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Fasted motor function of the small bowel is modified by antigen sensitization with or without strees in rats

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Abstract

AIM: Anaphylaxis has been shown to cause gut motor dysfunction. However it is not clear if antigen sensitization alone causes any change in gut motility. Moreover, the effects of mental stress another cause of motor dysfunction and food intolerance have not been studied at the same time to determine whether they are additive or interactive stimuli. We recorded small bowel motor activity in fasted ovalbumin (OA)-sensitised rats exposed to 'flood stress' to study the effect of antigen sensitization and mental stress on small bowel motility.

METHODS: We recorded the electromyogram (EMG) in the proximal jejunum of 4 groups (n = 8 in each group) of fasted Hooded Lister rats (body wt 200-220 g) 14 d after treatment with OA sensitisation, or sham sensitisation with only adjuvant (Aluminium hydroxide) as vehicle control, or no treatment. In each animal, a bipolar AG/AG C12 electrode qas implanted on the jejunal serosa under general anaesthesia, with the electrode leads exteriorized through the lumen of a coiled spring tube that allowed free movenent after recovery. Seven days after surgery, animals were fasted and placed in an acrylic tank; The EMG was then recorded for 4 h. During the 2nd hour of recording the tank was partly flooded, compelling the animals to stand on a small plinth to avoid immersion. Finally, blood was taken to determine the IgE titre.

RESULTS: In the OA sensitized rats IgE titre was ≥ 64, but was not detectable in unsensitized animals. In the OA sensitized rats median of total spikes in the first hour of recording were 3730 (637-5541)/h, not significantly different from the 3 control groups. When the same data were grouped in one minute time epochs and plotted against time, peaks of spike activity were clearly seen corresponding to phase Ⅲ of the migrating motor complex (MMC). In OA-sensitized rats, the median spike activity in MMC phase Ⅲ was 134 (58-272)/min, which was significantly (P < 0.05) higher than in control groups. In the first hour the median number of MMCs in the OA sensitized group was 6 (4-9)/h, not significantly (P > 0.05) different from the control groups. During and after stress, MMCs were disrupted and replaced by prolonged irregular spike activity. This disturbance in OA sensitized group lasted for 120 (90-180) min, significantly (P < 0.05) longer than those in control groups. Our data show that in fasted rats the effect of antigen sensitization on spike activity is only seen during the peaks of spike activity that occur in phase Ⅲ of MMC, but OA sensitization prolongs the MMC disturbance induced by mental stress. These findings suggest that activated immune cells in gut influence gut motility, and superimposed mental stress exaggerates the effect. The data provede a biological basis for the hypothesis that food intolerance and stress may interact in the pathogenesis of human functional disorders such as the 'irritable bowel syndrome'.

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