

Insights from a magnetic resonance imaging-based evaluation of five hiatal hernia patients pre- and postoperatively on heart-mediated peristaltic action for reflux control and clearance: a case report

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Background: Reflux disease is commonly attributed to the lower oesophageal sphincter and multifactorial aspects. New clinical data show that repositioning the oesophagus to its anatomically correct position and stable hiatus reconstruction alone can immediately restore reflux control and resolve symptoms without fundoplication or other antireflux procedures. The aim of the study was to understand the interdependence of oesophageal position and function, and how the surgical repositioning of the oesophagus alone can fully restore reflux control.

Case Description: Five patients with symptomatic gastroesophageal reflux were successfully operated on by laparoscopic repositioning of the oesophagus and hiatus reconstruction without fundoplication. The pre- and postoperative hiatal statuses were retrospectively compared using magnetic resonance imaging (6-mm cine-mode sequences) and X-ray contrast swallow evaluation. Different functional aspects of the oesophagus were detected in the patients, which formed in its entirety a new operating principle of the oesophagus function. The oesophagus was shown to be involved in a direct interaction with the heart. The heart acted as the central organ of a peristaltic pumping system that triggered a downward impulse along the oesophagus. This heartbeat-mediated rollout of the oesophagus within the detected cardioesophageal and diaphragmatic interaction system was crucially ensured by the architecture of the oesophagus hiatal unit, which was impaired preoperatively in all patients and repaired after surgery. Postoperative images showed that reflux appeared to be functionally prevented by a continuous downward pulsatile counter-current at the cardioesophageal junction in a basically open system.

Conclusions: The data revealed a new operating principle for the heart, the oesophagus, and the diaphragm. The beating heart was identified as the central motor of the peristaltic pumping system for clearance and reflux control. The oesophagus was only passively embedded in this powerful system. These findings contradict the common concept of a lower oesophageal sphincter and indicate a paradigm shift in the understanding of the pathophysiology of gastroesophageal reflux. The data are deposited in the archives of Berlin centre for hernia and reflux surgery.

Keywords: Hiatal hernia; heart-mediated reflux control; cardioesophageal pump; lower oesophageal sphincter (LES); case report

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Introduction

For decades, the common pathophysiological hypothesis for gastroesophageal reflux has been the impairment of the lower oesophageal sphincter (LES). However, anatomists have failed to find a corresponding welldefined morphological structure within the smooth, slowacting circular fibres of the soft oesophageal wall, which are only 1–2 mm in diameter (1-3). Therefore, a long list of speculative genetic, neurological, hormonal, and environmental cofactors, flaps, valves, and flap valves have been added to this concept of a diseased oesophagus to explain the persistent inconsistencies (4-8).

In contrast, empirical data show that returning the oesophagus to its anatomically correct position and reconstructing a stable hiatus can immediately restore reflux control and resolve symptoms, even with strict avoidance of fundoplication or other common antireflux procedures (9). The aim of the case series was to understand the interdependence of oesophageal position and function, and how the surgical repositioning of the oesophagus alone can fully restore reflux control. I present the following case in accordance with the CARE reporting checklist (available at https://ls.amegroups.com/article/view/10.21037/ls-22-22/rc).

Case presentation

In this retrospective case series, five patients with symptomatic gastroesophageal reflux disease underwent surgery in 2012 and 2013. Preoperatively, patients experienced heartburn, chest pain, hoarseness, upright sleeping position, and food intolerance and were taking proton pump inhibitor (PPI) medication 40–80 mg/die for relief. Gastroscopy confirmed a significant axial hiatal hernia and gastroesophageal reflux. Preoperative diagnostics such as X-ray contrast swallow and cine-mode magnetic resonance imaging (MRI) were repeated postoperatively within 3 months due to chest pain and pressure sensations and to assess possible irregularities after surgery. Patients had no history of heart disease and no cardiac diagnosis other than physical examination and 12-lead electrocardiogram (ECG). Detailed inclusion criteria have been described elsewhere (9).

The patients underwent successful surgery by the new technique of laparoscopic oesophagohiatal DeltaMesh enhancement, which focuses exclusively on the correct anatomical reconstruction of the oesophagohiatal unit and correct repositioning of the oesophagus. Fundoplication, other anti-reflux procedures, or any sutures to the oesophagus or stomach were strictly omitted in every patient. For long-term stabilisation, a new three-dimensional DeltaMesh was used for hiatal enhancement, which was specifically designed to meet the hiatal architecture and unique requirements of a destructed hiatus. All patients subsequently regained full reflux control in every respect. Details of the DeltaMesh and the surgical procedure have been described elsewhere (9). Surgeries were performed at Parksanatorium Dahlem, Germany. MRI was performed at the Department of Diagnostic Radiology, KMG Klinikum Luckenwalde, Germany. Cine-mode MRI scans were performed using Siemens Magnetom Avanto 1.5 T (Siemens Healthineers, Erlangen, Germany), producing identical continuous 6 mm True Fast Image sequences in the sagittal, frontal, and transverse planes without contrast medium.

All procedure performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patients for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Three patients were male and two were female. The median age was 49 years (range, 38-57 years) and the median BMI was 27.5 kg/m² (range, 21.5-31.1 kg/m²). One patient was classified ASA1 and four patients ASA2. Disease persisted symptomatic for 4 years (median; range, 2-31 years). Patients had no previous upper abdominal surgery. Patients were treated by one surgeon and all surgeries were completed laparoscopically. Intraoperative measurement showed a median hiatal defect of 5 cm (range, 4-5 cm) estimated as the distance between the dorsal oesophageal wall and posterior confluence of the crura after complete repositioning. The results presented are a summary of findings in all patients and could not be fully documented in every patient.

Postoperative sagittal cine-MRI scans showed a direct mechanical interaction between the heart and oesophagus. The systolic heartbeat exerted strong rollout compression on the oesophagus from oral to aboral over a length of approximately 7 cm, resulting in an effective antegrade pumping motion along the oesophagus (*Video 1*).

This observation was confirmed by X-ray contrast swallow evaluation. It was shown that, at the beginning of the mechanical cardioesophageal connection, a progressive pressure wave of the heart started that led to an increase

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Video 1 Cine magnetic resonance imaging sagittal plane, postoperative. Arrow marks the oesophagus and the area of the cardioesophageal pump. The directly transmitted cardiac impulse showed a rollout movement along the oesophagus from proximal to distal. in downward compression along the oesophagus. The rollout movement squeezed 80–90% of the oesophageal lumen from its original width of 12 to 2 mm. Maximum compression was reached in the far distal segment, where the oesophagus rose freely and elastically in a small concave curve, just before meandering through the hiatus (*Figure 1*).

The compression wave only affected the ventral wall of the oesophagus, while the dorsal wall remained stable and almost unaffected in shape and position. Propulsive or contracting peristaltic waves of the oesophagus were not detectable. During this process, the contrast medium in the oesophagus was pumped forward at high speed, and complete oesophageal clearance was achieved with only one cardiac compression wave and one heartbeat. The impulse appeared to start from the left ventricle and exerted such a strong force that even the adjacent stable aortic wall was



Figure 1 X-ray contrast swallow evaluation of the distal oesophagus sagittal plain, postoperative. (A) The oesophagus is filled with contrast medium and shows its reconstructed concave ascending curve around the heart to the hiatus. Position of the VEW is marked with a dotted line and arrows as the reference for its position before the compression wave of the heart. The DEW remains as a line. The diameter of the oesophagus at the measuring point is 12 mm. (B) The heart-mediated systolic impulse (black curved arrow) on the oesophagus starts to compress the VEW. The clearance of the contrast medium starts immediately. The diameter of the oesophagus at the measuring point is 10 mm. (C) The impulse rolls down the oesophagus and progressively compresses the VEW. The diameter of the oesophagus at the measuring point is 7 mm. (D) The rollout movement completely cleared the oesophagus by one heartbeat. The diameter of the oesophagus at the measuring point is 3 mm. (E) The compression of the oesophagus has reached a peak of 2 mm at the measurement point and the rollout ends. In the following diastolic phase, the renewed decompression of the VEW is proximally detectable (see double arrow). The DEW remained stable over time. Active peristaltic contractions were not detectable. E, oesophagus; H, hiatus; VEW, ventral oesophageal wall; DEW, dorsal oesophageal wall; C, contrast medium.



Video 2 Cine magnetic resonance imaging sagittal plane, slow motion, postoperative. The powerful impact of the cardiac impulse was shown by the fact that even the stable aortic wall was dented (arrow).



Video 4 Cine magnetic resonance imaging horizontal plane, postoperative. The cardioesophageal pump created a continuously pulsating downward counter-current in the gastroesophageal junction (arrow).



Video 3 Cine magnetic resonance imaging sagittal plane, slowmotion postoperative. The countermovement of the upward systolic blood ejection of the left ventricle led to a downward impulse on the tightly coupled oesophagus. This impulse led to a considerable compression of the lumen and a deflection of the oesophagus backwards. Dorsal support for the oesophagus had been provided by the systolic aortic wave, which synchronously passed by (arrow marks the oesophagus).

dented (Video 2).

However, it did not appear to be a separate pumping mechanism but rather the utilisation of the physiological downward countermovement of the heart during its vigorous upward systolic blood ejection (*Video 3*). Furthermore, the pumping motion of the heart led to both rapid oesophageal clearing and a permanent pulsating antegrade counter-current at the level of the gastroesophageal junction (*Video 4*).



Figure 2 MRI transversal plain, postoperative. The oesophagus is firmly embedded between the heart, spine, and aorta without the possibility of evading. At the moment of the heart impulse, the oesophagus is compressed into a flat oval shape (arrow). MRI, magnetic resonance imaging.

Horizontal MRI images at the beginning of the cardioesophageal junction showed that the oesophagus was firmly embedded and fixed between the heart, spine, and aorta, thereby preventing any evasion and possible impairment of pulse transmission (*Figure 2*). As it progresses, the elastic deflection of the oesophagus at the moment of ventral impulse appears to be cushioned by the systolic aortic wave running dorsally downwards and increasing the efficiency of the pumping motion (*Video 3*).

Cine MRI showed that the oesophagus was as firmly anchored at the start of the cardioesophageal interaction preoperatively as it was postoperatively. Differences in the transmission of the cardiac action to the oesophagus were



Video 5 Cine magnetic resonance imaging sagittal plane, preoperative. At the beginning of cardioesophageal coupling, the heart still transmitted its impulse to the firmly embedded oesophagus. In the following, however, the oesophagus had flattened and thus escaped the crucial rollout motion of the heart because the hiatus had collapsed (arrow).



Figure 3 Computed tomography sagittal plain, regular hiatal area as reference. The aorta remains dorsal while the oesophagus ascends in a concave curve (arrows), closely connected to the heart to finally reach its ventral passageway through the hiatus. The angle of the ascent of the oesophagus is about 45 degrees. A, dorsal; E, oesophagus; Ht, heart; H, hiatus; T, trachea.

not discernible. However, a clear difference was observed in the distal oesophagus and hiatus area. The crura, which usually secure the oesophagus in its crucial ventral position, proved to be fallen apart preoperatively, and the oesophagus now passed flattened in its course straight through the diaphragm, far dorsally, and almost parallel to the aorta (*Video 5*). Correspondingly, the regular angle of ascent of approximately 45° (*Figure 3*) decreased to $10-15^{\circ}$ and the



Figure 4 MRI sagittal plain, preoperative. At the origin of the cardio-oesophageal interaction (circle), the oesophagus appeared to be as firmly anchored preoperatively as postoperatively. However, distally, the loss of crural support in the hiatus resulted in a marked dorsal shift of the oesophagus almost parallel to the aorta (arrows). The oesophagus was far distant from the cardiac pressure zone (double arrow). E, oesophagus; MRI, magnetic resonance imaging.

oesophagus was significantly distanced from the cardiac pressure zone (*Figure 4*). In particular, the oesophageal area of maximum compression by the cardiac impulse lost its contact with the heart, and the close three-dimensional connection was disintegrated (*Video 5*, *Figure 4*). In contrast, postoperative imaging showed that surgical hiatus reconstruction was successful in returning the oesophagus to its ventral passage through the hiatus and its concave ascent along the heart (*Figure 5*).

Discussion

The findings of this study seem to point to a new hypothesis regarding the physiology of oesophagus function based on the complex interaction of organs. In particular, the data indicate the crucial role of the three-dimensional location of the oesophagus in the hiatal passage through the diaphragm, thus answering the initial question of why surgical repositioning of the oesophagus alone can fully restore reflux control and other related functions. Comparable observations are not available in the literature. Therefore, the data are discussed in terms of falsification or plausibility with respect to existing studies and clinical observations.

The observed downward rollout compression of the heart technically resembles a continuously running peristaltic pump, commonly known as a roller pump. Roller pumps are based on pulsation-free slow rotation, and, therefore, require a semicircular rotor-tube contact with a reversed



Figure 5 MRI sagittal plane, postoperative. The oesophageal hiatal unit has been surgically restored and the oesophagus rises concavely along the heart. The pressure wave of the heart can be transmitted again to the anterior wall of the oesophagus (arrows). The angle of ascent of the oesophagus is again about 40–50 degrees. E, oesophagus; H, hiatus; MRI, magnetic resonance imaging.

pumping direction. In contrast, the cardioesophageal pump is driven by a rapid downward cardiac rollout impulse along the oesophagus. This allows the contact zone between the pump and the tube to be reduced to a small segment of a circle, thus creating the crucial feature of an antegrade pumping direction (*Figure 6*). However, a decisive ascent of the oesophagus into this concave segment of the circle, and thus, close contact with the heart pump is still necessary. These are secured by the hiatus, which guides the oesophagus to its necessary ventral passage through the diaphragm. This set-up describes a clockwork-like three-dimensional interaction of the heart, oesophagus, and diaphragm, collectively referred to here as the cardiooesophageal and diaphragm interaction system (CODIS).

With this new pathophysiological perspective, the heart appears to be the central motor providing the driving rollout impulse to the oesophagus as an energy-free byproduct via the countermovement of systolic cardiac contraction (*Figure 7*). It appears that the oesophagus is predominantly the passive recipient of the cardiac rollout motion, and its known soft thin wall is ideal for effective impulse transmission. The three-dimensional architecture of the system, the crucial oesophageal angle of ascent and, thus, the length and intensity of cardioesophageal coupling is morphologically determined by the oesophagus hiatal unit.



Figure 6 Schematic illustration of a peristaltic pump transformation, sagittal plane. (A) Peristaltic roller pump pulsation-free rotating with 180 degrees tube-pump contact (curved arrows) leading into a reverse pumping direction (straight arrows). (B) Transformation from rotation to pulsing. (C) The pulsating cardioesophageal pump is driven by a fast downward rollout impulse on the ascending tightly connected tube. This allows the contact zone between tube and pump to be significantly reduced and an antegrade pumping direction to be achieved. However, significant elevation of the tube through the crura is required to ensure close contact with the heart and regular function. T, tube; C, crura.

The surprisingly rapid clearance of the oesophagus found in X-ray contrast swallow evaluations was described early on by Helm, suggesting that oesophageal clearance is maintained extremely rapidly by just one or two suggested peristaltic sequences (10). However, no peristaltic contractile activity could be detected in the patients studied, which is hardly to be expected in principle with only 1–2 mm thin, exclusively slow-acting fibres in the distal oesophagus. Furthermore, the dorsal oesophageal wall was completely



Figure 7 X-ray contrast swallow evaluation sagittal plane, postoperative. Combined schematic visualization of the cardio-oesophago-diaphragmatic interaction system. The crura safeguard the crucial ascent of the oesophagus and its tight connection to the heart (fine arrows). Long arrows show the pulsating rollout impulse of the heart on the ventral oesophageal wall.

unaffected during this emptying process, so that some kind of asymmetric semicircular contraction of the oesophageal musculature would have to be postulated. However, there is no conclusive evidence for this (5). This gap could be filled by assuming that the one or two rapid peristaltic sequences described for oesophageal clearance may in fact reflect rapid rollout motion by one or two heartbeats along the oesophageal wall (*Figure 1*).

Similarly, Lin *et al.* described the four phases of oesophageal bolus transit in terms of pressure, velocity, and topography (11). These data show, among other things, the transition of the bolus from the low-pressure transition zone to the high-pressure oesophageal phase III, which corresponds exactly to the takeover by the cardioesophageal pump. This phase ends distally at the so-called contractile deceleration point, which is described as a sudden but unknown muscular or neurogenic collapse within the oesophageal wall. Further, this point exactly marks the end of the powerful cardiac rollout movement just before the gastroesophageal junction.

Many other aspects of the analysed swallowing process seem to precisely correspond to the results of CODIS, however from a different physiological perspective.

Accordingly, the disconnection of the oesophagus from the cardioesophageal pump would also be expected to impair the physiological swallowing process. In fact, frequent symptoms found in hiatal hernia patients include unexplained functional symptoms such as dysphagia, globus sensation, aerophagia, rapid eating, and frequent belching.

Most importantly, the vital function of the oesophageal clearance should be re-evaluated. Due to the lifethreatening potential of every drink or meal by fatal aspiration, oesophagus clearance has to be very rapid, reliable, and completely independent of effects such as posture, movement, or awareness. This undoubtedly requires a permanent and unobstructed outflow option from the oesophagus at all times. Any barrier or even minimal latency in the opening would constantly pose a risk for a sudden life-threatening aspiration. This danger would inevitably be present with a complex controlled nervemuscular sphincter system of even smooth, slow-working muscles in the distal oesophagus.

In addition, it has to be considered that the rapid clearance function would not demand reflexive closing but the immediate reflexive opening of a hypothetical LES. However, smooth muscle sphincter opening is a very slow and passive procedure of decreasing activation. In any case, observation of LES activity in one direction or the other during gastroscopy is not described in the literature.

However, the indispensable requirement of a continuously opened and undisturbed outflow from the oesophagus requires a highly effective backflow protection. CODIS may actually ensure these two functions of rapid clearance and powerful backflow protection simultaneously. However, the underlying mechanism appears to be different from the one that is commonly discussed. MRI suggests that reflux control is not due to an actual closure of the oesophagus itself, but to functional prevention of reflux by a continuous pulsating counter-current directly in front of the gastroesophageal junction. On the basis of 70 heartbeats per minute, such a system generates a highly efficient downward current with a frequency of >100,000 pulses/day, energy-free as the side effect of the heartbeat and lifelong independent of body posture, stomach-filling, sport, illness, oesophagitis, consciousness, etc., as long as the heart beats.

In this way, the gastroesophageal junction may remain fundamentally open, reliable clearance is ensured at any time, inevitable reflux is simultaneously controlled, and the life-threatening risks of outflow obstruction by a complex reflex-controlled sphincter system are excluded. Indeed, the continuously pulsing downward counter-current in the gastroesophageal junction was only detectable in patients postoperatively (*Video 4*).

Data show that CODIS seems to be highly reliant on the three-dimensional arrangement of its parts. The oesophagus hiatal unit secures the oesophagus in its crucial



Figure 8 Schematic illustration of the origin of reflux. (A) Stable crura keep the oesophagus in its ventral position distanced from the aorta. They ensure a regular concave ascent of the oesophagus by about 45 degrees and thus the decisive functional coupling to the pressure zone of the heart. (B) Collapse of the oesophageal hiatal unit. The crura spilt apart and the oesophagus slipped dorsally. The angle of ascent is flattened and the oesophagus is decoupled from the heart. The three-dimensional composition of the cardioesophageal pump and thus its function is impaired. C, crura; E, oesophagus; A, aorta.

ventral passageway through the diaphragm with the help of the superior and inferior phrenoesophageal ligaments and by the dorsal support of the columnar crura. Risks for this structure can arise from various factors, such as tenderness of the fascia-free crura muscles, their unprotected position with anchorage in the mobile centrum tendineum, anatomical variations, and permanent axial and bilateral forces (12,13). Any harm, especially the loss of crural support, leads to the dorsal displacement of the oesophagus from the cardiac contact area and jeopardises impulse transmission and related functions (*Figure 8*).

The importance of the oesophagus hiatal unit for reflux control was studied in an experimental *ex vivo* peristaltic antegrade roller pump system working against the pressure of an elevated water reservoir (14). The success of reflux control in the tube was crucially dependent on the angle of inclination of the tube in the roller pump. Increasing the tube angle prevented reflux from the reservoir, decreasing it and thus simulating hiatal damage allowed reflux. These experimental results are consistent with clinical data of 1,351 patients showing that surgical reconstruction of the oesophagus angle alone fully restores oesophageal function (9). Therefore, GERD should not be understood as an unknown disease of the oesophagus itself, but as a consequence of its displacement in CODIS due to a yielding oesophagus hiatal unit.

In the absence of comparable data on CODIS, it seems necessary to apply this hypothesis to various clinical observations. Therefore, besides swallowing, clearance, and reflux control, the fourth vital function of the distal oesophagus is vomiting. Given a powerful and fast-acting reflux control system such as CODIS, vomiting seems impossible without competitive struggle between the contracting stomach and the cardioesophageal pump. Thus, a significant restriction of CODIS function appears to be urgently required to at least temporarily override the powerful closure mechanism in the gastroesophageal junction and allow vomiting and perhaps other forms of retrograde passage to occur.

The heart pump itself must not be inhibited. However, considering the direction of the muscle fibres in the crural diaphragm, which anatomically belong to the striated abdominal muscle shell (15), a strong core contraction should pull the oesophagus down in the hiatus, similar to a pathological dorsal oesophageal dislocation in reflux patients. This should flatten the angle of the ascent of the oesophagus and interrupt the cardioesophageal junction, similar to the situation in reflux patients. Powerful CODIS reflux control would significantly be impaired for a short time, facilitating vomiting and retrograde flow. This approach may be a physiological response to the conflict between strong and reliable reflux prevention and the need to overcome this function in vital situations.

A system like CODIS should structurally have at least three vulnerable components of clinical significance. First, the functional importance of the architectural arrangement of all components, especially the oesophagus hiatal unit, has been demonstrated. Second, the efficient transmission of cardiac impulse requires a smooth and tender oesophagus wall. Consequently, any form of wall induration and thickening should significantly impair the transmission of the cardiac impulse in various degrees, causing dysfunction. In fact, this is what happens in primary achalasia, pseudoachalasia, and severe inflammatory disease (16). Dysphagia, regurgitation, oesophagus distension, and uncoordinated secondary and tertiary contractions of the oesophagus may occur to overcome CODIS impairment, especially in the early stages and to varying degrees. Breaking the indurated oesophageal sheath by surgical myotomy or pneumatically and thus facilitating the

transmission of cardiac impulses to the oesophagus could contribute to the success of the therapy (17).

Third, the heart as the central engine of CODIS must also be considered a fundamentally vulnerable component. The continuous, rhythmic rollout of the ventricle along the oesophagus seems to be fundamental for all CODIS functions. Therefore, an interruption of rhythmic ventricular motion should lead to a dysfunction of the system. This assumption has been confirmed by studies showing that the occurrence of frequent premature ventricular complexes and cardiac arrhythmias immediately leads to dysphagia and loss of reflux control (18).

The close relationship between the heart and the oesophagus has long been known described as the oesophagocardial reflex. Acid exposure or distension of the oesophagus, thus mimicking reflux and food stasis, easily leads to cardiac effects such as arrhythmia and supraventricular tachycardia (19,20). The background of this neural interconnection remains unclear. However, in the light of such a complex, interacting system as CODIS, an intense neural interweaving of all the organs involved even seems to be imperative. Therefore, an acute threat to the vital functions of reflux control and clearance through acid exposure and wall dilatation, as simulated in the studies, could plausibly trigger direct activating impulses from the oesophagus to the heart as the organ responsible for these functions. Indeed, there is increasing clinical evidence of cardiac symptoms in GERD patients (21-23).

Various clinical findings and observations appear explainable against the background of the new hypothesis of CODIS. The results presented were clearly limited by the small number of patients, the lack of a control group, and the examinations could not show all described functional aspects in detail in all patients at the same time. Furthermore, no concomitant manometric studies were performed. This could be of great interest because although the oesophagus is not the main player in this system, it seems likely that the transmission of pressure from the heart pump to the oesophageal lumen can be influence by relaxing, contracting and changing the tone of the muscles in terms of fine-tuning. So, the actual process will certainly go beyond this data. However, it may be the first step to develop a new overall concept of this complex organ interaction.

Conclusions

The data presented here are new and preliminary and

comparable references in the literature are not available. However, the results point towards a new and surprisingly plausible physiological hypothesis of oesophageal function that can explain many clinical findings and observations. The identification of the heart as the central antireflux organ in a complex interaction with the oesophagus and the diaphragm reveals a highly effective biological solution that meets all the specific requirements of the distal oesophageal segment. The most significant weakness of the system appeared to be the oesophagus hiatal unit, which occupies the key architectural position for CODIS functionality. Reflux disease thus appears to be due to a malposition of the oesophagus in CODIS rather than a malfunction as a result of an unidentified disease of an anatomically nonexistent LES. The presented pathophysiological hypothesis based on CODIS clearly contradicts the common LES concept. It conclusively provides an answer to the initial question of why the surgical approach of reconstructing the oesophagus hiatal unit alone, without fundoplication or other antireflux procedures, results in a significant recovery of all oesophageal functions (10). It is hoped that further research will uncover more details of this fascinating oesophagocardiac network in humans, paving the way to a fundamentally new understanding of the oesophagus and its functioning.

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Footnote

Reporting Checklist: The author has completed the CARE reporting checklist. Available at https://ls.amegroups.com/article/view/10.21037/ls-22-22/rc

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Conflicts of Interest: The author has completed the ICMJE uniform disclosure form (available at https://ls.amegroups.com/article/view/10.21037/ls-22-22/coif). The author has no conflicts of interest to declare.

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Ethical Statement: The author is accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedure performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patients for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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References

- Goyal RK, Chaudhury A. Pathophysiology of normal oesophageal motility. J Clin Gastroenterol 2008;42:610-9.
- Lenglinger J, See SF, Beller L, et al. The cardia: esophageal or gastric? Critical reviewing the anatomy and histopathology of the esophagogastric junction. Acta Chir Iugosl 2012;59:15-26.
- Gill KR, Ghabril MS, Jamil LH, et al. Variation in Barrett's esophageal wall thickness: is it associated with histology or segment length? J Clin Gastroenterol 2010;44:411-5.
- Herbella FAM, Schlottmann F, Patti MG. Pathophysiology of gastroesophageal reflux disease: how an antireflux procedure works (or does not work). Updates Surg 2018;70:343-7.
- Herregods TV, Bredenoord AJ, Smout AJ. Pathophysiology of gastroesophageal reflux disease: new understanding in a new era. Neurogastroenterol Motil 2015;27:1202-13.
- Mikami DJ, Murayama KM. Physiology and pathogenesis of gastroesophageal reflux disease. Surg Clin North Am 2015;95:515-25.
- Boeckxstaens G, El-Serag HB, Smout AJ, et al. Symptomatic reflux disease: the present, the past and the future. Gut 2014;63:1185-93.

- 8. Chatila AT, Nguyen MTT, Krill T, et al. Natural history, pathophysiology and evaluation of gastroesophageal reflux disease. Dis Mon 2020;66:100848.
- 9. Löhde EH, Thomas F. Results of a prospective, uncontrolled, single-arm study of 1,351 patients with hiatal hernia operated on exclusively with DeltaMesh hiatal reconstruction over a 10-year period. Laparosc Surg 2022;6:22.
- Helm JF. Esophageal acid clearance. J Clin Gastroenterol 1986;8 Suppl 1:5-11.
- Lin Z, Yim B, Gawron A, et al. The four phases of esophageal bolus transit defined by high-resolution impedance manometry and fluoroscopy. Am J Physiol Gastrointest Liver Physiol 2014;307:G437-44
- Loukas M, Wartmann ChT, Tubbs RS, et al. Morphologic variation of the diaphragmatic crura: a correlation with pathologic processes of the esophageal hiatus? Folia Morphol (Warsz) 2008;67:273-9.
- Costa MM, Pires-Neto MA. Anatomical investigation of the esophageal and aortic hiatuses: physiologic, clinical and surgical considerations. Anat Sci Int 2004;79:21-31.
- 14. Wiezcorek A. Development of an ex-vivo model of the cardio-oesophageal peristaltic pump. (Aufbau eines ex-vivo Modells zur gastroösophagealen Refluxkrankheit). Institute of Biotechnology, Faculty III, Medical Biotechnology, Technical University Berlin, Germany. Master's thesis 2015:1-86. Available from: https://www.medtech.tu-berlin. de/menue/kontakt/aktuelle_mitarbeiterseiten/prof_dr_ ing_m_kraft
- Sefton EM, Gallardo M, Kardon G. Developmental origin and morphogenesis of the diaphragm, an essential mammalian muscle. Dev Biol 2018;440:64-73.
- Ates F, Vaezi MF. The Pathogenesis and Management of Achalasia: Current Status and Future Directions. Gut Liver 2015;9:449-63.
- Moonen A, Boeckxstaens G. Current diagnosis and management of achalasia. J Clin Gastroenterol 2014;48:484-90.
- Stec S, Tarnowski W, Binda A, et al. Videofluoroscopic modified barium swallow study for premature ventricular complexes-associated dysphagia. Circ Arrhythm Electrophysiol 2008;1:e1.
- Wright RA, McClave SA, Petruska J. Does physiologic gastroesophageal reflux affect heart rate or rhythm? Scand J Gastroenterol 1993;28:1021-4.
- Makk LJ, Leesar M, Joseph A, et al. Cardioesophageal reflexes: an invasive human study. Dig Dis Sci 2000;45:2451-4.

- 21. Maruyama T, Fukata M, Akashi K. Association of atrial fibrillation and gastroesophageal reflux disease: Natural and therapeutic linkage of the two common diseases. J Arrhythm 2019;35:43-51.
- 22. Roman C, Bruley des Varannes S, Muresan L, et al. Atrial fibrillation in patients with gastroesophageal reflux

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disease: a comprehensive review. World J Gastroenterol 2014;20:9592-9.

23. Fuertes Á, Alshweki A, Pérez-Muñuzuri A, et al. Supraventricular tachycardia in newborns and its association with gastroesophageal reflux disease. An Pediatr (Barc) 2017;87:206-10.