26 January 2006

Charles Barnett
CEO and President
Daughters of Charity Health Services of Austin

Dear Mr. Barnett,

In your position as CEO and President I am sure you are very concerned about the safety and effectiveness of any treatment offered at your institution. You will be interested in a few highlights from the scientific literature addressing the tremendous controversy of electroshock treatment. I have summarized below just a few medical articles addressing ECT safety issues.

I strongly urge you to use this data to inform your considerations about the future of electroshock at your institution.

Respectfully submitted,

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Even proponents admit that the safety of ECT is not established.

ECT proponent C. Edward Coffey published a study in Archives of General Psychiatry describing 35 ECT patients demonstrating that eight had new changes on MRI after shock. That's 22%, or greater than one in five, with anatomic brain effects. Among those with the brain changes, one patient suffered a stroke and two had new abnormal neurologic signs on exam within six months of the ECT.¹

In a guest editorial in the 2003 Journal of ECT Coffey made these statements:²

Re: safety of ECT, Coffey admits: “...we lack firm data on the risk of major morbidity...”

and

“Again, we lack consistent data on the variation in ECT safety outcomes in this country...”

Re: effectiveness of ECT, Coffey raises unanswered questions: “What do we know about the general effectiveness of ECT in the community? How much of this care is based on evidence?”

And Coffey concludes: “In summary, although we lack definitive information on many of these dimensions of ideal care, the available data and our experience suggest a system of ECT care that is far from perfect.”

Serious scientific concerns about ECT are described in an article in Neuropsychology Review,³ in which the author concludes: “...there are still some findings that raise questions about safety.”

There is abundant scientific documentation of ECT-induced brain damage.

A study in Archives of General Psychiatry⁴ documented that cerebral atrophy (brain shrinkage) was significantly more common in those patients who had ever received electroshock therapy.

Another brain imaging study reported in Acta Psychiatraca Scandinavia⁵ confirmed that brain shrinkage was significantly more common in ECT recipients.

Archives of General Psychiatry⁶ reported that MRI scans demonstrate a strong correlation between the number of previous ECT treatments to loss of brain tissue.

A study appearing in Psychological Medicine⁷ found that ECT recipients were twice as likely to have a measurable loss of brain tissue in the front area of the brain and a tripling of the incidence of a loss of brain tissue in the back of the brain. The authors’ state:
“Most significantly, the brain abnormalities correlated only with ECT, and not with the age, alcohol use, gender, family history of mental illness, age at the time of psychiatric diagnosis, or severity of mental illness.”

Neurology\textsuperscript{8} published a review of the literature on the well-known ECT complication of epilepsy. Scientists concluded:

“The age-adjusted incidence of new seizures after ECT was fivefold greater than the incidence found in the non-psychiatric population.”

Animal studies provide more evidence of ECT-induced brain damage. An animal study reported in A Experimental Neurology\textsuperscript{9} documents definite changes in the brain caused by electroconvulsive shock, changes that can’t be seen by the naked eye or by routine scans, but are appreciated using specialized tissue labeling and detection techniques. Electroshock caused loss of brain cells, the birth of new brain cells that are not necessarily integrated into existing brain structure, and introduces bizarre electrical activity into existing cells.

An article in Brain Research\textsuperscript{10} found that repeated seizures from electroconvulsive shock in rats caused long lasting and functionally significant brain changes even in the absence of structural damage that would be visible on routine microscopic exam. They found that shocks cause the sprouting of fibers that develop abnormal connections with other neurons, possibly explaining why epilepsy tends to develop.

Over twenty years ago, it was reported in Science\textsuperscript{11} that ECT disrupts the production of protective protein by brain cells. More recent studies reported in the Journal of Biological Chemistry\textsuperscript{12} show that electric shocks to the brain also cause an increase in inflammatory proteins inside brain cells.

An animal study\textsuperscript{13} reported no difference in the brain damaging effects of ECT-induced seizures when the subjects were treated with oxygen and vitamins, thereby disproving the claim that modern ECT methods (complete with anesthesia and oxygen) are any less damaging to the brain than uncontrolled seizures.

Memory loss is the most common effect of ECT.

The medical literature includes abundant documentation of the memory-impairing effects of ECT. Memory researchers and experts in ECT have made these observations:

In an article in British Journal of Psychiatry\textsuperscript{14} scientists document significant memory deficits in 42 patients followed three months after initial ECT and found the memory loss to be the same as the effect of brain damage, stating:
“The two types of memory deficit appear to follow the pattern of
intellectual impairment associated with organic [physically apparent] cerebral lesions.”

A study reported in the American Journal of Psychiatry\textsuperscript{15} found that both unilateral and bilateral ECT gave memory impairment, but that memory loss after bilateral ECT was worse. The authors conclude: “... if electrodes are placed over the dominant hemisphere, memory is impaired in spite of a significant relief of depression.”

An article in the journal Neuropsychologia\textsuperscript{16} notes that long-term memory loss is seen in cases of unusually severe head injury. The author compares memory loss caused by ECT, and concludes:

“The present results indicate, quite clearly, however, that long term memory is vulnerable to the effects of ECT.”

The author further states that ECT is known to cause electrophysiological abnormalities in the temporal lobe of the brain, including areas known to be involved in learning. He speculates that the same structures are needed for recall of remote memory. A more recent article in Neuropsychologia\textsuperscript{17} likens the effect of ECT to the known memory-impairing effects of physical brain damage.

In an article in Behavioral Biology\textsuperscript{18} researchers state that memory deficits after ECT are very similar to the effect of alcoholic blackouts – well after recovery from the blackout, memory is still impaired. They conclude:

“ECT stimulation markedly impairs memory for events that immediately precede or follow its administration.”

A review of the electroshock literature published in Biological Psychiatry\textsuperscript{19} reiterates an earlier finding that 60% to 70% of patients reported persistent memory complaints six to nine months after ECT.

An updated study reports that 14 items on a memory questionnaire were significantly affected after ECT, including the most commonly reported deficit: ‘My ability to search through my mind and recall names or memories I know are there.’

The British Journal of Psychiatry\textsuperscript{20} reported significant memory impairment seven months after ECT. Recipients of bilateral ECT had poorer memory than those who had undergone unilateral ECT.

An article in British Journal of Psychiatry\textsuperscript{21} reported on 166 ECT patients interviewed six to eighteen months after ECT. Only 21% felt that they were given adequate information about the treatment ahead of time; 74% complained of memory problems; and 30% said their memories had never returned to normal.

Acta Psychiatrica Scandinavica\textsuperscript{22} provides a literature review on
the topic of the effect of ECT lead placement and choice of electrical impulse correlating to memory loss. The opening sentence establishes: “Electroconvulsive therapy produces both retrograde and anterograde memory impairment...”

There are 67 citations in the review, supporting the following conclusions:

- Amnesia increases with increasing number of treatments;
- Amnesia is worse with sine wave stimulus than with brief pulse stimulus;
- Slowing of brain waves (as measured by EEG) is more common as the number of treatments increases.

Original research reported in the *Journal of ECT* documents direct objective proof of the brain-damaging effects of ECT by showing that post-ECT cognitive deficits correlate with brain wave changes. Brain wave abnormalities not only occur in the part of the brain most affected by ECT, but also occur most often in the patients who suffer amnesia. An editorial in this same issue by the lead researcher on this study includes these statements:

“On the other hand, virtually all patients experience some degree of persistent and, likely, permanent retrograde amnesia. A series of recent studies demonstrates that retrograde amnesia is persistent, and that this long-term memory loss is substantially greater with bilateral than right unilateral ECT. ... It has also become clear that for rare patients the retrograde amnesia due to ECT can be profound, with the memory loss extending back years prior to receipt of the treatment.”

The author further states that there is little objective evidence to support the “belief” that memory loss is infrequent.

**REFERENCES:**


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