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LETTER TO THE EDITOR

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Symmetrical selective neuronal necrosis in solitary tract nuclei

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Dear Sir,

In our paper "Solitary tract nuclei in acute heart failure" (De Caro et al. 2000) we reported symmetrical selective neuronal necrosis in the solitary tract nuclei in five adults with short survival intervals after acute heart failure. We suggested that given the limited extent of the symmetric medullary lesions, cerebral ischemia could not be the only factor responsible for them. In fact, the absence of lesions of other brainstem nuclei accounts for the selective vulnerability of the neurons of the solitary tract nuclei, which may be explained with reference to vascularization of the medullary tegmentum. Furthermore, we suggested that dendritic lesions indicate that cell death had an excitotoxic component (Olney 1972) that can be ascribed to hyperexcitation of neurons in the solitary tract nuclei in the post-ischemic period.

We must point out that in the publication of Lorin de la Grandmaison et al. (2001), two original aspects of our paper (De Caro et al. 2000) are wrongly reported: owing to the placing of the clauses, our conclusions are apparently attributed to other authors. We consider this incorrect.

In particular, considering the limited axial (bulbar) and transverse (tegmental: nuclear) extension of the brainstem lesions, we proposed that, due to intense metabolic activity (McLean et al. 1999), the subependymal portion of the bulbar tegmentum, mainly the solitary tract nuclei, could be particularly vulnerable to ischemia after a critical decrease in cardiac activity. Lorin de la Grandmaison et al. (2001) arbitrarily attribute our sentence to McLean et al. (1999). In reality, this conclusion was proposed by ourselves, according to our own findings with reference to the idiodentritic configuration of the limited extent of the lesions, accounting for selective necrosis of a group of neurons with greater vulnerability.

We also proposed that the location of the lesions at the level of the solitary tract nuclei after acute heart failure may be explained with reference to vascularization of the medullary tegmentum. We quoted Foix and Hillemand (1925), who described three areas of vascularization in this district: median, fed by the paramedian arteries for the motor nuclei; middle, very small and fed by the short circumferential arteries for the ala cinerea; and lateral, fed by the posterior inferior cerebellar artery for the restiform body. We concluded that because the solitary tract nucleus is localized at the watershed zone between the terminal branches of these arteries, it is predictable that the nucleus should be particularly exposed to ischemia after acute heart failure.

Lorin de la Grandmaison et al. (2001) quote Foix and Hillemand (1925) as the originators of this hypothesis, whereas in fact they only studied vascularization of the medulla and never considered the effects of critical reduction in blood flow in the vertebrobasilar system. On the contrary, based on the theory of Opitz and Schneider (1950), we proposed that the solitary tract nuclei represent the "most distant field" in the medullary tegmentum.

In both cases, the introduction of the sentences of our paper were placed later, so that the whole sense was attributed to authors who had in fact never proposed the conclusions that Lorin de la Grandmaison et al. (2001) attributed to them.

Our paper (De Caro et al. 2000) represents the first report of symmetrical selective neuronal necrosis in the dorsal part of the solitary tract nuclei, without gliomesodermic

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reaction, following acute heart failure. Our findings indicate that small, roundish, symmetrical, eosinophilic areas in the solitary tract nuclei should be evaluated as initial features of selective neuronal necrosis, and that the commonly accepted resistance of the medullary centers to ischemic hypoxia in adults may apparently be due to the short survival interval before death, which prevents the evolution of reactive processes.

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