6th European Conference on Computational Mechanics (ECCM 6)
7th European Conference on Computational Fluid Dynamics (ECFD 7)
11 – 15 June 2018, Glasgow, UK

BIOMECHANICS OF GASTROPARESIS

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Key Words: *Human Stomach, Mathematical Model, Gastroparesis.*

The model presented here is an extension of previous research on mathematical modeling of the gastrointestinal tract [1]. It enables study of the mechanisms of neuroneuronal and neuromusclular signal transduction, the phenomena of receptor polymodality, neurotransmitter co-localization and co-transmission as well as mechanical contractile activity in the human stomach. The model has been used to simulate a state of gastroparesis.

The results of simulations reveal, for the first time, the peculiarities of interplay between neuroregulatory and biomechanical mechanisms of the human stomach which could be attributed to the pathophysiological changes seen in patients with gastroparesis. It is demonstrated that, under physiological conditions, the strongest passive membrane forces are produced in the antrum of the stomach, 155 mN/cm and the lowest in the fundus, 94 mN/cm. Maxima active forces are generated in the body and antrum of the stomach, 82.9 mN/cm, whilst being significantly less in the fundus, 41.5 mN/cm. The amplitude of contractions also varies across the organ from the smallest recorded in the fundus, 8.5 ÷ 11.8 mN/cm, to the greatest recorded in the antrum, 19.6 ÷ 35.3 mN/cm. The activation of inhibitory mechanisms results in a considerable reduction in the intensity of forces across all regions of the stomach, a fall in intraluminal pressure and overall expansion of the organ (gastric accommodation). By contrast, in the gastroparetic stomach there is a weak accommodation response. The organ retains the configuration homeomorphic to the initial shape. There are high intensity total membrane forces in the body and the antrum compared to the norm. Smooth muscle layers generate tonic-type contractions albeit low in strength. No coordinated peristaltic activity can be seen in the gastroparetic stomach.

Although the biomechanics of gastroparesis *per se* cannot be analyzed with precision at this stage due to the lack of information required concerning the degree of morphostructural tissue damage and altered electromechanical properties, extrapolations can be made based on the results of modeling investigations of affected gastric motility. The results show that disconnected ganglia within the myenteric plexus, despite their close proximity, cannot sustain the propagation of electromechanical waves of contraction in the organ. The integrity and functionality of the myenteric nervous plexus is essential to achieving the desirable physiological effect. Further investigations are now required to assess the validity and applicability of the model and the results to simulate the effects of drugs used in treatment of gastroparesis.

REFERENCES

[1] R.N. Miftahof, *Biomechanics of the human stomach*, Springer-Verlag, Cham, 2017.