From: Critical Psychiatry Network list [mailto:CRITICAL-PSYCHIATRY@JISCMAIL.AC.UK] **On Behalf Of** Bernard Carroll
Sent: Friday, May 16, 2014 11:06 PM
To: CRITICAL-PSYCHIATRY@JISCMAIL.AC.UK
Subject: ECT thread

All, first please excuse the hiatus… I have been *hors de combat* for medical reasons this week – happily with a good outcome.

It seems that at least some CPN folks want this ECT thread to continue, so I will try to oblige. This post will discuss the issue of brain damage related to ECT. This post is a long one – but you asked for it. It is long because Peter Breggin has disseminated so much reckless dogma to suit his dire narrative of ECT and its horrid dangers. Other CPN members have raised some further issues, which I will think about for a future post. Sufficient for this day is the content thereof.

In this thread I already put paid to Peter Breggin's misinformation about cognitive side effects of ECT. The Sackeim report isn't what he says it is. Now he darts to the issue of brain damage caused by ECT. Okay, let's look at that.

Within this thread, Peter Breggin has asserted "*In very careful animal experiments, we see small hemorrhages and cell death*" (associated with ECT). He also stated "*ECT is closed-head injury caused by multiple traumatic effects of ECT (heat injury, electrical injury, breakdown of the blood brain barrier, exhaustion of neurons from extreme seizures,…*" He further mentioned *"…how ECT disconnects the frontal lobes in depressed patients…"* His major source for these claims – called "the most important study" on his website – is a 1952 report by Hans Hartelius from Sweden about the neuropathological effects of ECT in cats [Acta Psychiatr Neurol Scand. 1952;27(Suppl. 77):1-128].

Yesterday I did some homework reading Hartelius – all 128 pages of his monograph. Except for one key section, it is exemplary for its judicious balancing of the evidence and for its consideration of potential bias. Sadly, I cannot say the same for Peter Breggin's account of its findings – his characterization of Hartelius is just as biased and tendentious as was his characterization of Sackeim.

Regarding brain hemorrhages, Hartelius was crystal clear that hemorrhages are NOT caused by the ECT. Hartelius returned to this subject several times in his monograph. He concluded that hemorrhages occurred during the surgical process of extracting the brains from the cats, which were still alive at that time. They occurred in control and shocked animals. He opined that ECT made the shocked cats more likely than controls to develop these traumatic surgical hemorrhages, but he went no further than that. This clear and balanced statement was transformed into misinformation by Peter Breggin, who asserted that ECT causes brain hemorrhages. It doesn't. Period.

As for cell death, Peter Breggin again misrepresents what Hartelius reported. Here is Hartelius: In the frontal lobes, "…the nerve cell changes noted were slight and fairly infrequent. The majority of the nerve cells, even in those animals subjected to most intense treatment, exhibited no changes" (page 100). Finding possibly irreversible neuronal changes (shadow cells) was like finding a needle in a haystack – the proportions were not stated explicitly, but from the context it seems they numbered under 1% of neurons. For instance, only 18 such cells were seen in 3 frontal lobe regions across 31 cats that received ECT on very intensive schedules (3-4 ECTs daily at 2-hour intervals for up to 4 days). This represents examination of 93 frontal lobe region specimens, with hundreds of cells viewed in each section. Hartelius commented, "If the extremely large number of nerve cells examined

– several hundred in each specimen – is taken into account, the very small figures are remarkable." **Most importantly, there was *no difference* on this measure between control and ECT cats** (page 102). [Keep in mind that brain cells are constantly dying. Large numbers of them die during migration and differentiation, and we now are aware of neurogenesis and migration of neurons in the adult brain – something of which Hartelius in 1952 had no knowledge.] Hartelius goes on to state "True neuronophagia seemed to be still more rare. It was not observed in any of the control animals and could only be suspected on seven occasions [out of many hundreds of cells examined] in the animals subjected to ECT." Hartelius went on to state: "Single, small areas with *suspected* dropping out of cells were observed sporadically in animals subjected to larger series (11-16) of ECT's and with a longer survival time (group B**).** In only one specimen could this finding be considered as definite… It was a question of a few cells in 7 specimens out of the total 282examined…" (page 103).

On the basis of these findings involving either 7 dying cells out of unspecified hundreds or a few suspected but not definite cells in 7 specimens out of 282 or just a single definite observation in 282 specimens, and supported by no statistical analysis, Hartelius concluded that the question of irreversible neuron damage should be answered in the affirmative. He went on to qualify this conclusion as follows: "The changes found were not, however, extensive; they affected only a small minority of the nerve cells and occurred principally in those animals given the largest series of ECT's." Those animals were subjected to intensive ECT regimens – three to four treatments at 2-hour intervals daily for 3 to 4 days (11-16 ECTs total over 3-4 days)."With regard to the animals given less intensive treatment – i.e., 4 ECT's only [at 2-hour intervals on a single day] – it may be concluded that it was not possible to demonstrate any irreversible nerve cell damage of any consequence" (page 103).

The first question for us is how solid is the key finding? It is impossible to tell, because the results were described so vaguely and inconsistently. This is one area where Hartelius' rigorous presentation of the data was substandard. A hard-nosed statistician would say the key finding is not solid – the Fisher Exact 2-tailed probability on the one confident call out of 282 versus zero for the control group is 0.9999. The second question for us is how generalizable are these results anyway to the clinical setting today? The old regimens of regressive ECT or multiple monitored ECT are not in use today, so the ECT schedule (11-16 ECTs over 3-4 days) of the cats in which Hartelius found "true neuronophagia" is not comparable to today's clinical context. Considering the small effect size, there is no basis in Hartelius for Peter Breggin's assertion of *"disconnection of the frontal lobes"* resulting from the minor neuronal dropout reported.

In saying this I do not adopt a cavalier attitude towards loss of any neurons. At the same time, I do call out Peter Breggin for extrapolating his inference about functional significance well beyond what can be supported by the observed changes.

Turning now to the alleged heat injury caused in the brain by ECT, Hartelius once again contradicts Peter Breggin. What heat injury? Hartelius discusses this matter on page 106 as follows:"The quantity of electricity passing through the brain was therefore so small that it would only raise the temperature in it by at most 0.003° C. … this thermal effect… would never reach such a level at any site that it could be considered as a possible pathogenic factor in the neuropathological changes." So, this scare tactic by Peter Breggin is not based on solid science – or on any science. It is just another fabrication in service of Peter Breggin's dogmatic narrative.

Next we have Peter Breggin's claim of electrical injury. Once again, here is Hartelius (pages 23 and 106): "Broadly speaking, the existing experimental data warrant the conclusion that, with the doses of current applied in ECT, the current is distributed relatively evenly over the

whole brain, with a moderate increase in the direct path between the electrodes. In other words, the brain behaves as a relatively homogeneous conductor." And, "… the greater part of the current was received by the integument and only a small proportion – about 5 per cent – by the brain." Finally, "In view of the small quantity of energy, it does not appear reasonable to ascribe the neuropathological changes associated with ECT directly to the effect of the electric current." Once more, Peter Breggin's scare tactic has no foundation in Hartelius – whose work Breggin himself calls "the most important study."

Another claim by Peter Breggin in this thread is that *"exhaustion of neurons from extreme seizures"* occurs. Again, Hartelius disagrees: "Summing up the observations made in various physiological experiments, it may be stated that a considerable increase in neuronal activity, with concurrent relative hypoxaemia, takes place during the seizure.

"It nevertheless appears unlikely, on several grounds, that neuronal hyper-activity – either exclusively or mainly – could explain the neuropathological observations made in the present study. It is scarcely conceivable that only a minority of the nerve cells would take part in this activity, yet few of them exhibited changes. On the contrary, Toman *et a1*.[105] pointed out that all the neurons could be assumed to partake in the output of energy during the convulsions. Therefore, if this factor is to be assumed to contribute to the pathogenesis, it must reasonably only be in combination with some other factor and would then play only a minor ro1e" (pages 107-108). Here again, Peter Breggin's assertion is not supported by the primary source – quite the opposite, in fact.

Additional, nonspecific, pathological changes were described in the form of microvascular changes, glial reaction, increase of satellite cells, breakdown of the blood-brain barrier, and altered chromaffinity of nerve cells (increased in the nucleus and decreased in cytoplasm). Based on these pathologic features, Hartelius attempted an unbiased (i.e., blinded) global assessment of shocked versus not-shocked status in brain sections. These global judgments were only moderately accurate. The casewise accuracy for correctly recognizing that the cat had received ECT was 7 of 13 (54%) at 2-4 days. At 8 days the accuracy was only 1 of 9 (11%). He found the nonspecific changes most often in the period between 2 days and 4 days after the series of ECTs. By 8 days, the incidence of these changes decreased significantly: 54% (20/37) of frontal lobe specimens examined at 2-4 days were rated as having received ECT, and at 8 days only 26% (7/27) were so rated ($p < 0.025$) (page 47). Hartelius did not look past 8 days, so his data do not allow a statement of the permanence of these nonspecific pathologies. Clearly, however, they were resolving by 8 days. Moreover, in the hippocampus and cerebellum, these nonspecific pathological changes were even less obvious than in the frontal lobes, and control cats could not be distinguished from the ECT cats (page 76). Once again, the drastic picture suggested by Peter Breggin is not supported by the primary source. In particular, there is no basis here for the assertion of "permanent" brain damage resulting from even these highly intensive ECT schedules.

In summary, the 1952 report of Hartelius is described by Peter Breggin as "the most important" study of brain pathology following ECT. However, Dr. Breggin's interpretations of the Hartelius study either have no basis in the primary report or go well beyond permissible inference. In addition, the research design in respect of the ECT session schedule was inappropriate for today's clinical context, and the duration of follow-up was inadequate to address the question of permanent brain damage. The key reported finding in Hartelius of neuronal death after ECT is not supported by strong evidence; it has no statistical power; and it cannot support the strong inference claimed by Peter Breggin about ECT as a "brain-disabling" treatment.

Barney.