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Contents

Editorial
Weighing the Evidence ........................................................................................................ 3
Lonn Myronuk

Invited Article
Single Case Studies In Geriatric Psychiatry .............................................................. 4
Campbell Clark & Steve Holliday

Opinion
Optimising ECT for Elders ...................................................................................... 8
Peter Chan

Pharmacologia
Perfect Study– Imperfect Results ........................................................................... 10

President’s Report ..................................................................................................... 12
David Conn

Millennium Project
National Invitational Symposium on Mental Illness in Long Term Care Facilities for the Elderly ........................................................ 15
Shelly Haber & David Conn

Membership Report ................................................................................................. 16
Susan Lieff

Evidence Based Medicine
Modafinil: Evidence for use in anergia? ............................................................. 17
Rob van Reekum
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Dr. L. Myronuk
Editor, CAGP Bul.
1450 Waddington Rd.
Nanaimo, BC
V9S 4V9

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Editorial

Weighing the Evidence
The challenges of basing clinical practise on “the evidence”

LONN MYRONUK

This issue of the Bulletin expands the focus on evidence-based medicine, with an examination of limitations that conventional experimental designs pose for geriatric psychiatry. Dr. Herrmann’s examination of the results from a double-blind study on treatments for behavioural and psychological symptoms in dementia (BPSD) on pages 10-11 highlights the ways through which a seemingly well-designed and executed trial can produce conclusions that are “imperfect.” Dr. Herrmann puts these findings in context for us, and utilises his expertise to synthesise his closing opinion, which is that he does not agree with the conclusions of Teri and colleagues.

Is this the dismissal and suppression of facts in which Kuhn thought the scientific establishment would engage prior to an inevitable paradigm shift? I do not believe that it is. Rather, it is an acknowledgement that it is very difficult to arrive at simple, unequivocal conclusions about the complex behaviours of complex systems. Each person that we treat in our roles as geriatric psychiatrists is a complex system with a unique combination of diseases, treatments and idiosyncratic developmental experiences. The more rigorous the experimental designs are in attempting to control these variables, the less well the results can be generalised back to uncontrolled “real life” situations.

In their invited article on pages 4-7 of this issue, Clark and Holliday propose one possible means of addressing the generalisability-gap in everyday clinical practice. Rather than rely on the publication and dissemination of large, double-blind placebo-controlled trials that subsequently require review and synthesis (e.g., the Evidence-Based Medicine reviews of van Reekum and colleagues published in the Bulletin), they propose a systematic approach to observing, recording and sharing data on individual patients’ responses to intervention.

The value of Clark & Holliday’s approach is that individual treatment decisions are tested and either supported or refuted, without the logically dubious step of reverse-generalisation from group results to individuals. As governments and health care organisations struggle with the costs of their drug formularies, this approach of objectifying treatment response for individuals could become a powerful tool for ensuring the availability of treatments for those who experience their benefits. Evidence of a costly treatment having a positive impact on the expected course of a chronic disease in a person can be an eloquent argument for that patient to have the therapy remain available to her.

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The symptoms of Alzheimer’s disease are diverse and include cognitive and non-cognitive phenomena. For a number of reasons, cognitive symptoms and particularly memory function have been widely regarded as the core features of AD and have been the primary focus of treatment and evaluation. Clearly, other symptoms may be equally or more disturbing to the patient or their caregivers. For example, sleep disturbances, aggression, psychosis, and depression are much more likely to lead to acute hospitalisation and long-term institutional care than are memory loss, language dysfunction, loss of reasoning skills, and disturbances of praxis. Moreover, many of the currently available treatments may be effective in ameliorating some of these symptoms. The purposes of this paper are one, to describe potential disruptive symptoms often observed in a patient with AD and two, to suggest empirical/statistical methods for ascertaining if a given treatment is effective for a single case or a small number of cases.

I: SYMPTOMS OF AD: HISTORICAL AND CURRENT VIEWS

The first report of what we now know as Alzheimer’s disease contains information that is remarkably consistent with the current neuropsychiatric perspective on AD. That report identifies early paranoia, behavioural disintegration, delirium, hallucinosis, aggression, and disinhibition as symptoms of the disease together with the characteristic losses of cognition including short-term memory loss, disorientation, language dysfunction (paraphasia, loss of comprehension), disturbance of praxis. Notably, Alzheimer noted paranoid jealousy as the first obvious symptom with the cognitive symptoms appearing later in the disease.

From a historical perspective it is interesting to note that the early diagnostic guides for AD were consistent with this concept of global deterioration focusing on personality change, behavioural deterioration and cognitive loss as being important markers of the onset of AD (see for example DSM versions). By the mid-1980s however, the non-cognitive features were either removed or diminished in importance in all diagnostic systems so that it became possible to diagnose AD solely on the basis of cognitive symptoms. As one of us has argued, a similar situation occurred in the study of schizophrenia in the 1960s when the florid, positive symptoms were elevated in status above the negative symptoms. In that case, the result was a restricted treatment focus, which actually led to a decreased ability to fully treat this complex disorder.

In the late 1990s the non-cognitive features were re-introduced as central features of AD (see IPA reports on the non-cognitive features of dementia). While the broader view has not yet been fully represented in diagnostic criteria, current neuropsychiatric concepts of AD view depression, psychosis, agitation, sleep disturbance, emotional lability, disinhibition, and a variety of more specific problems as being expectable occurrences over the course of dementia. Although the work on the staging of these symptoms is incomplete, it is now abundantly clear that all patients with AD will exhibit both cognitive and non-cognitive symptoms throughout the dementing process.

The fact that clinical psychiatrists will carry the primary responsibility for treating/managing those symptoms is also abundantly clear. The careful monitoring of these symptoms is one of the central tasks of the clinician. To achieve success in this task the clinician must directly address several challenges. The first is to accept the idea that symptom variability is a central feature of
AD. While all patients exhibit both cognitive deterioration and non-cognitive changes, both the specific symptoms and the specific patterns of presentation vary widely. Second, the clinician must be prepared to adopt an assessment strategy suitable for properly monitoring and evaluating symptom change in individual clients. Third, the clinician must become knowledgeable in analytic procedures suitable for assessing the significance of observed changes in clients in both the presence and absence of treatment.

Our contention is that the traditional strategies for evaluating treatment effects in AD are more suitable for evaluating group effects than for monitoring individual response to treatment. For example, the Folstein MMSE and the ADAS-Cog, two of the most commonly used tools in diagnostic and treatment protocols, are not well suited for detecting individual treatment effects. This situation raises the question of what instruments and strategies are best suited for clinical evaluation. The following discussion focuses on some of the measurement issues that we feel are central to the problem of measuring both symptom presentation and treatment response in AD.

II: SELECTION OF MEASUREMENT INSTRUMENTS

As discussed above, many patients with AD have multiple symptoms that may interfere with their ability to cope with the demands of daily living. The first step in determining whether a particular intervention has a beneficial effect on a specific symptom set is to select an instrument for measuring the symptoms prior to and after intervention. One approach is to select a standardised test with known reliability and face validity. Knowledge of these properties allows one to compare the performance of a subject relative to a control group. Specifically, if the test/retest reliability and score stability over time are known then it is possible to apply a statistical test to determine if a 'significant' change has occurred for a single subject (see below for a description of the statistical method).

The property of face validity is simply whether the test measures the behaviour of interest. Given the variety of symptoms exhibited by AD patients, appropriate tests may be found in the psychiatric, neurolinguistic or geriatric literature. One cautionary note is that although a test may be very reliable, it should also be sensitive to change. Trivially, height in an adult is an obviously very reliable measure of a specific behaviour, record its frequency prior to treatment and repeat the measurement after treatment. For example, if the problem behaviour is

incontinence, one can measure the number of episodes per day (or week) and then apply an intervention strategy (e.g., taking the patient to the toilet on a fixed timetable) and determine if the number of episodes is substantially reduced. This approach has some advantages over the first approach. First, the measurement tool can be targeted at specific behaviours or symptoms unique to the patient. Often these behaviours are the most disruptive for the caregiver. Second, the outcome measure has inherent face validity and hence it may reinforce the caregiver to maintain the intervention. Finally, if the behaviour is measured a number of times before the intervention, the magnitude of the change can be estimated.

III: DESIGN AND POTENTIAL CONFOUNDS

Although null hypothesis testing and random assignment to treatment conditions are essential for a large scale clinical trial to have 'validity', this methodology has many drawbacks and limitations. First, the statistical tests employed usually compare group means or survival rates; the assumption being that each 'treated' subject will have an identical response to treatment (i.e., the general linear model). Such an assumption is tenable if the symptom of interest has a common aetiology. However, if one considers psychiatric symptoms such as depression or psychosis, the research studies clearly indicate differential responses to a specific drug treatment. Such findings suggest different aetiological causes may underlie the behaviour. Simply, for some subjects the treatments may fully ameliorate the symptoms while for others the drug may have little or no effect.

Second, although the actual treatment effect required for improvement varies dependent upon the sample size, the conventional level (p<0.05) for rejection of the null hypothesis (i.e. significance) is not truly compelling in terms of clinical efficacy. For example, in a two-group study with each group having twenty subjects, the treated group’s mean improvement must exceed the 68 percentile of the untreated group for a significant difference to be found. The size of this difference decreases as the sample size increases or if repeated measures designs are used (i.e. subject as their own control). Dependent upon the outcome measure, such changes may not be particularly compelling clinically. For example, in a study of depression, the majority of treated patients will still meet the criteria for major depressive disorder but on a more sensitive measure of depression (e.g. the Hamilton Depression Scale) the group will improve.

However, medicine has advanced often by using a single case design with replication. Perhaps the most dramatic recent example of this approach is the treatment of ulcers with antibiotics. Here, the investigator drank a so-
lution containing h. pylori bacteria, developed the early signs of an ulcer and then successfully treated himself with antibiotics. Although the original hypothesis being tested was consistent with laboratory findings and large-scale clinical trials did follow, the original clinical experiment was a simple single case study. Moreover, as the treatment did not require the development of a new drug and threatened the profitability of existing drug treatments, the idea was met with considerable resistance by vested interests. As well as illustrating the potential power of the single case methodology, this finding also highlights the current issues in today’s research environment that may constrain the development of potentially effective treatments.

The design of a single case study is simple. First, a target behaviour is selected (e.g. outbursts of temper and aggression) then an appropriate measurement instrument is selected and series of measurements are made (e.g. number of outbursts per day). Once a baseline is established, a treatment is applied. This treatment may be a drug therapy or a behavioural intervention but it must be consistent. After an appropriate period of treatment, the behaviour is re-assessed and compared to the baseline. As with any experimental design, a number of threats to the validity of such an experiment exist. For example, without a control group, one may argue that the patient may have improved naturally or some extraneous variable was responsible for the effect. In the case of aggression, a patient may have become friends with another patient or have had a dispute with a particular staff member who is no longer on the wards.

These threats to experimental validity or potential confounds may be eliminated or reduced in two ways. First, the investigator may end treatment and determine if the behaviour returns. For example, if a prescribed drug has a short half-life, one may allow the drug to clear and re-assess the patient’s behaviour. Such an approach is commonly used when assessing the effects of Ritalin on children with ADHD. Although such methods can be used in pharmacological interventions, caution should be exercised in behavioural interventions, as the unwanted behaviour could then become even more difficult to extinguish.

A second major limitation of this solution in a clinical setting is that the staff may not wish the problem behaviour to return and it may contradict a primary assumption of medicine, do no harm. This approach may be best described as a time series analysis on a single case. The second solution is to simply replicate the experiment with a patient with a similar problem (i.e. single case methodology with replication). If the initial result is replicated then one’s confidence in the treatment is increased while if it is not, one must address the question of why.

In conclusion, the overall appeal of the single subject design is that in essence it is consistent with good clinical practice, but more formal in terms of measurement, design and analysis. In particular, the treatment is targeted to a particular patient’s problems, not the diagnosis per se.

IV: STATISTICAL ANALYSIS

A: Standardised Tests

In 1957, Payne and Jones7 adapted the Student’s t-test to the single case methodology. They suggested that this test could be used to determine if a particular intervention had a significant effect on a patient’s behaviour. Given one has the appropriate standardisation data (i.e., Means, Standard Deviations for a test/retest format and the test/retest correlation), one can determine if an observed change is greater than one expect from chance variation alone. Specifically, one can use the standardisation sample as the control group. The test is as follows:

\[
Z_d = \frac{Z_{post} - Z_{pre}}{S_{post}^2 + S_{pre}^2 - 2rS_{pre}S_{post}}
\]

where:
1) \(S_{post}\) is the standard deviation of the standardisation sample on retest,
2) \(S_{pre}\) is the standard deviation of the standardisation sample on the initial assessment,
3) \(r\) is the test/retest correlation, and
4) \(Z_{pre}, Z_{post}\) are the subject’s scores minus the mean of the standardisation sample divided by the sample standard deviation at the respective time, either pre or post. Specifically, these are simple Z-scores for pre or post observations.

Once this calculation is done, one may simply consult a Probability Table for a Normal Distribution to determine the probability of a given \(Z\) occurring.

B: Non-Standardised Tests

If an appropriate standardised test is not available, then one can use the subject as his or her own control by estimating the degree of variation prior to the implementation of treatment. For example, one may measure the number of times a behaviour occurs per day over a one-week period. From these data, one can then calculate a mean or average number of occurrences per day and a standard deviation. Alone, these data provide insight into the magnitude/frequency of the problem and its consistency over time. Sometimes, the fact that a patient’s behaviour is highly inconsistent may suggest that the behaviour is situation-dependent and not a fixed trait of the
patient. For example, a colleague was asked to assess an ‘aggressive’ Huntington’s patient in a nursing home. After discussion with staff, it was determined that the aggressive behaviour occurred only when a nurse attempted to bathe the patient. The patient was simply uncomfortable being bathed by an unknown female. The patient agreed that his daughter could bathe him and the problem was resolved.

However, when the behaviour is consistent (e.g., the behaviour multiple times per day on a daily basis) then these data provide the basis for estimating the efficacy of a given intervention. One may simply compare the magnitude/frequency of the behaviour prior to and after treatment. Similarly, one may determine if the observed after-treatment value falls outside the normal range observed prior to treatment (Mean + 2SD).

**V: SHARING OF RESULTS AMONGST CLINICIANS**

If indeed one accepts the argument outlined above, one outcome will be a large increase in the number of formal case studies of AD. It is reasonable to anticipate a need to share the information gained in those case studies. By comparing results, for example, it may be possible to identify similarities within treatment situations (for example, a variety of aggressive behaviours may respond to a similar management strategy). Similarly, pooling treatment information may help clinicians to better understand the sequencing of symptoms and may consequently help the clinician to be better prepared to anticipate and intervene with problems as they emerge.

Unfortunately, there is not presently a forum in which such studies can be easily made available to colleagues. While most journals accept case studies, publication is largely limited to exceptional cases with preference given to reports of unanticipated negative treatment effects, paradoxical occurrences, and rare or atypical presentations of disorders.

The situation that arises if clinicians participate in single case studies is somewhat different. The need in this case is for a forum in which participants can both present results from “n-of-one” studies and review results obtained from colleagues who have dealt with similar types of problems in similar patients.

One solution that suggests itself is the use of the Internet as a medium for information exchange. The Canadian Academy of Geriatric Psychiatry has the capability to develop an information exchange forum using chat room or web page technology. Members could, for example, write up and post case studies, assigning each study to an appropriate category (aggression, depression, cognition, etc.) Other members could then review those cases to obtain information on effective treatment strategies and to provide feedback to the authors.

Such a strategy also allows for the creation of pooled data sets that can be used to make either direct comparisons across individuals receiving similar treatments or meta-analyses of treatments within symptom categories.

In summary, the careful use of single-subject data collection and analysis techniques can be useful in bringing some system and clarity of analysis to the treatment of AD. The methodology discussed is particularly useful when there is both symptom variability and a range of treatment options. The use of case study procedures in conjunction with the creation of an information forum can lead to both better case management and enhanced professional communication.

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Opinion

Optimising ECT for Elders
Implications of New Research on Technique and a Call for Training

PETER CHAN

Electroconvulsive Therapy (ECT) is a safe and effective treatment for certain psychiatric, and some medical, disorders in demented or non-demented elders. Recently published American Psychiatric Association (APA) Guidelines for ECT reflect primary and secondary indications for use of ECT, and consider its role in the elderly. Nevertheless, recent articles appearing since the APA guidelines have promoted discussion of how ECT technique can be optimised for the elderly. The explosion of research on ECT over the past decade and its impact on technique has led to various week-long training programs offered in the US, and the start of an annual “hands-on” day long course (with exam) as conducted by the Association for Convulsive Therapy (ACT).

Since Dr. Harold Sackeim and his colleagues debunked the two myths that “any seizure is a good seizure” and “the longer the (cumulative) seizure the better”, much research interest has been generated into how electrode placement, frequency, and EEG morphologies can used to maximise therapeutic response and minimise cognitive side effects. Most ECT research is not specifically targeted to an elderly population, but the average age in most studies tends to be older (age 50 and above). Two recent prospective studies looking at the efficacy of high dose right unilateral ECT have provided a call to consider using a dose of six times above seizure threshold, or 75% of the maximum energy of the ECT device.

Sackeim randomised 80 inpatients into four groups (mean age of 54 to 62 in the groups): 1.5 times threshold right unilateral, 2.5 times threshold right unilateral, 6 times threshold right unilateral, 2.5 times threshold bilateral. Respectively, response rates were 35%, 30%, 65%, and 65%. There were more cognitive side effects in the bilateral over all of the right unilateral groups at one week after stopping ECT (retrograde and anterograde amnesia) and at two months after stopping ECT (retrograde amnesia). Sixty-nine percent of non-responders to right unilateral ECT responded favourably after crossing over into bilateral ECT. The study’s main conclusion is that 6 times threshold right unilateral ECT is equivalent to efficacy of bilateral ECT, without as much risk of persistent or severe cognitive impairment.

McCall’s study excluded “extremely severely depressed” (actively suicidal or refusing food) patients. Seventy-two outpatients and inpatients were randomised into two groups: 2.25 times threshold right unilateral and fixed high-dose right unilateral (403 mC; 75% of maximum energy output on their Mecta device), after titrating initial seizure thresholds in both groups. The response rates were 39% and 67% respectfully. Those in the latter group were more likely to respond if they were 8 to 12 times over threshold, but “global cognitive disturbance” within two days of completing the ECT course was higher depending on how much the stimulus was above threshold (up to 8 to 12 times ECT threshold). Moreover, those in the fixed high-dose group had more problems with retrograde autobiographical recall during this time. The study’s conclusion is that there appears to be a dose-response effect of ECT, which holds true up to 12 times threshold for right unilateral.

One facet of these studies to note is that ECT is usually given three times weekly in the US. This is typical of other US studies where, presumably, utilisation of resources and managed care is more of an issue. Decreasing the frequency of treatments to twice weekly, which is considered comparable in efficacy to three times weekly but with a slightly slower response, is an option especially in those elderly individuals where there is the risk of cognitive impairment. Another facet to consider is that with the elderly generally having higher initial seizure thresholds, six times threshold could exceed the energy an ECT device can deliver, leading to a call by some to increase the maximum energy level that federal regulations have set forth.

Although both groups titrated to obtain initial seizure threshold at the first ECT session, the authors appear to differ in their recommendation whether “to titrate..."
or not to titrate”. Sackeim appears to favour titration at the first session and has a protocol developed for his “turbo-charged” investigational ECT device, while McCall appears to favour starting at a fixed dose. This mirrors the current debate within the ECT literature whether titration is necessary. Proponents indicate there is a 40- to 50-fold variation in initial seizure threshold amongst individuals, while detractors point out that this variation is much less so for most individuals. Only 10.5% of patients have an initial seizure threshold more than 168 mC, and aged-based methods can help estimate the initial stimulus energy.10

How can this debate apply to ECT for elders? Firstly, Sackeim suggests that aged-based estimates of threshold are fraught with inaccuracy, as age accounts for only up to 10% of the variability when determining initial seizure threshold. Secondly, we know that grossly exceeding the seizure threshold will lead to more risk for cognitive impairment, with the therapeutic window being even narrower for those with pre-existing dementia. Moreover, the APA guidelines and recent studies hint at the possibility of permanent retrograde amnesia occurring rarely. It is unclear how an excessive dose of electrical energy might relate to this risk, or whether this is more likely to occur in the elderly. Given the lack of clarity around the issue and the need to exercise caution with the elderly, we as ECT practitioners should strongly consider titrating initial seizure thresholds for most elderly patients.

The arguments against initial titration include a subconvulsive stimulus (or stimuli) leading to bradycardia or asystole, that it might require an additional dose of general anaesthetic, and that it would render the first treatment sub-optimal in effect. Slowing of the heart occurs only transiently most of the time, and pre-treatment with atropine or glycopyrrolate can counteract this. Up to four to five sub-convulsive stimulations for the titration sessions can be done as frequently as 20 seconds apart, so that this should add up to no more than 2 to 2 ½ minutes—a time frame that a regular dose of anaesthetic should adequately cover. It is debatable how effective a barely supra-threshold seizure is, but one needs to weigh the risk of an inefficacious treatment against the risks of cognitive impairment by not titrating at the outset. Certainly, if there is a lot riding on the first session (example: profound cachexia and dehydration, high suicidal risk, high medical risks with ECT), titration should be abandoned for these individuals. Thus, the reasons given all appear to be rationalisations and diverting from what I think is the primary reason why ECT practitioners may not choose to titrate: Titration should entail monitoring the EEG, and some practitioners are uncomfortable in setting up the EEG monitoring or interpreting the EEG data.

These and other reasons lead to a call for formal ECT training in Canada. The US-based courses are certainly considered “gold standard”, and based on my experience it is worth taking the ACT course. However, I think some ECT practitioners would consider these courses to be too expensive to take (average $1,000.00 to $2,000.00 US), too time consuming, or less of a priority compared to other courses in psychiatry. There should be a formalisation of an ideal training program developed within Canada, hopefully spearheaded by the Canadian Academy of Geriatric Psychiatry (CAGP) with the blessing of the Canadian Psychiatric Association (CPA). Since no such organisation such as ACT exists in Canada, there should be a group of individuals interested in this process who can guide the development of such a program and decide whether it is best to promote developing various local training programs, or a national program that could be run in conjunction with the annual CPA/CAGP Meeting.

Hopefully, all this will lead to less angst when considering whether “to titrate or not to titrate”, and make this less of a philosophical question but instead a scientific one to be answered.

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Over the years we have all developed approaches to treat behavioural and psychological symptoms in dementia (BPSD). Some of us have attempted to utilise an evidence-based approach even though evidence is often lacking for many of these interventions. For example, while we always espouse the use of non-pharmacological approaches, the evidence available for these approaches usually derive from very small, within-subject designs that are never replicated in large, randomised, controlled studies (Opie et al. 1999). There are also many pharmacological approaches recommended by expert panels that have little data to support their claims.

It was, therefore, with great interest and anticipation that we received the Alzheimer’s Disease Cooperative Studies Report on the treatment of agitation in Alzheimer’s disease (Teri et al., 2000). This randomised, placebo-controlled trial attempted once and for all to demonstrate which treatment is best for agitated behaviours: pharmacotherapy with an antipsychotic (haloperidol) or trazodone, or a non-pharmacological approach utilising behaviour management techniques (BMT). One hundred and forty-nine outpatients with AD who had at least a two week history of agitated behaviours were recruited. Patients were randomised to four arms (haloperidol, trazodone, BMT or placebo) for a sixteen week trial. Medication could be titrated slowly to a maximum of 3 mg per day for haloperidol or 300 mg per day for trazodone. BMT consisted of weekly or bi-weekly sessions with a trained, experienced therapist who provided education and strategies for managing agitated behaviours, utilising assignments and videotape training. The primary outcome was clinical global impression of change and secondary measures included a number of scales for behaviour, activities of daily living, cognition and caregiver burden.

Results suggested that only 34% of patients improved and, more surprisingly, that there was no difference among any of the three treatments or placebo! The final dose of haloperidol was 1.8 mg per day, and trazodone was 200 mg per day. There was a fairly high dropout rate (57/149) and, while there was no difference in the percentage of dropouts among the different arms, there were differences in the reasons for dropping out: increased agitation in the trazodone group, adverse effects in the haloperidol group, and caregiver difficulties in the BMT group. The secondary analyses revealed few differences but suggested that patients treated with trazodone had greater declines in their MMSE scores.

So what went wrong with this study? In an accompanying editorial (Herrmann and Black, 2000), I suggested a number of possible methodological difficulties. These included the fact that, on average, these outpatients were less impaired cognitively and less agitated than the patients in many of the recent atypical antipsychotic trials, which included severely impaired institutionalised patients. It is also possible that the dose of haloperidol was too low and the dose of trazodone was too high. A previous RCT with haloperidol demonstrated a dose response effect with doses of .5 to .75 mg per day being no more effective than placebo, but doses of 2 to 3 mg per day demonstrating significant benefit (Devenand et al. 1998). Similarly, in the only randomised, placebo-controlled trial of trazodone for BPSD, benefit was demonstrated with an average daily dose of 120 mg (Lawlor et al. 1994). It is also possible the results were affected by the high dropout rate of 38%. Of note, this rate is significantly higher than the dropout rates in the three large RCTs of atypical antipsychotics that have been recently published. Finally, it is also important to note that in these studies sample sizes of 200 to 600 patients were required to demonstrate...
statistically significant differences among treatments in-
cluding placebo.

I disagree with the conclusion of the study’s au-
thors who noted that these therapies have “marginal effi-
cacy for most patients”. I am convinced by the results of
the RCTs with the atypical APs and several other phar-
macological approaches reviewed previously in this col-
umn. I also believe (even in the absence of evidence) that
pharmacological approaches must be augmented with
environmental and behavioural approaches (or vice versa).
What I don’t argue with is the need for more studies such
as this that put our clinical assumptions to the test. It is
only with further research that we will be able to provide
our patients with truly rational evidence-based care.

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CLINICAL DIRECTOR, GERIATRIC PSYCHIATRY
PROGRAM

The Centre for Addiction and Mental Health is
seeking a Clinical Director for its Geriatric Psychiatry
Program. This is a clinical program that provides inpa-
tient, outpatient and community psychiatric and addic-
tions services to a geriatric population in Toronto.
CAMH, Canada’s largest academic health sciences cen-
tre devoted to mental illness and addictions, is fully af-
iliated with the University of Toronto. The Geriatric
Psychiatry Program is a component of the Geriatric Psy-
chiatry Division of the University of Toronto Depart-
ment of Psychiatry.

The Clinical Director is responsible for evolving
the Program into a treatment, education and research-
driven enterprise on a national and international level.
CAMH runs on a program management model and pro-
grams are co-led by a Clinical and an Administrative
Director. The Clinical Director is accountable for mod-
ellng excellent clinical practice, developing the Pro-
gram’s clinical vision, and providing clinical and aca-
demic leadership. In collaboration with the Administra-
tive Director, the Clinical Director will co-ordinate ma-
jor Program initiatives, develop Program policies and
procedures, direct resource allocation, and participate
in budget monitoring and development. The Clinical
Director will also take a leadership role in recruitment,
selection, orientation, and performance appraisal and
management of staff.

The Clinical Director is accountable to the Vice-
President, Clinical Programs, and to the Physician-in-
Chief. The Clinical Director is also accountable to the
Head of the Geriatric Psychiatry Division of the Univer-
sity of Toronto.

The successful candidate will possess an MD or
PhD in a relevant clinical discipline as well as licensure
with the appropriate college. He/she will have demon-
strated interest, experience, and leadership in relation to
this clinical population. Some management experience
in a complex health care setting is necessary. Eligibility
for an academic appointment at the University of To-
ronto is required.

Interested applicants should contact:

David S. Goldbloom, MD, FRCPC
Physician-in-Chief, Centre for Addiction and
Mental Health
Professor of Psychiatry, University of Toronto
250 College Street
Toronto, Ontario
Canada M5T 1R8
Tel: 416.979.6915
Fax: 416.979.6834
E-mail: david_goldbloom@camh.net
President’s Report

DAVID CONN

I hope that you all had a restful and enjoyable summer. I look forward to seeing many of you at our Annual Scientific Day in Montreal on November 15, just prior to the CPA meeting. Our plenary sessions will be simultaneously translated into English and French and we will have workshops in both languages. Many thanks again to the Organising Committee for their excellent work (Drs. Thorpe, Primeau and Rousseau).

It gives me great pleasure to congratulate Dr. Mark Rapoport, the first recipient of the new Lilly CAGP Fellowship. This award provides the fellow with $40,000 for one year of study. We would like to thank Lilly for their generous sponsorship of this program. The previous CAGP Fellowship has now been renamed the CAGP Resident Award. We look forward to meeting Dr. Rapoport and the recipients of the Resident Awards in Montreal.

I am pleased to let you know that our new website will be up and running shortly (details to follow). We look forward to your feedback and ideas.

The plans for a National Invitational Symposium on Mental Health Services in Long-Term Care Facilities are progressing well. We have obtained enough sponsorship funding from industry to support the meeting. It will be held in Toronto on April 28 and 29 next year. Please see details of the meeting in this edition of the Bulletin.

I would like to let you know about a new Canadian peer-reviewed journal called the Journal of Geriatric Care. The first edition will appear early in 2002. The target audience includes primary care physicians and geriatric specialists. The articles will be primarily evidence-based reviews. If you have an interest in contributing to this journal, please contact me.

We would like to ensure that the Bulletin meets your needs. If you would like to contribute an article, news update regarding your region, conference report or letter to the editor, please contact Lonn Myronuk (myronuk@geripsych.com).

David Conn
416-785-2456
dconn@baycrest.org
CAGP Annual Scientific Meeting
Preliminary Program
Geriatric Psychiatry 2001:
From the community to the nursing home
Thursday November 15, 2001
Fairmont Queen Elizabeth Hotel
Montréal, Québec

Objectives
To provide a forum to explore issues in geriatric psychiatry. Lectures and workshops will focus on different aspects of clinical practice and research and will allow for discussion of clinical and research challenges.

Participants
The content will be of particular interest to psychiatrists and physicians who are treating the elderly in a variety of settings. Students and residents are welcome.

Program
08:00 Registration and coffee

Jolliet room
08:30 Welcome and opening remarks*
(David Conn)
08:45 Reducing the burden of mental illness in elderly populations*
(Martin Cole)
09:30 The Millennium Project*
(David Conn and Ken LeClair)
10:15 Coffee
10:30 Rural service delivery through teleconferencing
(Lonn Myronuk)
11:00 Evaluation of out-patient referrals for elderly depressed patients from the community*
(F. Primeau and D. Proulx)
11:30 APO-E genotyping*
(J. Poirier)
12:00 LUNCH SYMPOSIUM:
CAGP Fellows presentations*
Saint-Maurice Room
14:00 Concurrent workshops

a) Facteurs de risque pour retarder le placement des patients agés dement en centre d’accueil
(M.Elie) Chaudière Room
b) Efficacité thérapeutique des inhibiteurs de la cholinestérase sur les symptômes neuropsychiatriques de la démence.
(F.Rousseau) Matapédia Room
c) How to set up a rural teleconferencing program
(L.Myronuk) Saguenay Room
d) Starting a dementia clinic from scratch (B. Campbell)
15:00 Coffee
15:30 Concurrent workshops repeated
16:30 Closing remarks
Saint-Maurice Room
17:00 CAGP Annual General Meeting
18:00 Wine and cheese reception
18:30 Dinner (optional)

* Simultaneous translation
Registration

Registration will include all activities including lunch and coffee breaks. Delegates interested in attending an optional dinner following the meeting should check the appropriate section with details of food preferences.

<table>
<thead>
<tr>
<th>Members</th>
<th>Non-members</th>
<th>Students</th>
</tr>
</thead>
<tbody>
<tr>
<td>$80.00</td>
<td>$100.00</td>
<td>Free</td>
</tr>
</tbody>
</table>

Registration is also available on site. A $30 administration fee will be charged for cancellations which will only be accepted before September 11.

Registration Form

Name Last  First  Speciality and affiliation

City  Province  Postal Code

Tel. bus  home  CAGP member  □ yes  □ no

Indicate your choice of workshops:
14:00  15:30
1st  1st
2nd  2nd

Dinner preferences:
I will ___ will not ___ be attending dinner after the meeting. My food preferences are:

Forward this form with the appropriate registration fee (cheque made out to the CAGP) to:

Dr. Isabel Martens
CAGP Treasurer
L.A. Miller Centre
100 Forest Road
St John’s NF
A1A 1E5

For further information on the Canadian Academy of Geriatric Psychiatry Annual Meeting, contact: Dr. Lilian Thorpe, Clinical Gerontology, Saskatoon City Hospital, 701 Queen St., Saskatoon, Sask., S7K0M7, Tel: (306)655-7997, Fax: 655-7995, email: thorpel@sdh.sk.ca
The CAGP has provided crucial leadership in the mental health system by initiating the Millennium Project, through the CAGP co-chairs, David Conn and Ken LeClair. The Project’s purpose is “to improve mental health of the elderly in long term care through education, advocacy and collaboration”. To further this goal a National Invitational Symposium or “think tank” has been scheduled for April 28th and 29th, 2002 in Toronto, at the Toronto Colony Hotel. The Symposium is being developed in collaboration with the following organisations:

♦ The Canadian Academy of Geriatric Psychiatry
♦ Canadian Society of Consulting Pharmacists
♦ College of Family Physicians of Canada
♦ Health Canada
♦ Canadian Geriatrics Society
♦ Canadian Mental Health Association
♦ Canadian Alzheimer Association
♦ Canadian Nurses Association
♦ Canadian Association of Retired Persons
♦ Canadian Association for Community Care

This Symposium or think tank will provide an opportunity to discuss the issues and opportunities and create an implementation plan or blueprint for action. Approximately 150 leaders representing key stakeholder agencies will be invited to participate in this two-day event. The participants will be asked to define strategic issues and opportunities in advance of the conference.

The discussions will form the basis of action plans for change. Participants will be asked to create an action plan that details on-going initiatives to enhance mental health care to elderly clients in long term care settings. The action plan may include but is not limited to:

♦ the development of a national educational series for health care professionals and families
♦ national guidelines/best practices
♦ new opportunities for research activities
♦ advocacy opportunities
♦ network development
♦ provincial and other local initiatives

Participants will be asked to make on-going commitments to participate in the follow-up initiatives. A final report will be developed and sent out across Canada to all relevant stakeholders.

The members of the Millennium Committee will represent the CAGP at the Symposium.

PROCESS

The Symposium will be run in an Open Space concept. The participants will be asked to define what they believe are the issues. All issues will be “posted” for discussion and participants will self-select their discussion groups. Written reports from each discussion group that highlight key ideas/actions will be made available and circulated to all the participants. On the second day, the participants will cluster the themes and choose their priority areas. Participants will, once again, self select discussion groups. In the final discussion groups participants will identify achievable actions, champions and a work plan for follow-up. A final report that details these decisions will be made available to all interested stakeholders.

CONTACT

If you require additional information regarding this event please contact:

Shelly Haber
36 Warwick Avenue
Toronto, Ontario
M6C 1T8
Phone: (416) 781-2886
Fax: (416) 787-6157
s.haber@sympatico.ca
SPONSORSHIP
The National Invitational Symposium on Mental Illness in Long Term Care Facilities for the Elderly has been made possible by the generous donations of our sponsors.

Platinum Sponsor: Eli Lilly Canada
Gold Sponsor: Wyeth-Ayerst
Silver Sponsor: Janssen-Ortho Inc.
Bronze Sponsor: Organon Canada

Contributions have also been made by:
♦ Lundbeck Canada
♦ Merck Frosst Canada & Co.
♦ Pfizer Canada Inc.

Membership Report

SUSAN LIEFF

Our new member recruitment campaign is underway and we are having a very nice response to our mailings to the AMPQ and the geriatric section of the CPA. It has also forced the Board to clarify some of the criteria for the various types of membership. It was resolved that the training criteria can be applied for consideration of membership regardless of when the training occurred. The practice criteria, however, refer strictly to the applicant’s practice of geriatric psychiatry at the time of application and not previous practice. In addition, given the fact that many who apply for affiliate status may not know of colleagues who are members of the CAGP it is not required that their references be members of the CAGP. At the time of preparation of this report we have a total of 164 members, 39 of which have not paid their 2001 fees. They will shortly receive a letter encouraging them to take advantage of our 3-year renewal option, as I am sure that some of them are unaware of their status and probably lost their renewals somewhere on their desks.

Please welcome to membership the following colleagues:

Full members
Michele Doering
Marie Claire Baril

Affiliate members
Xi Min Lee
Bryan Mestelman
Abdulkarim Jiwa
Donald MacRae
Marie-Pascale Perrier
Phillip Wessels

Member-in-training
Nadeem Bhanji

Pierre Parenteau
Leon Kagan
Bharat Chawla
Nicole Robert
Jacques Potvin
Samuel Dey Jr.
Rosita Punti
Anysia Rusak
Joshua Benjamin
Cesar Garcia
Rob Jaunkalns
Carole Murphy
Evidence Based Medicine

Modafinil: Evidence for use in anergia?

**ROB VAN REEKUM**

The CAGP Bulletin presents the second in a series of evidence-based medicine reviews, based on work performed by educators and learners at the Baycrest Centre for Geriatric Care in Toronto. Requests or suggestions of topics for review or evaluation are welcome, and can be directed to Dr. van Reekum via E-mail care of the Baycrest Department of Psychiatry, arizzuto@baycrest.org.

**Clinical Case:**
A 67-year-old man presented with complaints of excessive fatigue following a traumatic brain injury.

**Clinical Question:**
Is modafinil efficacious for the treatment of anergia (lack of energy, fatigue) in persons with disorders of the brain (not including narcolepsy)?

**Literature Search:**
PsyINFO: modafinil (title word), 1984-2000
EMBASE: modafinil (key word), 1980-2001
MEDLINE: modafinil (title word), 1997-2000

<table>
<thead>
<tr>
<th>Conclusions</th>
<th>Study type</th>
<th>Evidence</th>
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<tr>
<td>Modafinil may be useful as an augmenting agent in depression (especially for the fatigue of depression).</td>
<td>Case series¹</td>
<td>7/7 subjects with treatment-resistant DSM-IV depression improved quickly on the HAM-D (especially regarding fatigue) with modafinil (100-200 mg/day) augmentation.</td>
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<tr>
<td>Modafinil may be helpful for the sleepiness of idiopathic hypersonnia.</td>
<td>Case series²</td>
<td>8/17 subjects with idiopathic hypersonnia were felt to improve clinically.</td>
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<tr>
<td>Modafinil produces subjective effects similar to caffeine and unlike amphetamines.</td>
<td>RCT-crossover¹</td>
<td>16 young, healthy volunteers reported that a single dose of modafinil felt similar to a dose of caffeine, and dissimilar to a dose of placebo or dextroamphetamine.</td>
</tr>
<tr>
<td>Modafinil may increase cerebral alertness and cognitive functioning in recently abstinent cases of alcoholic dementia.</td>
<td>RCT²</td>
<td>Alcoholic dementia (ICD 9:291.2) cases improved cognitively with modafinil 200 mg BID vs. placebo. Their EEG's showed less delta / theta activity.</td>
</tr>
<tr>
<td>Modafinil may maintain attentional and cognitive functioning in sleep deprivation.</td>
<td>RCT-crossover¹</td>
<td>Attentional speed was maintained with modafinil 300 mg/d over 60 hours of sleep deprivation while it declined with placebo.</td>
</tr>
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<td></td>
<td>RCT³</td>
<td>Modafinil improved cognitive performance during 64 hours of sleep deprivation vs. placebo, but also led to &quot;over-confidence&quot; in self-monitoring.</td>
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</table>
Conclusions:
Modafinil lacks sufficient evidence for routine use as treatment for anergia at the present time, but shows promise for the future. Randomised clinical trials of modafinil for anergia are supported by the currently available evidence.

REFERENCES