

TUMOUR-LIKE CHAGASIC ENCEPHALITIS IN AIDS PATIENTS

TO THE EDITOR

I am a reader of *Arquivos de Neuro-Psiquiatria*. I have a comment on the article 2008;66(4):881-884; Tumor-like Chagas encephalitis in AIDS patients, Sica, Gargiullo and Papayanis. I congratulate the authors on the abundance of their material. I would like that the photomicrograph of the parasitic nest (Figure 2) had been made in oil immersion, to demonstrate the blepharoplasts characteristic of amastigotes of Chagas disease.

L.C. Mattosinho França
São Paulo, Brazil

THE AUTHORS REPLY

We thank the comments of Dr. L.C. Mattosinho França. Unfortunately, we have not pictures showing the parasite by employing microscopical immersion techniques.

Within the material that we have collected and studied in the last years, encompassing 18 AIDS patients with tumour-like chagasic encephalitis, 8 of them had positive sera test for toxoplasmosis as well; however, in all of them

appropriate treatment targeting the toxoplasma neither rendered positive results nor modified the cerebral images; furthermore, their clinical manifestations worsen during treatment. Therefore, in those circumstances we carried on serological tests and looked for trypomastigotes in their blood, spinal fluid when possible, and, when necessary, in cerebral tissues either by performing biopsy or by surgically removing the lesion. In every case we could find laboratory signs of the presence of the parasite which prompted us to start specific treatment.

Being these persons weak regarding their immunological system, it is possible to accept that they could house more than one opportunistic infection at the same time, as seems to be the case with toxoplasma and *T. Cruzi* coinfection in these patients, at least in regards to their serological tests.

R.E.P. Sica
G. Gargiullo,
C. Papayanis
Buenos Aires, Argentina

DOM PEDRO II DAYTIME SLEEPINESS

TO THE EDITOR

I read with much interest the excellent article in the September 2008, volume 66, 3B issue of the *Arquivos de Neuro-Psiquiatria*, on "Dom Pedro II daytime sleepiness", page 770. The thesis of the authors is plausible, but unfortunately we lack confirmation of Dom Pedro II's sleep apnea by the two possible observers, the ladies with whom he slept; they are the Empress Maria Teresa and the Italian countess with whom Dom Pedro II at times spent his nights when he was travelling in Europe. Nevertheless, the authors, Drs. Rubens Reimão, Marleide da Mota Gomes and Péricles Maranhão-Filho are probably right.

There is another possible explanation for Dom Pedro II's daytime sleepiness¹⁻³.

I have a special interest in it since I myself suffer from it. As all neurologists know, each person awakens for a few seconds from four to five times each night. Until old age,

since the awake periods are very brief and the slumbers are very long, people rarely are aware of these very brief awake periods. In old age this changes. The awake period lengthen to from ten to twenty or more minutes. Some old persons spend 20% or more of their sleeping hours awake, and hence have daytime sleepiness during their days.

I am one of these old persons. I am 84 years old. To combat the problem I keep by my bedside a thermos bottle of camomile hot tea, my pipe and mild tobacco, and warm milk. When I wake up I partake of these three things and, with luck, get back to sleep in ten or fifteen minutes. However, these awake periods sometimes stretch out to thirty minutes or so. During the day I struggle with my problem of daytime sleepiness with more camomile tea, mild pipe tobacco and walks to the garden which is behind my service of neurology mainly an active EEG ser-

vice which draws patients from a large part of south Bahia and northern Minas Gerais, and which is now in its 32nd year of existence. Despite the problem I manage to keep up with my neurological work during about fifty hours each week.

To recapitulate, the authors of this excellent article are probably right in their thesis that Dom Pedro II suffered from sleep apnea, but in the absence of eye witnesses of his sleeping time and patterns, there is the less likely possibility that he suffered from the problem discussed in this letter.

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*Arthur Harry Chapman
Vitória da Conquista*

ABSENCE OF MRI EXAMS IN EPIDEMIOLOGICAL STUDIES CAN LEADS TO CLINICAL OVERDIAGNOSIS OF ALZHEIMER'S DISEASE AND UNDERDIAGNOSIS OF VASCULAR DEMENTIA

TO THE EDITOR

We would like to comment some methodological issues that arised from the interesting paper of Tascone et al.¹ and that did not were cleared in the discussion. Until and unless we have neuropathological confirmation of diagnostic accuracy, our conclusions must be seriously questioned since we all know that the definitive diagnoses of Alzheimer's disease (AD), vascular dementia (VaD), and other dementias are based on neuropathological criteria. Obviously, this limitation should not hinder epidemiological studies like the excellent work of Tascone et al. However, caution should be taken related to the prevalence rates of each dementia form, since some of them are almost exclusively diagnosed with expensive neuroimaging methods as is the case of MRI for the correct diagnosis of an important and frequent form of dementia: Binswanger disease or subcortical vascular dementia.² As the authors did not mentioned if they used CT or MRI scans in their patients and because MRI is an expensive method, we assume that most of their patients probably was examined with CT scans.

The predominance of AD diagnosis found by the authors is remarkable (62.8%), but is in consonance with international literature. These findings, however, may reflect an overdiagnosis of Alzheimer's disease to the detriment of vascular dementia, since individuals with silent cerebrovascular disease or even subcortical cerebrovascular disease may not be detected without using MRI scans. In fact, in their study the diagnosis of "other dementias" which comprises patients with vascular dementia (VaD) and other forms of dementia is surprisingly low (14.2%),

contradicting vascular dementia rates in other studies.³ Adding contribution to this debate, we should mention that one of the most important authorities in the field postulated that the two main causes of VaD – stroke and ischemic heart disease (IHD) – may be responsible for the majority of cases of dementia in the elderly. This author remembered that cerebrovascular disease (CVD) is the second leading cause of death worldwide. About 1/3 of stroke survivors [range: 25–41%] 65 years old and above develop VaD within 3 months following the ictus. In the USA alone, 125,000 new cases/year of VaD occur after ischemic stroke (about 1/3 of the 360,000 incident cases of AD). Therefore, more than 1 million elderly people are currently affected by poststroke VaD in the USA. Besides that, IHD leading to congestive heart failure (CHF) will become the leading cause of disability worldwide. Vascular cognitive impairment occurs in 26% of patients discharged from hospitals after treatment for CHF. Cognitive dysfunction correlates with left ventricular dysfunction and systolic blood pressure below 130 mm Hg. CHF is a leading cause of hospital admissions in Western nations (4.5 million cases in the USA alone) and is a growing problem in developing countries. Furthermore, over 800,000 patients/year undergo coronary artery bypass graft (CABG) surgery worldwide, including 300,000 patients in the USA. Measurable cognitive dysfunction occurs post-CABG in 80–90% of patients at hospital discharge. Long-term (5 years) incidence of cognitive defects is 42% 4,5.

Other factor that may have contributed to the low incidence of VaD in this study should be the psychiatric ori-