

C-FOS expression in the subnucleus gelatinosus of the human nucleus tractus solitarii

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SUMMARY

The dorsal portion of the nucleus tractus solitarii (NTS) shows selective dendritic lesions in adults who died after an episode of acute heart failure, suggesting excitotoxic glutamate-induced neuronal death. Cerebral hypoxia and/or ischaemia also produce hyperexpression of specific genes (c-fos, c-jun) which may be involved, in vulnerable districts, in the mechanisms of excitotoxic neuronal death. In the present study, we examined NTS in 23 adults, who died shortly after an ischaemic and/or hypoxic event, on transverse serial sections of the medulla, stained with haematoxylin-eosin, Nissl, Klüver-Barrera and Luxol fast blue, and immunohistochemical staining by anti-tyrosine hydroxylase and anti-Fos.

In 10 cases, at the level of the dorsal portion of the NTS, corresponding to the subnucleus gelatinosus, bilateral symmetrical hypereosinophilic areas and strong Fos-like immunoreactivity were detected. The hypoxic-ischaemic origin of these findings was supported by the strong hypereosinophilic appearance and shrinking of neurons, and by selective Fos-like immunoreactivity, the expression of Fos being mainly associated with cellular damage and subsequent death following hypoxic-ischaemic injury. In 7 other cases, Fos-like immunoreactivity was found at the level of the subnucleus gelatinosus, in the absence of hypereosinophilia or pyknosis in histological staining. The immediate-early gene expression induces to ascribe the absence of morphologically evident hypoxic-ischaemic lesions to the rapidity of death.