COGNITIVE IMPAIRMENT IN PARKINSON’S DISEASE

TO THE EDITOR
We would like to comment some methodological issues that arose from the interesting paper of Piovesan et al. and that did not were cleared in the discussion.

As it is known, many antiparkinsonian agents may cause cognitive and psychiatric symptoms, mainly those with anti-cholinergic effects. Piovesan et al. stated that none of the patients in the PWID group (Parkinson Group with depression) were taking medication with a potential anti-cholinergic effect, but did not mention anything about the parkinson group without depression (PWOD). If PWOD was taking anti-cholinergic drugs, this may have biased some results found in the paper, such as those stated here:

a) “comparison of individuals with and without depression did not reveal any statistically significant data that indicated that depression could have an influence on cognitive function in this group.” In other words, parkinsonian patients with depression may have similar cognitive performance just when compared with parkinsonian patients without depression but using anti-cholinergic agents, what will naturally impair the real cognitive potential performance of PWOD group.

b) “the PG group as a whole, rather than just those individuals whose scores indicated that they were depressive, had more obvious cognitive deficits than the CG group.” Once more, use of anti-cholinergic drugs may have biased this statement.

Besides that, another antiparkinsonian drugs (excluding this time the anti-cholinergic ones) may cause cognitive and psychiatric symptoms, including depression, thus explaining, at least in part, some discrepancies between the prevalence of depression in the parkinsonian group when compared with controls. Another important issue is that some psychiatric side effects (sleep disturbances, inapetence, concentration difficulties, disturbed thoughts) of dopaminergic agents may score and contribute to higher pontuations on Montgomery-Asberg scale in Parkinson’s Disease (PD).

Finally, we would like to comment on the difficulties related to the diagnosis of depression in PD. Because features of PD frequently overlap with typical manifestations of major affective disorder (or mild dysthymia), diagnosis of this comorbidity is challenging. Some of these interactive features include cognitive and speech deficits and impairments in emotional expression (e.g., PD-related facial masking) or processing. Apathy as well can be extremely difficult to distinguish from depression in PD.

REFERENCES

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AUTHORS’ REPLY
We would like, first of all, to thank Caixeta et al. for their interest in our article and for their constructive comments.

We wish initially to clarify that the group of patients suffering from Parkinson’s disease without depression were not using antidepressants.

It may not have been clear throughout the article that we took great care to avoid a methodological bias as a re-

Table. Comparison between the groups and subgroups.*

<table>
<thead>
<tr>
<th>Groups</th>
<th>1 x 2</th>
<th>1 x 3</th>
<th>2 x 3</th>
<th>4 x 5</th>
<th>1 x 4</th>
<th>1 x 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>MADRS</td>
<td>P=0.009</td>
<td>P=0.001</td>
<td>P=0.712</td>
<td>P=0.000</td>
<td>P=0.004</td>
<td>P=0.000</td>
</tr>
<tr>
<td>MEEM</td>
<td>P=0.001</td>
<td>P=0.000</td>
<td>P=0.619</td>
<td>P=0.209</td>
<td>P=0.000</td>
<td>P=0.001</td>
</tr>
<tr>
<td>FAR (FAS)</td>
<td>P=0.035</td>
<td>P=0.023</td>
<td>P=0.846</td>
<td>P=0.364</td>
<td>P=0.036</td>
<td>P=0.001</td>
</tr>
<tr>
<td>Animals</td>
<td>P=0.003</td>
<td>P=0.028</td>
<td>P=0.779</td>
<td>P=0.010</td>
<td>P=0.067</td>
<td>P=0.000</td>
</tr>
<tr>
<td>Errors/Attention</td>
<td>P=0.108</td>
<td>P=0.000</td>
<td>P=0.176</td>
<td>P=0.860</td>
<td>P=0.000</td>
<td>P=0.015</td>
</tr>
<tr>
<td>House</td>
<td>P=0.004</td>
<td>P=0.000</td>
<td>P=0.429</td>
<td>P=0.209</td>
<td>P=0.001</td>
<td>P=0.000</td>
</tr>
<tr>
<td>Clock</td>
<td>P=0.002</td>
<td>P=0.000</td>
<td>P=0.745</td>
<td>P=0.276</td>
<td>P=0.000</td>
<td>P=0.001</td>
</tr>
</tbody>
</table>

* Mann-Whitney test. Group 1: Controls (n=30); Group 2: PGWIA (Parkinson group with anticholinergic) (n=11); Group 3: PGWOA (Parkinson group without anticholinergic) (n=19); Group 4: PGWOD (Parkinson group without depression) (n=23); Group 5: PGWID (Parkinson group with depression) (n=07).
sult of a possible influence of anticholinergic drugs on the
cognitive tests in patient groups and subgroups. Although
not mentioned in the article, the patients were analyzed
in separate groups (users and non-users of anticholineric
drugs in the Parkinson group). There was no significant
difference between the groups (Table). When compared
with the control group, a statistically significant differen-
tce was observed in all the tests for the anticholinergic
users, except in the errors/attention test. However, there
was a statistically significant difference between the con-
trol group and the group that did not use anticholinergics
in all tests (Table).
Caixeta et al. point out that the Parkinson group with
depression yielded scores similar to those for the Parkin-
song group without depression, and they attribute these
values to the fact that the Parkinson group without de-
pression was probably compromised by the use of anti-
cholinergic drugs. It can be seen from Table that MADRS
was significant, since this is a test for depression. The only
difference between the groups occurred in the animals
test. This could be because of greater difficulty in cate-
gory rather than phonetic verbal fluency.
Some antiparkinsonian drugs can have some effect on
cognition and depression. Levodopa has limited or no an-
tidepressant effect and can occasionally be responsible
for depression. However, the reported frequency of de-
pression before and after the start of levodopa therapy
was similar. This does not support the idea that this drug
may increase the frequency of depression, although the
possibility that it plays a role in precipitating or exacer-
bating the condition cannot be definitively excluded. Do-
paminergic agonists can also affect mood and generally
lead to an improvement in depressive symptoms. Mood
changes in response to these drugs tend to vary more than
the motor responses. Drugs with a potential anticholin-
ergic effect have little influence on mood changes, and al-
though in some cases they can lead to mild euphoria, they
are relatively ineffective as antidepressants. The possible
effects on cognition can vary according to the stage of
the disease and the extent to which the extranigral do-
paminergic pathways are compromised.
Diagnosis of depression in Parkinson’s disease can
be extremely complicated. The question remains as to
whether it is the result of basic physiopathological mech-
isms or secondary to motor impairment.

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GLOSSOPHARYNGEAL NEURALGIA WITH SYNCOPE
AS A SIGN OF NECK CANCER RECURRENTCE

TO THE EDITOR
I have read the article (case report) “GLOSSOPHARYN-
geal neuralgia with syncope as a sign of neck cancer rec-
currence” by Reinaldo Teixeira Ribeiro et al., and found
it extremely interesting. I would like to give a historical
contribution; not regarding the rare relationship between
glossopharyngeal neuralgia with syncope and neck cancer,
because authors approached this very well, but in respect
to the following citation in the first paragraph of the dis-
cussion: “Among the Brazilian cases of classical gloss-
opharyngeal neuralgia previously reported...” Here, the au-
thors missed an opportunity to include Professor Pedro
Sampaio’s fundamental work on this issue.

The first and most important study in Brazil concern-
ing glossopharyngeal neuralgia was made by Professor Pe-
dro Sampaio when he made his Livre Docência Thesis to
Universidade do Brasil in 1954, and published his results
that same year in the Jornal Brasileiro de Neurologia.
Pedro Sampaio made an extensive clinical study de-
scribing ten cases of glossopharyngeal neuralgia, exper-
imental research utilizing eight dogs, and a broad bibli-
ographic review. In respect to two of his patients; one
with glossopharyngeal neuralgia and fainting sensations
(case 9) and the other with concomitant tonic-clonic sei-
zures (case 10), he created the term “neuralgia sinocarot-
tidea” to denominated the algic form localized in the pos-