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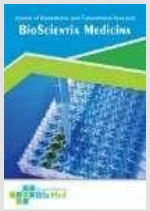

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Intraoperative Vagal Excitation During Rigid Esophagoscopy for an Esophageal Earring in a 4-Month-Old Infant: A Case Report

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A B S T R A C T

Background: Esophageal foreign body ingestion is a prevalent otorhinolaryngologic emergency in the pediatric population but is epidemiologically rare in infants under six months of age. While rigid esophagoscopy remains the gold standard for extraction, it involves stimulating the highly innervated aerodigestive tract. This case highlights a life-threatening vagal reflex—an underreported complication in infants—during foreign body removal. **Case presentation:** A 4-month-old female infant presented with a history of accidental earring ingestion three days prior, manifesting as hypersalivation and feeding refusal. Radiographic imaging confirmed a radiopaque foreign body at the T1 vertebral level or thoracic inlet. The patient underwent rigid esophagoscopy under general anesthesia. During the extraction phase, mechanical manipulation of the esophageal mucosa triggered profound vagal excitation, resulting in severe bradycardia and oxygen desaturation. The procedure was immediately paused, and the patient was successfully resuscitated using vagolytic agents and hyperoxygenation by the anesthesiology team. A second attempt was successful without recurrence of the reflex. Post-operative recovery was uneventful. **Conclusion:** Foreign body ingestion in early infancy requires a high index of suspicion and meticulous perioperative planning. The manipulation of the esophageal inlet can trigger potent vagovagal reflexes, particularly in infants with high vagal tone. This case underscores the necessity of deep anesthetic planes, prophylactic vagolytic preparation, and seamless communication between the surgeon and anesthesiologist to manage hemodynamic instability.

1. Introduction

Foreign body ingestion represents a pervasive and significant clinical burden within the landscape of pediatric emergency medicine, accounting for a substantial and rising number of hospital admissions and urgent interventions globally. This phenomenon is not merely a medical curiosity but a frequent public health challenge that demands substantial healthcare resources and vigilance.[1] The epidemiological data paint a clear picture of the typical patient: a toddler between the ages of one and three years. This peak incidence is inextricably linked to early childhood development, specifically the oral phase, a critical developmental stage characterized by the innate drive to explore the environment through oral tactile sensory processing. For the toddler, placing an object in the mouth is a mechanism of learning; however, this exploratory behavior frequently precipitates accidental ingestion.

In stark contrast to this well-documented toddler demographic, the occurrence of foreign body ingestion in infants under the age of six months is exceptionally rare and statistically anomalous.[2] At this rudimentary stage of motor development, an infant lacks the fine motor coordination, pincer grasp, and intentionality required to independently locate, grasp, and place a

foreign object into the oral cavity. Consequently, the etiology of ingestion in this youngest age group differs fundamentally from that of older children. Ingestion in early infancy is almost exclusively an accidental and passive event, rather than an active exploratory one. It is frequently secondary to environmental factors, such as the actions of older siblings who may feed the infant, the inadvertent negligence of caregivers, or the unsafe proximity of small objects—such as jewelry or safety pins—within the infant's sleeping or play environment. This distinct mechanism of injury necessitates a higher index of suspicion from clinicians, as the history may be vague and the presentation non-specific.

To understand the severity of this condition, one must appreciate the complex geography of the pediatric esophagus. The esophagus is not merely a passive conduit; it is a dynamic muscular tube connecting the pharynx to the stomach, characterized by specific physiological constrictions that serve as anatomical bottlenecks for ingested objects.[3] In the standard anatomical model, three distinct areas of narrowing are identified where foreign bodies are most prone to impaction. The first is the proximal constriction at the cricopharyngeal muscle (approximately the C6 vertebral level), which forms the upper esophageal sphincter. The second is the middle constriction, created by the external compression of the aortic arch and the left main bronchus (approximately the T4 vertebral level). The third is the distal constriction at the lower esophageal sphincter (approximately the T10 vertebral level), where the esophagus traverses the diaphragm.

While these landmarks are universal, the dimensions in an infant are drastically reduced, altering the clinical risk profile.[4] The anatomy of a 4-month-old is proportionally smaller and less distensible than that of an older child or adult. Anthropometric studies indicate that in a 3-kg infant, the esophageal diameter varies precariously between only 4.8 mm and 8.48 mm. Furthermore, the length of the esophagus in an infant of this size is significantly shorter, estimated at approximately 8.18 cm based on predictive height-based equations. Within such confined biological parameters, even objects that would be considered trivial in an adult—such as a small earring or a safety pin—become highly obstructive, capable of causing complete occlusion or significant mucosal pressure. The thoracic inlet, corresponding roughly to the T1 vertebral level, is a particularly critical zone. Here, the esophagus transitions from the cervical to the thoracic compartment. This area is anatomically distinct because the esophagus lacks a serosal layer and is less distensible, creating a rigid choke point. Impaction at this level is not only common but fraught with danger due to the proximity of vital mediastinal structures and the airway.[5]

Given the anatomical risks, the management protocols for esophageal foreign bodies overwhelmingly favor prompt endoscopic retrieval. While flexible endoscopy is an option, rigid esophagoscopy remains the traditional gold standard and the most effective method for extracting impacted objects in the pediatric population, particularly when the object is sharp, metallic, or possesses a complex shape like an earring.[6] The rigid esophagoscope allows for superior optical visualization, the use of robust grasping forceps (such as alligator forceps), and the ability to shield the sharp edges of a foreign body within the lumen of the scope during withdrawal, thereby protecting the mucosal walls. However, the efficacy of rigid esophagoscopy comes at a cost; the procedure is not benign. It requires the insertion of a rigid metal tube into a soft, edematous, and highly friable aerodigestive tract. The mechanical risks are well-known and include mucosal laceration, perforation, and mediastinitis. Yet, beyond these mechanical hazards lies a category of physiological complications that are often underappreciated but pose immediate, life-threatening risks: neurogenic reflexes.

The aerodigestive tract is not an inert pipe; it is a sensory-rich organ densely innervated by the autonomic nervous system. Specifically, the larynx, pharynx, and proximal esophagus receive extensive

sensory innervation from the vagus nerve (Cranial Nerve X) via its branches, the superior laryngeal nerve and the recurrent laryngeal nerve. These nerves are responsible for potent airway protection reflexes, guarding the entrance to the respiratory tree.[7] Mechanical distension, traction, or manipulation of the esophageal mucosa in these highly innervated zones—such as that occurring during the disimpaction of a foreign body—can trigger a potent vagovagal reflex arc, often described as the esophago-cardiac reflex. The pathophysiology of this reflex is profound. Mechanical stimulation of the esophageal wall activates afferent sensory fibers that travel via the vagus nerve to the brainstem, terminating in the nucleus tractus solitarius (NTS) in the medulla oblongata. The NTS acts as an integration center for visceral sensation. From the NTS, excitatory signals are relayed to the dorsal motor nucleus of the vagus, which serves as the primary efferent center.[8]

Activation of the dorsal motor nucleus results in a massive, unchecked increase in parasympathetic (vagal) output directed to the heart, specifically the sinoatrial (SA) node. At the cellular level, this vagal discharge causes a rapid release of acetylcholine, which binds to muscarinic receptors on the cardiac pacemaker cells, inducing hyperpolarization. The clinical manifestation of this reflex is abrupt and terrifying: precipitous bradycardia, decreased cardiac output, and, in severe cases, asystole or cardiac arrest. This hemodynamic collapse can lead to secondary hypoxemia, further compounding the physiological crisis. Infants are uniquely vulnerable to this reflex due to their immature autonomic nervous system.[9] They possess a high resting vagal tone compared to older children and adults, making their threshold for triggering this reflex significantly lower. Consequently, what might be a minor stimulus in an adult can precipitate a cardiac crisis in a 4-month-old infant.

Despite the clearly defined anatomy and the theoretical understanding of autonomic physiology, the medical literature specifically documenting severe vagal excitation during esophageal foreign body removal in infants under one year of age remains limited. The majority of existing case reports and retrospective studies focus predominantly on surgical techniques, the types of objects ingested, or mechanical complications such as perforation and mediastinitis. There is a paucity of detailed clinical reports analyzing the hemodynamic sequelae of esophageal manipulation in early infancy, specifically regarding the esophago-cardiac reflex. This paper addresses this critical gap by presenting a rare and instructional case of a 4-month-old infant who developed severe intraoperative vagal excitation, characterized by profound hemodynamic instability, during the rigid endoscopic removal of an ingested earring.[10]

The primary aim of this case report is to move beyond the surgical narrative and provide a comprehensive pathophysiological analysis of the esophago-cardiac reflex in the context of pediatric rigid esophagoscopy. We aim to detail the neuroanatomical pathway of the vagovagal reflex triggered by esophageal distension, underscore the unique physiological susceptibility of infants under six months to high-tone vagal events, and critically evaluate the role of the anesthesiologist and the necessity for specific safety measures, including deep anesthetic planes and the prophylactic availability of vagolytic agents, to mitigate these risks. This study is novel in its specific focus on the physiologic rather than mechanic complications of rigid esophagoscopy in the rare demographic of early infancy (under 6 months). By documenting the successful management of a life-threatening reflex through interdisciplinary collaboration, this report serves as a vital educational tool for otolaryngologists and anesthesiologists, advocating for a shift in perioperative planning from purely anatomical considerations to a holistic physiological approach.

2. Case Presentation

Written informed consent was obtained from the patient's legal guardians (parents) for the publication of this case report and any accompanying images. The

parents were fully briefed on the nature of the report and the protection of the patient's anonymity. All procedures performed were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments.

A 4-month-old female infant, weighing 8 kg with a height of 61 cm, was referred to the Emergency Department of Dr. M. Djamil General Hospital Padang from a district hospital. The chief complaint was the suspected ingestion of a gold earring three days prior to admission. According to the alloanamnesis provided by the mother, the infant was sleeping alone when she suddenly woke up crying loudly. Upon attempting to soothe the infant, the parents noticed that one of the infant's earrings was missing. Over the subsequent 72 hours, the infant became increasingly irritable and demonstrated distinct behavioral changes, including a refusal to breastfeed and excessive drooling. The parents noted the infant appeared to have difficulty swallowing and pain upon opening the mouth. There was no history of cyanosis, coughing, choking, or respiratory distress immediately following the event, suggesting the airway was patent. No prior attempts at manual extraction had been made by the family.

Upon admission, the patient was moderately ill but alert and compos mentis (Table 1). Vital signs were within the upper limits of normal for age, likely due to stress and mild dehydration. The pulse rate was 120 beats per minute, the respiratory rate was 30 breaths per minute, the body temperature was 36.9 degrees Celsius, and the oxygen saturation was 99% on room air. Physical examination of the head and neck revealed no external abnormalities. Oropharyngeal examination was limited due to the patient's irritability but showed no pooling of secretions in the oral cavity proper. Thoracic auscultation demonstrated symmetrical chest expansion with vesicular breath sounds bilaterally; no stridor, wheezing, or rhonchi were audible, ruling out immediate airway compromise or foreign body aspiration into the tracheobronchial tree.

Laboratory investigations were conducted to assess for infection or electrolyte imbalance due to poor intake. Results were unremarkable: Hemoglobin 9.8 g/dL, Leukocytes 14,760/mm^3, and Platelets 519,000/mm^3. Renal function and electrolytes, including Sodium 136 mmol/L and Chloride 109 mmol/L, were within normal limits. Radiographic imaging was decisive. An anteroposterior and lateral cervical X-ray with extension positioning revealed a radiopaque foreign body with the configuration of an earring lodged in the proximal esophagus (Figure 1). The object was projected at the level of the T1 to T4 vertebrae. The sharp hook portion appeared directed superiorly. The tracheal air column remained patent with no deviation or compression effects visible on the lateral view. The chest X-ray showed clear lung fields with no evidence of pneumomediastinum or subcutaneous emphysema, suggesting no perforation had occurred prior to admission. A sketch provided by the parents indicated the earring was approximately 2 cm in length with a potentially sharp clasp.

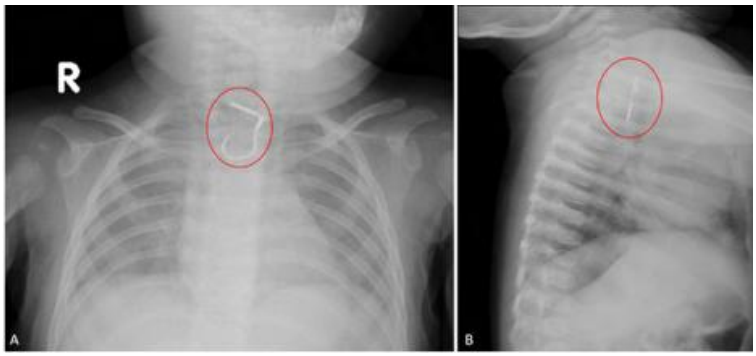

Figure 1. Radiographic examination; (A) Thorax X-ray, (B) lateral cervical X-ray, (red ring) foreign body.

TABLE 1. SUMMARY OF CLINICAL FINDINGS ON ADMISSION

1. Patient Demographics & Profile	
Age / Sex	4 Months / Female
Anthropometry	Weight: 8 kg | Height: 61 cm
General State	Moderately ill, Alert, Compos Mentis
2. History of Present Illness	
Chief Complaint	Suspected ingestion of Gold Earring
Duration of Impaction	3 Days (72 hours) prior to admission
Mechanism	Passive/Accidental (Infant was sleeping alone; sudden crying; earring noted missing by parents)
3. Vital Signs (Upper Limit of Normal)	
Heart Rate	120 beats per minute
Respiratory Rate	30 breaths per minute
Body Temperature	36.9°C
Oxygen Saturation (SpO2)	**99% on Room Air**
4. Specific Symptoms (Esophageal Phase)	
Behavioral Changes	Increased irritability, crying
Gastrointestinal / Oral	**Hypersalivation** **Refusal to Breastfeed** **Excessive Drooling** **Odynophagia (Pain on opening mouth)**
5. Physical Examination Findings	
Head & Neck	No external masses; No cervical crepitus
Oropharynx	No pooling of secretions visible in oral cavity proper (exam limited by irritability)
Thorax / Respiratory	Symmetrical chest expansion Vesicular breath sounds bilaterally **No Stridor, No Wheezing, No Rhonchi** (Rules out aspiration)

The patient was diagnosed with an esophageal foreign body at the thoracic inlet. Given the sharpness of the object and the duration of impaction, urgent removal was indicated to prevent perforation or mediastinitis. The patient was classified as American Society of Anesthesiologists (ASA) class II due to the pediatric age and urgency of the procedure. Consultations with the Department of Pediatrics and the Department of Anesthesiology confirmed there were no contraindications for surgery.

The patient was transferred to the operating theater and placed in the supine position with standard ASA monitoring (ECG, NIBP, SpO_2, and Precordial Stethoscope). Pre-induction vital signs showed a heart rate (HR) of 135 bpm and oxygen saturation (SpO_2) of 99% on room air. General anesthesia was induced via inhalation with Sevoflurane (8%) in 100% oxygen. Following loss of consciousness, intravenous access was established. To facilitate rigid esophagoscopy and prevent patient movement or laryngospasm, muscle relaxation was achieved using intravenous Rocuronium (0.6 mg/kg). The airway was secured with a size 3.5 mm uncuffed endotracheal tube (ETT). Anesthesia was maintained with Sevoflurane (2.0 vol%) and a continuous Propofol infusion (100 mcg/kg/min) to ensure adequate depth. The patient was mechanically ventilated to maintain normocapnia.

TABLE 2. DIAGNOSIS, TREATMENT, FOLLOW UP & OUTCOME

1. Diagnostic Investigation	
Laboratory Profile	▪ **Hemoglobin:** 9.8 g/dL (Mild Anemia) ▪ **Leukocytes:** 14,760 /mm³ (Mild Leukocytosis) ▪ **Platelets:** 519,000 /mm³ (Normal) ▪ **Electrolytes:** Normal (Na 136, Cl 109 mmol/L)
Radiographic Findings (Cervical X-Ray)	**Foreign Body Detected** **Location:** T1 to T4 Vertebral Level (Thoracic Inlet). **Configuration:** Radiopaque, earring-shaped, sharp hook directed superiorly. **Airway:** Tracheal air column patent; No deviation. **Complications:** No pneumomediastinum or surgical emphysema visible.
Final Diagnosis	**Esophageal Foreign Body (Earring) at Thoracic Inlet**
2. Operative Management	
Procedure	Rigid Esophagoscopy under General Anesthesia (GA)
Anesthetic Protocol	**Induction:** Sevoflurane 8%. **Muscle Relaxant:** Rocuronium 0.6 mg/kg (IV). **Airway:** Intubated with **ETT Size 3.5 mm (Uncuffed)**. **Maintenance:** Sevoflurane 2.0 vol% + Propofol Infusion.
Instrumentation	10x12 Rigid Esophagoscope (Length 20 cm) + Alligator Forceps.
Intraoperative Complication	**Severe Vagal Excitation (Esophago-Cardiac Reflex)** Triggered by traction on the foreign body. **Findings:** Precipitous Bradycardia (128 → 55 bpm) followed by Desaturation (99% → 82%).
Rescue Management	1. Immediate cessation of stimulus (scope withdrawal). 2. **Vagolysis:** Atropine 0.02 mg/kg IV administered. 3. **Oxygenation:** 100% O2 ventilation. 4. **Anesthetic Depth:** Propofol Bolus 1 mg/kg.
Surgical Outcome	**Attempt 2:** Successful extraction after hemodynamic stabilization. **Mucosal Assessment:** Laceration at 11 cm; mild edema; no active bleeding.
3. Postoperative Course & Outcome	
Medical Therapy	IV Fluid Resuscitation (Ringer Lactate) Antibiotic: Oral Cefixime Analgesia: Paracetamol
24-Hour Evaluation	**Stable** Afebrile, tolerating breastfeeding well. No respiratory distress or cervical crepitus.
Discharge Status	Discharged on Postoperative Day 1.
Follow Up (1 Week)	Patient asymptomatic with normal feeding habits. Complete recovery.

A 10x12 rigid esophagoscope (20 cm length) was inserted using the standard technique. The foreign body was visualized at 10 cm from the upper incisors, embedded in the mucosal fold at the T1 level. The surrounding mucosa exhibited mild edema. During the extraction phase, the surgeon grasped the earring with alligator forceps. At the moment traction was applied to disimpact the sharp hook from the esophageal wall, the patient developed sudden, severe hemodynamic instability. The heart rate dropped precipitously from a baseline of 128 bpm to a nadir of 55 bpm (sinus bradycardia) within 8 seconds. This

was immediately followed by a decrease in oxygen saturation from 99% to 82%. This sequence—bradycardia preceding desaturation during mechanical stimulation—confirmed a diagnosis of severe vagovagal reflex (esophago-cardiac reflex) rather than primary hypoxic cardiac arrest (Figure 2).

The surgeon immediately released the foreign body and withdrew the esophagoscope to the oropharynx to cease vagal afferent stimulation. Simultaneously, the anesthesia team intervened: (1) Vagolysis: Intravenous Atropine (0.02 mg/kg) was administered immediately; (2) Oxygenation: Ventilation with 100% oxygen was continued; (3) Deepening Anesthesia: A bolus of Propofol (1 mg/kg) was administered to suppress the autonomic reflex arc before re-attempting extraction. Vital signs returned to baseline (HR >110 bpm, SpO_2 100%) within 90 seconds following these interventions. See Table 1 for the comprehensive timeline of the event.

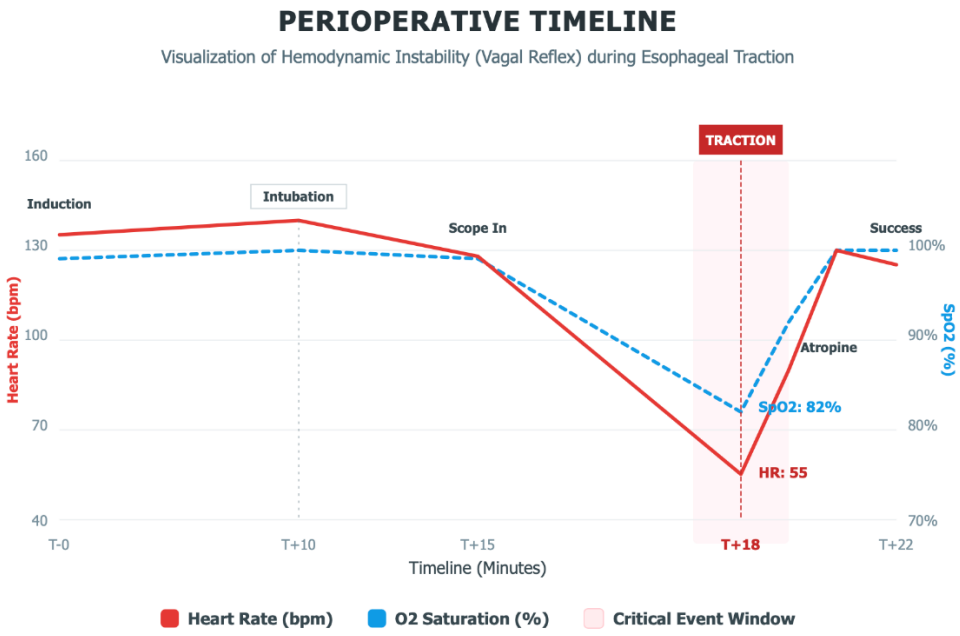

Figure 2. Perioperative vital signs and event timeline.

Once the patient was hemodynamically stable and a deeper plane of anesthesia was confirmed, a second attempt was made. The esophagoscope was re-inserted. The foreign body was visualized, and the head of the earring was securely grasped to shield the sharp point. The object was extracted using gentle, swift traction without recurrence of the vagal reflex. A re-evaluation esophagoscopy was performed to inspect the esophageal mucosa. A mucosal laceration was identified at 11 cm from the upper incisors, corresponding to the site of impaction. The area showed mild edema but no necrotic tissue and no active bleeding. The esophagoscope was withdrawn slowly. The extracted object was confirmed to be a gold earring measuring 2 cm in length.

The patient was monitored in the recovery room and subsequently transferred to the Otorhinolaryngology ward. Post-operative therapy included intravenous Ringer's Lactate fluid resuscitation, oral Cefixime, and Paracetamol for analgesia. At the 24-hour follow-up evaluation, the patient was afebrile, tolerating breastfeeding well, and displayed no signs of respiratory distress, cervical crepitus, or difficulty swallowing. Thoracic

examination remained normal. She was discharged on postoperative day one with instructions for outpatient follow-up. At the one-week follow-up visit, the patient remained asymptomatic with normal feeding habits.

3. Discussion

The case presented herein highlights a critical and underreported physiological hazard during the rigid endoscopic management of esophageal foreign bodies in early infancy: the esophago-cardiac reflex. While the mechanical removal of the foreign body—a gold earring—was ultimately successful, the intraoperative course was complicated by severe, life-threatening vagal excitation. This event serves as a pivotal learning opportunity, shifting the focus from mere surgical dexterity to a broader understanding of neurophysiology and anesthetic safety. The following discussion dissects the epidemiological anomalies of this case, provides a detailed anatomical and pathophysiological analysis of the reflex arc, and establishes a framework for perioperative safety in this vulnerable demographic.[11]

The ingestion of foreign bodies is a ubiquitous phenomenon in pediatric emergency medicine, typically following a predictable epidemiological curve that peaks during the toddler years (ages 1 to 3). This peak coincides with a distinct developmental stage characterized by increased mobility, the acquisition of a pincer grasp, and the oral phase of exploration, where objects are mouthed as a means of sensory learning.[12] In stark contrast, foreign body ingestion in a 4-month-old infant is a statistical and developmental anomaly. At four months of age, an infant's fine motor skills are rudimentary; the voluntary coordination required to locate, grasp, and deliberately place a small object into the mouth is not yet fully established. Consequently, ingestion in this demographic is almost invariably a passive or accidental event rather than an active exploratory one. The literature suggests that such incidents are frequently secondary to environmental factors, such as the actions of older siblings (sibling feeding), the inadvertent negligence of caregivers, or the unsafe proximity of small objects within the infant's sleeping or play environment.

In the present case, the ingestion of a gold earring likely occurred while the infant was sleeping alone, suggesting the object was loose in the bed linens or crib. This highlights a critical intersection between clinical medicine and public health: the necessity for rigorous parental education regarding safe sleep environments. The data further corroborates that jewelry and safety pins are among the most common hazardous objects ingested by female infants, often mediated by cultural practices involving early ear piercing or the use of safety pins for clothing. This distinct etiology demands a high index of suspicion from clinicians, as the history provided by caregivers may be vague, and the infant is unable to verbalize distress.[13]

A profound understanding of esophageal morphometry is a prerequisite for the pediatric endoscopist.[14] The infant esophagus is not simply a miniature version of the adult organ; it possesses unique dimensional and compliance characteristics that predispose it to foreign body impaction. While the adult esophagus measures between 20 to 30 cm, the infant esophagus is significantly shorter and narrower. To precisely correlate the clinical findings with anatomical landmarks, we employed predictive auxological equations. For our patient, with a height of 61 cm, the predicted esophageal length was calculated to be approximately 8.18 cm. The foreign body was visualized intraoperatively at 10 cm from the upper incisors. When accounting for the distance from the incisors to the cricopharyngeus, this location correlates precisely with the thoracic inlet (T1 vertebral level). This localization is clinically significant for three reasons. First, the thoracic inlet represents the first constriction of the thoracic esophagus, where the transition from the cervical esophagus occurs. Second, at this level, the esophagus is crowded by the rigid bony structures of the thoracic aperture and lacks a serosal layer, making it less distensible and more prone to pressure necrosis from impacted objects. Third, the diameter of

the esophagus in a 4-month-old infant is critically small, ranging between 4.8 mm and 8.48 mm. A 2 cm rigid metallic object, such as the earring in this case, essentially acts as a complete obturator in a lumen of this size, explaining the severe symptomatology of hypersalivation and feeding refusal.[15]

The most educational aspect of this case—and its primary contribution to the literature—is the documentation of the esophago-cardiac reflex. While surgeons are frequently vigilant regarding mechanical complications such as perforation or mediastinitis, the neurogenic risks of rigid esophagoscopy are often underestimated. The mechanism underlying the patient's precipitous bradycardia and desaturation involves a potent autonomic reflex arc similar to the well-known oculocardiac reflex (Trigemino-vagal) encountered in ophthalmic surgery.[16] The afferent limb of this reflex originates in the aerodigestive tract. The pharynx, larynx, and proximal esophagus are densely innervated by mechanoreceptors sensitive to stretch and distension. Sensory impulses from these receptors are carried via the superior laryngeal nerve (SLN) and the recurrent laryngeal nerve (RLN)—both branches of the vagus nerve (CN X)—to the brainstem. These afferent signals terminate in the nucleus tractus solitarius (NTS) located in the medulla oblongata. The NTS serves as the central processing unit for visceral sensation. Upon receiving high-intensity afferent input—such as the sudden mechanical distension caused by traction on an impacted foreign body—the NTS relays excitatory signals to the dorsal motor nucleus of the vagus (DMN).[17]

The efferent limb of the reflex begins at the DMN, which sends increased parasympathetic output back via the vagus nerve to the heart. Specifically, these fibers innervate the sinoatrial (SA) node and the atrioventricular (AV) node. The massive release of acetylcholine at nerve terminals acts on muscarinic (M2) receptors, inducing hyperpolarization of cardiac pacemaker cells. Clinically, this manifests as profound sinus bradycardia, sinus arrest, or complete AV block. The subsequent drop in cardiac output leads to rapid hemodynamic instability and secondary hypoxemia, creating a vicious cycle where hypoxia further potentiates vagal tone. In our 4-month-old patient, the traction applied to disimpact the earring likely caused acute distension of the edematous esophageal wall, firing this reflex arc. Infants are uniquely susceptible to this phenomenon due to their high resting vagal tone and the immaturity of their sympathetic compensatory mechanisms.[18]

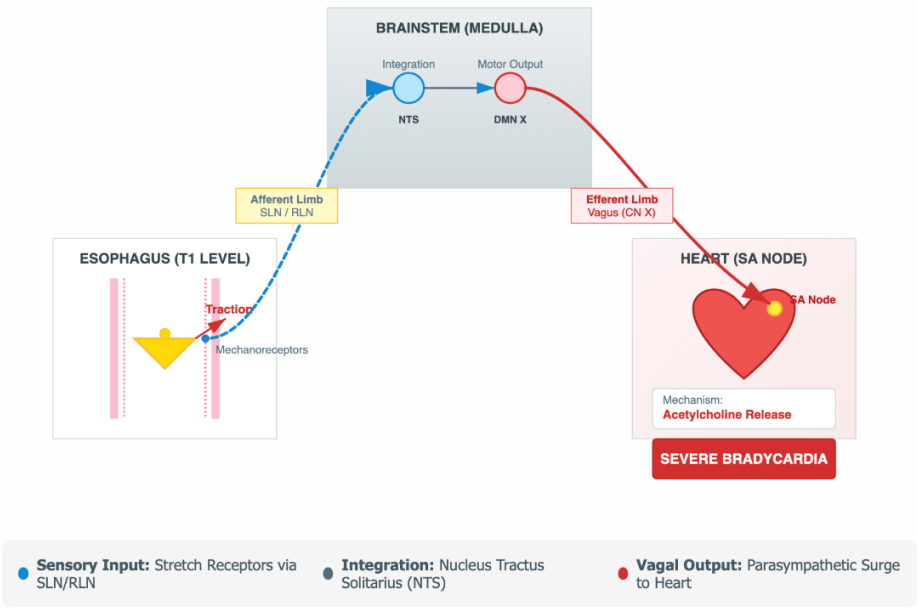

Figure 3. Pathophysiology of esophago-cardiac reflex.

The successful resuscitation and subsequent uncomplicated recovery of this patient illustrate a triad of safety protocol essential for managing high-risk pediatric endoscopy. The first pillar of management is the immediate identification of the event as a reflex rather than a primary respiratory failure. The surgeon's reaction—immediately releasing traction and withdrawing the esophagoscope—was the critical first step. Withdrawing the instrument removes the mechanical trigger for the afferent limb of the reflex arc, often allowing for spontaneous recovery of the heart rate. This case underscores that light anesthesia is a major modifiable risk factor. Reflex arcs are most active when the patient is in a light plane of anesthesia, where autonomic responses are preserved but conscious control is lost. The initial bradycardia suggests the patient may have been in a transitional plane during the first extraction attempt. The administration of a Propofol bolus to deepen the anesthetic plane was crucial before the second attempt. By suppressing central autonomic integration in the brainstem, deep anesthesia effectively blunts the reflex, allowing the surgeon to manipulate the esophagus without triggering a hemodynamic crisis. The use of anticholinergics remains a cornerstone of safety. While the routine prophylactic use of Atropine is debated in modern pediatric anesthesia, its availability as an immediate rescue medication is non-negotiable for procedures involving cervical manipulation. In this case, the administration of intravenous Atropine (0.02 mg/kg) directly antagonized the muscarinic receptors at the SA node, reversing the bradycardia and restoring hemodynamic stability.[19,20]

This report is limited by its design as a single retrospective case study. While the causal link between the surgical stimulus and the cardiac event is strong, we lacked continuous intra-esophageal pressure monitoring to quantify the exact force required to trigger the reflex. Additionally, the rarity of this condition in infants under 4 months makes it difficult to generalize findings to the broader pediatric population. Future research should focus on prospective, multi-center registries to determine the true incidence of vagal events in infant endoscopy. Furthermore, studies investigating the efficacy of prophylactic (pre-incision) anticholinergics versus *rescue* anticholinergics in this specific age group (0-6 months) would provide valuable evidence-based guidelines for anesthesiologists.

4. Conclusion

Rigid esophagoscopy in infants is a high-stakes procedure that demands more than just surgical dexterity; it requires a profound, interdisciplinary understanding of aerodigestive physiology. This case confirms that esophageal foreign body removal in a 4-month-old infant carries a distinct and immediate risk of severe vagal excitation (esophago-cardiac reflex). The mechanical stimulation of the laryngeal and esophageal mucosa—zones of dense vagal innervation—can trigger precipitous bradycardia and desaturation that mimics hypoxic arrest but requires a distinct management strategy. Safety in these cases cannot be assumed; it must be engineered through a triad of care: (1) Vigilance: Immediate recognition of the reflex and cessation of the mechanical stimulus; (2) Suppression: Maintenance of a deep plane of anesthesia (using agents like Propofol) to suppress autonomic reflexes prior to manipulation; (3) Rescue: The immediate availability and swift administration of vagolytic agents (Atropine). The successful outcome in this patient was not a matter of luck, but the result of the seamless, anticipated collaboration between the otolaryngologist and the anesthesiologist. This case serves as a reminder that in the youngest and most vulnerable patients, the surgeon must treat the esophagus not merely as a tube to be cleared, but as a sensory organ to be respected.

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