadeshwar S, Bhakta S, et al. The safety and efficacy of benzodiazepine-modified treatments as a special form of unmodified ECT. *J ECT.* 2010;26(1):23–29.
7. Shah DD. A protocol for administration of electroconvulsive therapy in elderly patients with fractures. *Int J Psychiatry Clin Pract.* 2000;4(2):101–104.
8. Li EH, Bryson EO, Kellner CH. Muscle relaxation with succinylcholine in electroconvulsive therapy. *Anesth Analg.* 2016;123(5):1329.
9. American Psychiatric Association. *The Practice of Electroconvulsive Therapy: Recommendations for Treatment, Training and Privileging. Task Force Report on ECT.* American Psychiatric Association; 2001.
10. Bowley CJ, Walker HAC. Anesthesia for ECT. In: Scott AIF, ed. *The ECT Handbook, 2nd ed: The Third Report of the Royal College of Psychiatrists' Special Committee on ECT.* Royal College of Psychiatrists; 2005:124–135.
11. Andrade C. Variations on a theme of unmodified electroconvulsive therapy: science or heresy? *J ECT.* 2010;26(1):30–31.
12. Sarpel Y, Toğrul E, Herdem M, et al. Central acetabular fracture-dislocation following electroconvulsive therapy: report of two similar cases. *J Trauma.* 1996;41(2):342–344.
13. Nott MR, Watts JS. A fractured hip during electro-convulsive therapy. *Eur J Anaesthesiol.* 1999;16(4):265–267.
14. Mirza A, Johnson TB. Acetabular fracture secondary to electroconvulsive therapy: a case report. *Am J Orthop.* 2006;35(7):327–328.
15. Baethge C, Bschor T. Wrist fracture in a patient undergoing electroconvulsive treatment monitored using the "cuff" method. *Eur Arch Psychiatry Clin Neurosci.* 2003;253(3):160–162.
16. Uebaba S, Kitamura H, Someya T. A case of wrist fracture during modified electroconvulsive therapy. *Psychiatry Clin Neurosci.* 2009;63(6):772.
17. Luccarelli J, Fernandez-Robles C, Berg SM, et al. Bilateral forearm fractures during modified electroconvulsive therapy in a male patient with a history of hyperparathyroidism and elevated pseudocholinesterase activity. *J ECT.* 2019;35(3):e35–e36.
18. Baker NJ. Electroconvulsive therapy and severe osteoporosis: use of a nerve stimulator to assess paralysis. *Convuls Ther.* 1986;2(4):285–288.
19. Wang G, Milne B, Rooney R, et al. Modified electroconvulsive therapy in a patient with gastric adenocarcinoma and metastases to bone and liver. *Case Rep Psychiatry.* 2014;2014:203910.
20. Coffey CE, Weiner RD, Kalayjian R, et al. Electroconvulsive therapy in osteogenesis imperfecta: issues of muscular relaxation. *Convuls Ther.* 1986;2(3):207–211.
21. Sienaert P, De Hert M, Houben M, et al. Safe ECT in a patient with the Ehlers-Danlos syndrome. *J ECT.* 2003;19(4):230–233.
22. Hanretta AT, Malek-Ahmadi P. Use of ECT in a patient with a Harrington rod implant. *Convuls Ther.* 1995;11(4):266–270.
23. Bhat T, Pande N, Shah N, et al. Safety of repeated courses of electroconvulsive therapy in a patient with Harrington rods. *J ECT.* 2007;23(2):106–108.
24. Dighe-Deo D, Shah A. Electroconvulsive therapy in patients with long bone fractures. *J ECT.* 1998;14(2):115–119.
25. Weller M, Kornhuber J. Electroconvulsive therapy in a geriatric patient with multiple bone fractures and generalized plasmocytoma. *Pharmacopsychiatry.* 1992;25(6):278–280.
26. Bryson EO, Liebman L, Nazarian R, et al. Safe resumption of maintenance electroconvulsive therapy 12 days after surgical repair of hip fracture. *J ECT.* 2015;31(2):81–82.
27. Tay YH. Safe electroconvulsive therapy use after hip fracture fixation without an increase in muscle relaxant. *J ECT.* 2017;33(4):e45–e46.
28. Otsuki K, Hayashi S, Nagahama M, et al. Favorable outcome after modified electroconvulsive therapy in the perioperative management of a patient with treatment-resistant schizophrenia and a femoral neck fracture. *J ECT.* 2021;37(3):e29–e31.
29. Briggs MC, Popeo DM, Pasculli RM, et al. Safe resumption of electroconvulsive therapy (ECT) after vertebroplasty. *Int J Geriatr Psychiatry.* 2012;27(9):984–985.
30. Baker M, Turner M. Use of ECT after maxillofacial repair. *J ECT.* 2000;16(4):421–422.
31. Watts BV, Groft A, Bagian JP, et al. An examination of mortality and other adverse events related to electroconvulsive therapy using a national adverse event report system. *J ECT.* 2011;27(2):105–108.
32. Blumberger DM, Seitz DP, Herrmann N, et al. Low medical morbidity and mortality after acute courses of electroconvulsive therapy in a population-based sample. *Acta Psychiatr Scand.* 2017;136(6):583–593.
33. Luccarelli J, Henry ME, McCoy TH Jr. Quantification of fracture rate during electroconvulsive therapy (ECT) using state-mandated reporting data. *Brain Stimul.* 2020;13(3):523–524.
34. Muzyka BC, Glass M, Glass OM. Oral health in electroconvulsive therapy: a neglected topic. *J ECT.* 2017;33(1):12–15.
35. Barnes WL, Relyea RP. A dental device for ECT anesthesia. *Hosp Community Psychiatry.* 1970;21(9):301.
36. McClure RE. A device for preventing dental injuries during ECT. *Hosp Community Psychiatry.* 1969;20(11):357–359.
37. Beli N, Bentham P. Nature and extent of dental pathology and complications arising in patients receiving ECT. *Psychiatr Bull.* 1998;22(9):562–565.
38. Göterfelt L, Ekman CJ, Hammar Å, et al. The incidence of dental fracturing in electroconvulsive therapy in Sweden. *J ECT.* 2020;36(3):168–171.
39. Minneman SA. A history of oral protection for the ECT patient: past, present, and future. *Convuls Ther.* 1995;11(2):94–103.
40. Morris AJ, Roche SA, Bentham P, et al. A dental risk management protocol for electroconvulsive therapy. *J ECT.* 2002;18(2):84–89.
41. Paparone P, Ee PL, Kellner CH. Oral protection in electroconvulsive therapy: modified technique using 2 bite blocks. *J ECT.* 2019;35(4):224.