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Major adverse cardiac events and mortality associated with

electroconvulsive therapy: Correcting and updating a 2019 meta-analysis.

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ABSTRACT

This paper updates a 2019 review of the research about the incidence of major cardiac events

following ECT, and critiques claims that these are rare and, therefore, inconsequential. It

concludes that the 2019 review, misrepresents its own findings which were, anyway, based on

a rather obvious miscalculation of the probability of cardiac events. Having corrected and

updated the review with five subsequent studies, it is calculated that the probability of ECT

causing one or more of six cardiac events (myocardial infarction, life-threatening arrhythmia,

acute pulmonary edema, pulmonary embolism, acute heart failure and cardiac arrest) is

between one in 15 and one in 30 patients, and that these cardiac events are a major cause of

ECT-related deaths. The ethical principle of informed consent is being routinely breached by

ECT psychiatrists.

ECT MORTALITY RATE

Electroconvulsive Therapy (ECT) is still used on more than a million people internationally

every year. Professional bodies and ECT researchers and advocates claim it is effective and

safe. Various official reports, most psychiatric textbooks, and many patient information

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leaflets have claimed, for decades, that the risk of death from ECT is so rare as to be inconsequential. For example, an American Psychiatric Association [APA] ECT Task Force Report (APA, 2001) claimed, with no supporting evidence, that 'A reasonable current estimate' is '1 per 10,000 patients or 1 per 80,000 treatments' (assuming the average course is eight individual ECT treatments). It should be noted that six of the eight Task Force members were Directors of ECT services. The group included the incoming and outgoing presidents of the Association for Convulsive Therapy, and three were financially involved with companies manufacturing ECT machines. This group of people devoted just one of their report's 245 pages to the deaths, which it euphemistically called 'General Issues'. Two decades earlier their predecessors, responsible for the 1978 APA Task Force Report had reported studies finding higher rates, of 6, 8, 30 and 80 deaths per 10,000 patients (APA, 1978, p.77).

Despite their being no supporting evidence for the 2001 claim, it was repeated, verbatim, three years later, in a report by the Royal College of Psychiatrists [RCP] in the United Kingdom (Benbow, 2004). It has since been regurgitated, always without supporting evidence, in textbooks and reviews (McGuffin & Farmer, 2008; Singhai, 2011) and patient information leaflets (Harrop, Read, Geekie, & Renton, 2021; Read, Morrison, & Harrop, 2023). The only figures about deaths in the important 154-page report by the Food and Drug Administration [FDA] in 2011 were, yet again, 'mortality rate of 1:10,000 patients, or 1:80,000 treatments' (FDA, 2011, p.70).

The APA and FDA reports also claimed that the ECT death rate is about the same as that associated with general anaesthesia for minor surgery. This ignores the rather obvious, but inconvenient, fact that even if this were true for an individual administration of ECT, the risk to ECT recipients is significantly greater than the risk involved with undergoing minor surgery because they typically receive between six and 12 treatments.

Some sources of information, including some patient leaflets in the UK (Harrop et al., 2021; Read et al., 2023) and the 380-page book 'Shock Therapy' (Shorter & Health, 2007) make no mention whatsoever of the risk of death from ECT.

Numerous studies show that the claim of the APA, RCP and FDA is a gross underestimate. Some of these studies were published well before the APA announced their one per 10,000 assertion, but were ignored. For example, as early as 1957 a report of 254 deaths caused by ECT had calculated a death rate of one per 1,000 patients (Impastato, 1957). This is ten times greater than the official line. The study had found an even higher rate among people over 60, of one in 200 people. The following year, a Norwegian survey found that three of 893 women ECT recipients had died as a result of the treatment, which is one in 298 (Strensrud, 1958). A 1978 review of 28 articles in which psychiatrists had spontaneously self-reported ECT-related deaths found 90 ECT-related deaths out of 130,216 patients, one death per 1,447 people (Frank, 1978).

A 1980 survey by the RCP asked British psychiatrists to report ECT-related deaths (Pippard & Ellam, 1981). Even excluding the six deaths that occurred more than 72 hours after treatment but within a few weeks, there were four deaths in 2,594 patients. That is one per 648.5 people. Of 2,279 ECT recipients at the Mayo Clinic in Minnesota, 18 (one in 144) died (Nuttall et al., 2004). The researchers argued that none of the 18 deaths were 'related to ECT', even though all 18 deaths had occurred within 30 days of ECT, and one was a stroke and another a cardiac arrest (within two days of ECT). Including only these two deaths, and excluding all the other 16, the death rate is one per 1,140.

Of 8,148 ECT recipients in Texas, seven died within two days of their last ECT. Even excluding the two that the researchers believed were 'unlikely to have been related to ECT' this is a rate of one per 1,630 people. If one includes the additional eight that died, from

'cardiac events' in the following 12 days, the rate is one per 627 (Shiwach, Reid, & Carmody, 2001).

All these studies finding rates far higher than the official position had relied on psychiatrists reporting deaths from a treatment they had prescribed themselves, a methodology open to understandable minimisation of mortality rates. To the best of my knowledge there are only two studies that are not dependent on psychiatrists acknowledging that their procedure has had a fatal outcome. The first is indubitably more objective. The researchers tried to contact 183 patients a year after ECT, to interview them. Twelve were dead (Freeman & Kendell, 1980). Four had killed themselves. In 6 cases 'death appeared to have been from causes entirely unrelated to ECT'. However:

'A 69 year old woman died 24 hours after her thirteenth treatment. Post mortem showed a myocardial infarction. She had had one previous infarct. A 76 year old woman also died 48 hours after her thirteenth ECT. Post-mortem showed a myocardial infarction 24-48 hours old.'

Including only these two deaths the rate was one per 92 people.

The other study was unique in that it was conducted by a profession other than psychiatrists. The anesthetists conducting the study found 'potentially life threatening' complications in 12 of 75 ECT recipients (16%). One had died, just four days after ECT (Tecoult & Nathan, 2001).

A 'systematic review' of 15 ECT mortality studies, again all relying on psychiatrists' self-reporting, estimated a rate of 2.1 per 100,000 individual treatments (Tørring, Sanghani, Petrides, Kellner, & Østergaard, 2017). This is only 1.7 times higher than the official line of 1 per 80,000 treatments. The review, however, excluded five of the eight studies summarised above and uncritically accepted researchers' opinions about deaths after ECT not being caused by ECT, including all 18 deaths (between one and 30 days after ECT) in the Mayo

Clinic study (Nuttall et al., 2004). A subsequent, more inclusive review, of 43 ECT studies with mortality data, including more than 75,000 patients, calculated that 'All-cause mortality was 0.42 deaths per 1,000 patients' (Duma et al., 2019). This is one per 2,380 people, more than four times greater than the official position of one per 10,000.

EARLY CARDIAC STUDIES

This paper seeks to demonstrate that a leading cause of all these deaths is heart failure. It is worth noting, therefore, that heart related deaths have been recorded from the earliest days of ECT. An early review (Alpers, 1946) noted numerous cases, including:

"....a patient of 57 years who received 13 electrical shock treatments (85 volts and 900 m.a. for 0.15 seconds) and who died one and a half hours following the last treatment. The heart showed a soft moist discolored area in the upper part of the anterior wall and the interventricular septum, and calcified plagues in the left coronary artery. Levy reports brain hemorrhages in a patient who died of heart failure after electrical shock treatment. There were a considerable number of dilated capillaries with hemorrhages which undoubtedly antedated the acute myocardial failure. . . . Attention to the role of circulatory failure in death from electrical shock treatment was directed by Jetter who reported death in three cases following shock treatment. His first patient was a man of 61 who died in 12 minutes following his eighth shock treatment. The heart revealed extensive obliterating coronary arteriosclerosis, a recent myocardial infarct, and hypertrophy and dilatation..... The second case concerned a patient of 70 years who died 12 minutes after the sixth treatment. The heart revealed obliterating coronary sclerosis, an old myocardial infarct, and hypertrophy and dilatation.The third case concerned a young subject of 23 ... with death ensuing about twelve hours after the eighth shock. Necropsy revealed severe pulmonary edema, an acutely dilated heart, acute diffuse glomerulonephritis and acute hyperemia of the brain. The death in Jetter's cases was attributed to heart failure. (pp. 365,366).

The reviewer concluded that 'Experience has shown amply that it is not a cause of death by virtue of brain damage, and that where death occurs it is usually the result of cardiovascular disease.' (p. 369).

A subsequent report of four autopsies of patients who died following electroshock treatment found that 'Only 1 case could be considered a cerebral death, the other 3 dying of cardiac failure' (Madow, 1956).

A 2019 REVIEW OF 'MAJOR ADVERSE CARDIAC EVENTS AND MORTALITY'

By 2019 a review by Duma and colleagues identified 82 studies of 'Major Adverse Cardiac Events and Mortality Associated with Electroconvulsive Therapy', involving 106,569 patients and 786,995 individual administrations of ECT. The reviewers state that 'the brief, yet intense, hemodynamic stress caused by seizure induction during ECT may increase the risk of cardiovascular events, especially in patients with pre-existing cardiovascular conditions' (p. 84). The six 'major adverse cardiac events' included in the review were: 'myocardial infarction, life-threatening arrhythmia, acute pulmonary edema, pulmonary embolism, acute heart failure and cardiac arrest'. [19] The reviewers estimated the pooled probabilities of each of the six events 'using two different methods that were considered equally appropriate for a meta-analysis of rare or zero events studies', the method of DerSimonian and Laird and the Poisson modelling approach. The current paper uses the former, which Duma et al. themselves selected for their Abstract and Discussion).

The review's Abstract reported that 'Major adverse cardiac events and death after ECT are infrequent and occur in about one in 50 patients'. The notion that one in 50 people experiencing major cardiac events is 'infrequent' would be unacceptable in any other setting. Bias is further evident when we discover that the one in 50 figure is, itself, a

misrepresentation of the review's actual findings and significantly underestimates the actual probability of a major cardiac event that the reviewers found.

Firstly, the rates per 1,000 patients of the two most commonly reported cardiac events were 24.0, for acute heart failure, and 25.83, for life-threatening arrhythmia. This represents one in 41.7 people and one in 38.7 people, respectively. Yet these rates somehow become 'about one in 50' in the summary provided by the Abstract. The misrepresentation is repeated in the first line of the Discussion section of the paper:

'Results of this systematic review and meta-analysis show that an estimated 25.83 [14.83-45.00] per 1,000 patients (approximately one in 50 patients) develop major adverse cardiac events after ECT (2%)' (p. 87).

The 'infrequent' term used in the Abstract is paralleled by the equally minimizing term 'low frequency' in the Discussion section (p.88.)' The paper ends by repeating, again, both the numerical misrepresentation of the actual findings and a minimizing descriptor:

'In conclusion, this systematic review and meta-analysis show that major adverse cardiac events after ECT are infrequent and occur in about one in 50 patients. (p.88)

Secondly, estimating the probability of suffering a major event on the probability of just one of the six events, the most frequent, is misleading. To estimate the actual probability of suffering at least one of any of the six events one must, according to the 'additive law of probability' add together the probabilities of each of the events (Howell, 1992). Table 1 shows these probabilities. Adding the six probabilities together comes to 65.47 events per 1,000 people, or one in 15.3. This figure, however, relies on the six events being independent of each other, which is very probably not the case. Since we do not know the rate of co-occurrence of all the possible pairs of the six events we can only conclude that the probability of experiencing at least one of the six is somewhere between one in 38.7 and one in 15.3 people.

Table 1. Incidence of each of six Major Adverse Cardiac Events in Duma et al., 2019

Major Adverse Cardiac Event	Events/Patients	Incidence per	
		1,000 patients*	
myocardial infarction	12 / 3,827	6.10	1:164
life-threatening arrhythmia	146 / 7.754	25.83	1:39
acute pulmonary edema	7 / 1,783	4.92	1:203
pulmonary embolism	1 / 1,477	0.69	1:1,449
acute heart failure	9 / 375	24.0	1:42
cardiac arrest	56 / 51,291	4.23	1:236
Combined probability#		65.47	1:15

^{*}using the DerSimonian & Laird random effects model (Table 1, Duma et al.)

We have already noted that the review also found that the all-cause mortality rate was 0.42 deaths per 1,000 patents. The review calculated that 'Cardiac death accounted for 29% (23 of 79 deaths) of deaths'. The review does not document the frequency of other causes of ECT related deaths. The Texas study had found that the most frequent causes of death within 14 days of ECT were cardiac events (33%) followed by suicide (27%) (Shiwach et al., 2001).

The 2019 Duma et al. review informs us that:

'To determine whether the risk of cardiac events after ECT may be higher in patients with pre-existing cardiovascular disease, we performed several subgroup analyses that were restricted to patients with (or without) known cardiovascular disease (Tables 3 and 4)' (p. 87).

These findings and tables are not referred to again in the paper. Perhaps this is because the tables do not show that cardiac events or deaths occur disproportionately or exclusively in people with pre-existing heart conditions. No studies had compared people with and without

[#] assuming that the six events are independent of each other

pre-existing cardiovascular disease in terms of cardiac deaths. Arrythmias were found in 30 per 1,000 patients with, and 31 per 1,000 patients without, pre-existing cardiac disease. The only other cardiac event studied in this way was cardiac arrest. One small study had found no arrests in the ten people with cardiac disease and four arrests in the 13 people without cardiac disease.

The reviewers highlight two studies:

'In two prospective cohort studies, Duma and colleagues and Martinez and colleagues showed that in about 5 - 10% of ECT treatments, patients develop cardiac troponin elevation, which indicates myocardial cell damage.' (p. 88).

The first of these studies found evidence of myocardial cell damage in eight of 100 patients (Duma et al., 2017). It also found that tachycardia and/or elevated systolic blood pressure developed after 'approximately two thirds' of the individual administrations of ECT. In 17% of the administrations systolic blood pressure surpassed 200. Two of the 100 patients suffered myocardial infarction directly following ECT. Despite these alarming findings Duma and colleagues announced that 'In the overwhelming majority of patients, ECT appears to be safe from a cardiac standpoint' (p. 650). The other study had found the same signs of cell damage in eight out of 70 patients, four of whom had shown elevated cardiac troponin prior to ECT (Martinez, Rasmussen, Mueller, & Jaffe, 2011). Two of the eight with elevated troponin, but none of the 92 other patients, died within three months of their last ECT. Rather than adopt a minimizing approach the researchers subtitled their paper 'The need for caution' and concluded: 'These data suggest that additional scrutiny both in the screening and follow-up of patients undergoing electroconvulsive therapy may be advisable' (p. 231).

Despite the conclusion by Duma et al. that major adverse cardiac events are 'infrequent,' their figures indicate these adverse outcomes are a leading, if not the leading, cause of ECT-related mortality

UPDATE OF THE 2019 REVIEW

Turning to studies since the 2019 review by Duma and colleagues, the author conducted a search of the Medline and PsycInfo data bases for 2019 – 2024, using 'electroconvulsive therapy' and 'cardiac events', and the six specific examples thereof used in Duma's review, plus elevated/high blood pressure. The search produced 80 potentially relevant publications. There were, however, only five studies reporting on cardiac events during ECT or within 30 days thereof, and providing some numerical data.

First, a study using Danish national registers found that, among depressed patients with no pre-existing 'somatic comorbidity,' those who received ECT were 3.7 time more likely to suffer adverse cardiac events than those who did not receive ECT (Osler, Rozing, Jorgensen, & Jorgensen, 2022).

In a Polish study of 65 ECT patients aged 65 or older, 15 (23%) suffered arrhythmia and 36 (55%) experienced increased blood pressure (Antosik-Wójcińska, et al., 2022). Of the four patients whose ECT was stopped because the adverse effects were so severe one was because of extreme increase in blood pressure and another was because of arrhythmia. The researchers concluded:

'Due to the fact that the cause of death and serious complications are most often cardiovascular complications, elderly patients are a population in which perioperative risk is increased' (p. 778).

A study of 98 ECT patients in the Netherlands found that only 3% of those aged 15-25 years suffered arrhythmia, compared to 15% of those older than 25 (Scheepens & Lok, 2023). Three percent is extremely high for teenagers and young adults, and the 15% finding is six times greater than Duma et al.'s 2.5% estimate.

A new 39-item checklist for short-term (48 hours) adverse effects of ECT was validated on 104 patients in India. Only one of Duma et al.'s six cardiac events, arrhythmia, was included. No cases of arrhythmia were found. However, 90% had significantly elevated blood pressure 'soon after the ECT-induced seizures' and 36% 'had persisting elevation of blood pressure during the early recovery period'. Even without including the other five cardiac events, 'Cardiovascular and musculoskeletal systems displayed the highest incidence' (Uppinkudru et al., 2024).

The most detailed data came from a Swedish register-based study. Of 20,225 ECT patients 123 (0.61%) died of 'medical causes' within 30 days of their last ECT (Lindblad, Nordenskjöld, Otterbeck, & Nordenskjöld, 2023). This is one death per 164 people. At one per 413 patients, the 49 cardiac-related deaths represented the most common medical cause of post-ECT death (40%). This was surpassed only by the 93 suicides (one per 217). Here are some of the details:

A total of 15 patients died of cardiac arrest within 30 days, 4 patients died within 1 day, 4 patients died at 2–7 days, and 7 patients died at 8–30 days. Myocardial infarction as a cause of cardiovascular death affected 4 patients within 30 days, 2 patients died within 1 day and 2 patients died at 8–30 days. Of the patients who died of pulmonary embolism, 3 patients died within 1 day, 2 died within 2–7 days, and 1 died at 8–30 days. (p. 529)

In total, 15 of the 23 patients who died within 24 hours of ECT were cardiovascular deaths.

The researchers rightly point out that:

Real-life observational studies based on registry data may demonstrate associations, but cannot determine causality. If medical records had been available, we would be better able to determine if deaths were due to the ECT, anesthesia, pre-existing medical conditions, or the mental disorder.

It is less clear, however, whether they are right to claim that 'ECT appears to be a low-risk medical procedure.'

Although it provides no new data, a 2022 commentary is quoted here because it is informative and offers some practical suggestions:

Electroconvulsive therapy causes periprocedural hemodynamic variability because of the surges in parasympathetic and sympathetic nervous systems after the administration of the electrical charge. Patients experience an increase in cardiac workload, which is potentially dangerous for patients with preexisting heart disease. ... Most major complications caused by ECT are related to the cardiovascular system; however, with an appropriate pre-ECT evaluation and a comprehensive multidisciplinary team approach, the cardiovascular complications can be well managed and minimized. Providing proper cardiac clearance can prevent cardiac complications and provide timely care to treatment-resistant populations who are at risk for excessive morbidity and suicide. (Hermida et al., 2022; p 2)

While it intuitively makes sense to be particularly cautious with people with preexisting heart conditions, we should remember that the 2019 Duma et al. review found no evidence that cardiac events are any less common in patients *without* pre-existing conditions.

We should also note that providing thorough cardiology assessments on all patients prior to ECT may prove difficult to implement because it would require suitably qualified staff. It is not a case of simply having technicians run ECGs and related tests, as psychiatrists may not be qualified to read them.

Asystole and bradycardia

My literature search also revealed a recent review of studies of two other heart problems:

The electrical stimulus and the subsequent seizure provoke significant hemodynamic changes. The stimulus causes a direct autonomic vagal nerve activation, which leads

to an initial parasympathetic cardiac response, evoking a brief period of heart rate reduction, hypotension, and sometimes a short asystole (Hartnett, Rex, & Sienaert, 2023).

The review by Hartnett and colleagues was concerned with the effects of electrode placement on incidences of bradycardia (slowed heart rate) and asystole (failure of the heart's electrical system, causing the heart to stop pumping) rather than total incidences. For example, it found rates of asystole, for unilateral electrode placement, of 48% and 63% of ECT sessions (Nagler, 2010; Stewart, Loo, MacPherson, & Hadzi-Pavlovic, 2011). Minimisation is present again. The reviewers state that unilateral ECT's 'association with bradycardia and asystole should not discourage its use' (Hartnett, Rex, & Sienaert, 2023). The researcher who found that 'Right unilateral EP was consistently linked with a slowing of heart rate and 63% of the 73 RUL sessions resulted in asystole' named their paper, published in the Journal of ECT, 'Absence of asystole during bifrontal stimulation in electroconvulsive therapy' (Nagler, 2010).

Other researchers, including prominent ECT champion Charles Kellner, acknowledge that "Acute post-stimulus bradycardia and asystole are common during ECT" and suggest that this is because the current diffuses down the trigeminal nerve into the brainstem, flooding cranial nerve roots and that this "massive irritation" of the trigeminal area triggers the trigeminocardiac reflex (Sartorius et al., 2022).

DISCUSSION

The findings of the 2019 review, and the subsequent five studies reviewed here, make it clear that cardiac events are not, as claimed by Duma et al., 'infrequent'. They are in fact a leading cause of ECT deaths, alongside suicide (which ECT is supposed to prevent). The three post-2019 studies reporting on arrhythmia found incidences of 0 (Uppinkudru et al., 2024), 7.1%

(Scheepens & Lok, 2023), and 23.1 % (Antosik-Wójcińska et al., 2022), producing a combined weighted average of 11.24% (30/267). This is far higher than the 2.58% calculated by Duma et al. (2019) and represents one in every nine ECT recipients. For arrhythmia alone, combining the findings of these three studies with the findings of the Duma et al. review (146 / 7,754), produces a rate of 3.33% (176 / 8,021), or one in every 30 patients,. Thus the risk of ECT patients having one or more of the six major cardiac events assessed by Duma et al. changes from 1:15 - 1:39 to 1:15 - 1:30 (even without including increased blood pressure, heart slowing and heart stopping/fatal asystole). This is what patients, and their family members, should be told.

Minimisation

The minimising language and calculations found in the Review by Duma et al. are paralleled throughout the ECT research literature and clinical practice. We have already seen that, like the cardiac events themselves, the mortality that can result from those events has been minimised for decades.

Most ECT research papers start with a firm statement that ECT is both safe and effective, often accompanied by a further claim that it is the *most* effective treatment for depression and/or is lifesaving. For example, the Duma et al. review starts with:

Electroconvulsive therapy ECT provides a potentially life-saving option for severe psychiatric conditions. Electroconvulsive therapy is generally considered safe.

The researchers who found that ECT patients were 3.7 times more likely to suffer cardiac events, start their paper thus:

Electroconvulsive therapy (ECT) has been established as the most effective treatment for severe unipolar and bipolar depression. ECT is generally considered safe, and short-term overall mortality of ECT is very low (Osler et al., 2022).

In England, reports from the Royal College of Psychiatry's ECT Accreditation Service (ECTAS) completely ignore cardiovascular effects (RCP, 2022b). A recent report from the equivalent organisation in Scotland does record 'cardiovascular complications' (a minimising term for adverse effects) (Langan-Martin et al., 2024). The report included 4,826 ECT episodes, from 2009 to 2019. Even using the biasing methodology of having the psychiatrists who prescribed or administered ECT assess for adverse effects, 2.2% of episodes resulted in 'cardiovascular complications' The average number of treatment episodes per patient was 9.6. Thus, the percentage of patients experiencing these 'complications' was somewhere between 2.2% (if all patients suffering cardiac events did so after all of their treatment episodes) to 21.1% (2.2 x 9.6) (if all patients suffering the events experienced them after only one of their treatment episodes). This range, which is actually between one in 45 and one in five people, was described as 'rare' by the lead researcher (European Psychiatric Association, 2024).

Minimisation of the adverse effects of ECT is most pervasive, however, in relation to the cognitive damage done by ECT, especially to memory (Fosse & Read, 2013; Read & Bentall, 2010; Read, Kirsch, & McGrath, 2019; Sackeim et al., 2007). Persistent loss of memories for life events prior to ECT (retrograde amnesia) have been found in between 12% (Sackeim et al., 2007) and 55% (Rose, Fleischmann, Wykes Leese, & Bindmann, 2003) of ECT patients.

Even the American Psychiatric Association (2001) has acknowledged that 'evidence has shown that ECT can result in persistent or permanent memory loss.' The ECT manufacturer, Somatics (2018), includes 'permanent brain damage' in its list of risks.

The UK's Royal College of Psychiatry's report on Scotland, mentioned above, found 'cognitive side effects' after 26.2% of treatment episodes. Given that patients received an average of 9.6 treatments, each with a one in four chance of cognitive side effects, it seems

remarkable that the report concluded, nevertheless, that ECT is a 'safe' procedure (Langam-Martin et al., 2024).

Similarly, recent researchers finding rates of memory deficits in more than two thirds of child and adolescent ECT patients have deemed their findings to be 'acceptable' (Chen et al., 2022) and to represent a 'high level of safety' (Li et al., 2023). Two psychiatrists and I wrote to the journal, and pointed out that:

The use of sufficient electricity, once, on a developing brain, in order to cause a seizure in that young brain, is a very serious matter. To do so five or six times in a few weeks is almost bound to cause brain damage in a significant number of recipients. The average age of these youngsters was just 15.5 years. Most (62 %) were girls. When even the psychiatrists themselves acknowledge that 69 % have impaired memories as a result, this does not indicate a treatment with a 'high' degree of safety. (Read, Ross, & Timimi, 2024).

Nine psychiatrists, including leading ECT advocate Richard Weiner, and the National Network of Depression Centers, replied to our letter. They argued that the 69% memory impairment rate is 'not a valid representation of cognitive effects and should not be viewed as clinically meaningful, as was subsequently claimed by Read (2024)' because, they argue, it was based on 'investigator-driven medical record review without any operational definition' (Ghaziuddin et al., 2024). If the clinical records of the clinicians caring for someone are not 'clinically meaningful', what is? Furthermore, records written by the staff responsible for administering a treatment are more likely to minimise damage done by that treatment, rather than overestimate it, as Ghaziuddin, Weiner and colleagues seem to be implying.

The information leaflets of 64% of ECT clinics in England minimise either the severity or prevalence of memory loss (Harrop et al., 2021), as do 83% of the clinics in the

rest of the UK (Read et al., 2023), and the current leaflet of the UK's Royal College of Psychiatrists (RCP, 2022a; Read et al., 2023).

In May 2024 the Journal of ECT published a paper entitled 'What Is Brain Damage and Does Electroconvulsive Therapy Cause It?'.It concluded that:

Claims that ECT causes "brain damage" or brain injury are not consistent with scientific understanding or knowledge, and dissemination of such claims amounts to misleading provocation of fear and alarm.

The author, Conrad Swartz, is described in the paper as 'a member of Somatics LLC, a manufacturer of ECT devices and supplies'. He is actually the co-owner of Somatics. His company was the object, in 2023, of the 'first lawsuit involving severe personal injury allegations against an electroconvulsive therapy (ECT) device manufacturer to proceed to trial' (Wisner Baum, 2023). The lawyers for the plaintiff'report that:.

Plaintiff Jeffrey Thelen alleged in his lawsuit that Somatics failed to adequately warn about the known risks associated with its ECT machines, including brain damage, severe permanent memory loss, permanent neurocognitive injuries, and others. After seven days of trial proceedings, the jury in Jeffrey Thelen v. Somatics, LLC found that Somatics failed to warn about the risks associated with its ECT devices. (Wisner Baum, 2023)

Meanwhile the efficacy of ECT is routinely exaggerated. Oft repeated claims that it is an effective, or even *the most* effective, treatment for depression ignore the facts that there have been no placebo-controlled studies since 1985, and that the 11 conducted forty or more years ago were small and flawed and produced very mixed results (Read & Bentall, 2010; Read et al., 2019. There has never been a single study finding that ECT has any benefit for depression, compared to placebo, beyond the end of the treatment period (Read & Arnold, 2017; Read et al., 2019; UK ECT Review Group, 2003). There *have* been three relatively

recent placebo studies with people diagnosed with 'schizophrenia', none of which found any benefit from ECT, short-term or long-term. (Melzer-Ribeiro et al., 2023; Sarita & Janakiramaiah, 1998; Ukpong, Makanjuola, & Morakinyo, 2002).

In some countries with unusually high rates of ECT, such as Australia and the USA, financial gain may be outweighing the very poor cost-benefit ratio of this treatment (McLaren, 2017).

Informed consent

The obligation to ensure informed consent is a core ethical principle of all health and mental health professionals. The World Psychiatric Associations Code of Ethics succinctly states:

In pursuing informed consent, psychiatrists should offer patients accurate information about their diagnoses, proposed treatments, risks, potential benefits and alternatives (WPA, 2020; p. 4)

There is very little research about whether ECT psychiatrists are adhering to the ethical principle of informed consent, either in general or specifically in relation to cardiac events and deaths. An audit of the patient information leaflets of 36 ECT clinics in England found that only six leaflets (17%) mentioned the risk of cardiovascular events following ECT (Read et al., 2021). Furthermore, only two (6%) acknowledged the risk of mortality without adding minimizing statements (e.g., comparing to safety of general anaesthetic or "minor surgical procedures"). Only seven (19%) acknowledged that ECT has a higher risk than one general anaesthetic because it involves about ten treatments. Most clinics (28; 78%) made unevidenced claims of very low mortality rates (e.g., '1:10,000 patients').

The corresponding percentages in a subsequent audit of 23 ECT clinics in Northern Ireland, Scotland and Wales were: cardiovascular risks – 22%; mortality risk – 9%; false claims of very low mortality rate – 43%; risk from multiple general anaesthetics – 9%.^[7].

One of the two U.S.A. manufacturers of ECT machines clearly acknowledges: Other serious adverse events have occurred, including ... cardiac complications, including arrhythmia, ischemia/infarction (i.e., heart attack), acute hypertension, hypotension, and stroke (Somatics, 2018).

Nevertheless, neither the information leaflet provided by the UK's Royal College of Psychiatrists (2022a) nor information for the public on the website of the American Psychiatric Association (APA, 2024) mention cardiovascular risks at all.

Meanwhile 2023 joint guidance by the World Health Organisation and the United Nations states:

'People being offered ECT should be made aware of all its risks and potential shortand long-term harmful effects, such as memory loss and brain damage. ECT is not recommended for children, and this should be prohibited through legislation.' (p. 53)

Limitations

A full search, rather than just for the five years since the Duma et al. review, might have revealed more papers than those found by Duma et al. Multiple reviewers might also have found more papers.

Recommendations

(i) Professional bodies should ensure their information and guidance regarding ECT is evidence-based.

- (ii) Psychiatrists providing ECT should adhere to the principle of informed consent and ensure they do not minimise adverse effects, including the high risk of cardiac events and small risk of cardiac-related death.
- (iii) Avoid, or at least minimise, the use of ECT with older people and those with cardiovascular conditions.
- (iv) ECT psychiatrists and their professional bodies should work together to start ensuring that patients damaged by ECT are offered rehabilitation and, where appropriate, compensation.

A Personal Anecdote

I recently published my worst clinical encounter with the issues discussed in this paper, 40 years ago:

At a staff meeting in my very first job as a clinical psychologist in the UK, I raised the issue of a man who had died on the ECT table the day before. I still recall the exact response of the psychiatrist: 'That is none of your business and I am personally insulted by your insinuation that we killed him.' When I pointed out that the man's notes included 'ECT contraindicated – serious heart condition', I was evicted from the meeting – physically. A colleague and I had copied that page of the notes, accurately predicting that it would quickly be removed from the file. I tried for two years to get the hospital, professional and governmental authorities to investigate. I failed. (Read, 2021).

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