

# Morphologic Alterations In N. Vagus System In Chronic Gastric And Duodenal Ulcer

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## Abstract

The problems of chronic gastric and duodenal ulcer pathogenesis are not studied completely despite of the disease infectious origin evidence. The role of central nervous system in ulcer persistence is not enough studied by means of morphologic methods.

The study revealed statistically significant decrease of number of neurons in transversal sections of nucleus dorsalis n. vagi in medulla oblongata in chronic gastric and duodenal ulcer.

These changes can be evaluated as obligate and play an important role in ulcer pathogenesis.

## INTRODUCTION

Infectious factor in ulcer disease is quite obvious today (<sup>1,2,3,4,5</sup>). But the problems of chronic gastric and duodenal ulcer pathogenesis are not studied completely despite of the disease infectious origin evidence (<sup>6,7</sup>). The persistence of ulceration and recidivation after complete eradication of *H. pylori* is to be understood by another, probably well-known pathogenetic mechanisms. The role of central nervous system in ulcer persistence is not enough studied by means of morphologic methods. In ulceration the degenerative and dystrophic processes are manifested in n. vagus trunks but the state of dorsal nuclei is not studied enough. Focal neuronal loss may result in constant changes in trophic innervation and development of pathologic processes in gastroduodenal mucosa and make appropriate conditions for ulcer persistence.

## METHODS

The research was undertaken on autopsy material. This approach is useful to reveal changes in neural centers that had been accumulated within individual life but reduces the range of applied methods to morphologic and morphometric studies of n. vagus dorsal nuclei, n. vagus trunks, gastric and duodenal mucosa.

Fragments of medulla oblongata were fixed in 10% formaldehyde and undergone standard histologic processing.

Cross-sections of medulla oblongata were prepared and stained by means of standard histological and histochemical

methods. The calculation of neurons in the right and left dorsal nuclei per field of vision (280x) was undertaken with mean number identification. This method was applied for standardization of the research.

New stereological techniques (<sup>8,9,10</sup>) have failed to confirm earlier findings regarding age-associated neural loss, but there is the evidence of focal neural death and synaptic or receptor loss. The application of techniques mentioned above was not quite correct in our research. The main reason is some uncertainty in the determination of nuclei size and belonging of particular neurons to functional units of this exact nucleus. It is well-known that singular neurons and small groups of neurons are diffusely located within the brain stem.

We applied cell counting per field of vision so that central part of nucleus (approximately 75% of cross-section square). Nuclear and cellular volumes of neurons were estimated additionally.

The condition of vagal trunks was evaluated by means of optical microscopy. Fragments of trunks taken on various levels were fixed in 10% formaldehyde and undergone standard histologic processing. Cross-sections of medulla oblongata were prepared and stained by means of standard histological and histochemical methods.

## RESULTS

96 autopsies were studied: in 50 cases chronic gastric or

duodenal ulcer was revealed, 46 cases formed control group of autopsies with cardiovascular pathology.

The results revealed the decrease of neurons amount in transversal sections of dorsal nuclei in medulla oblongata which made up 22,9%( mean quantity ) in cases of gastric of duodenal ulcer persistence comparing with control cases. Morphometry also revealed the decrease of cellular and nuclear volumes comparing with control ones for 14,1 %.

The results are presented in Table 1.

## Figure 1

Table 1: Number of neurons and their volumes in cross-sections of nucleus dorsalis n.vagi in group of autopsies with chronic gastric and duodenal ulcer and in control group.

|  | Main group            | Control group        |
|--|-----------------------|----------------------|
| Mean number of neurons                   | 21,14                 | 27,42                |
| Volume of neuron (mean number)           | -8 3<br>3,78 x 10 mm  | -7 3<br>1,9 x 10 mm  |
| Volume of neuronal nucleus (mean number) | -10 3<br>7,49 x 10 mm | -9 3<br>2,35 x 10 mm |

Presented results are statistically significant. We calculated Student criterion ( $P < 0,01$ ) and Pearson criterion that equals 80,92. This result is also statistically significant.

The condition of vagal trunks can be characterized by processes of dystrophy, sclerosis and chronic inflammation. These changes were observed mainly in the level of diaphragm. They are correlated with relative dilatation of diaphragm and are not obligate in ulcer.

## DISCUSSION

The study revealed morphologic evidence of correlation between the alterations in nucleus dorsalis n. vagi and chronic gastric and duodenal ulcer persistence. These changes can be evaluated as obligate for chronic peptic gastric and duodenal ulcer and play an important role in ulcer pathogenesis. Moreover, we can see obligate significant correlation between focal cell loss in neural regulatory centers for gastrointestinal system and persistence of pathologic changes in gastrointestinal mucosa. These changes are expressed by the most typical form of gastritis associated with ulceration: chronic antral gastritis.

Now the reasons of neuronal loss and morpho-functional activity decrease of remaining cells remains unclear. Further investigation is necessary to reveal what primarily occurs: focal loss of neurons or ulceration. But it is obvious that such focal damages in nervous regulatory centers may result in constant dysfunction of trophic vegetal innervation. Although there are certain reserves of neural stem cells in the human CNS, they are unable to generate new nerve cells in any useful amounts. This is evolutionary conditioned feature (11,12).

We suppose that focal neuronal loss may take place not only in neurodegenerative diseases but may be one of pathogenetic mechanisms of chronic somatic diseases.

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