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Cardiorespiratory complications directly related to endoscopy: associated or not with sedation?

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With the advent of routine sedation in digestive endoscopy, specifically the use of propofol, multiple reports have focused on the complications that may potentially emerge because of sedation, and whether administration is safer by anesthetists or trained gastroenterologists (1-3). Complications associated with endoscopy itself, whether diagnostic or therapeutic, have also been described. However, the fact that both upper and lower endoscopy per se may be accompanied by complications, both during the procedure or thereafter, is usually overlooked (4).

Two categories of endoscopy complications may be described: those arising during the endoscopic procedure, and those that occur once the procedure has been completed. The former are associated with the body’s response to the various stimuli engendered by the procedure (distension, mesenteric traction, etc.) and patient comorbidities (5), and include cardiorespiratory complications.

Many of the complications that take place after endoscopy are related to arterial thromboembolic events at any level, because of the discontinuation of anticoagulant or anti-platelet agents, or bleeding events because of the latter’s reintroduction (6). We should not disregard the changes in intestinal drug absorption as a result of the laxatives used for colonoscopy (7). Increased intestinal transit speed may result in irregular absorption of antiarrhythmic drugs, thyroid hormones, antihypertensive agents, and oral antidiabetics or contraceptives, among others, which may potentially bring about untoward consequences, including cardiac and pulmonary adverse events. Laxatives themselves may give rise to fluid and electrolyte losses, hence setting off hypovolemia and hypotension, in addition to vasovagal reflex from abdominal pain (8).
In this issue of the *Revista Española de Enfermedades Digestivas*, Vázquez-Rodríguez et al. (5) discuss the cardiorespiratory complications that may develop during endoscopy unrelated to sedation. These events include hypertension, hypotension, sinus tachycardia or bradycardia, premature heart beats, atrial fibrillation, supraventricular or ventricular tachycardia, myocardial infarction, hypoxemia, hypercapnia, and aspiration pneumonia. They are more common in patients with cardiopulmonary conditions such as asthma or chronic obstructive pulmonary disease (COPD), arrhythmias, or ischemic heart disease. The authors conclude that a medical record review and basic physical examination should be performed prior to endoscopy, as well as a brief questioning for potential symptoms, in order to reduce risk.

It is at this point that we are stricken with doubt on how events caused by endoscopy, by sedation, by preparation for colonoscopy, by changes in drug absorption, or simply by fasting can be told apart.

Finally, I would like to make reference to the use of CO₂ during endoscopy, which offers clear benefits, particularly regarding abdominal pain from abdominal distension, which is especially important in advanced endoscopy. Although it may induce a mild increase in blood CO₂ when compared with air insufflation, patients seemingly require less sedation and suffer from less abdominal pain, and overall tolerance improves even in patients with pulmonary disease with no increase in cardiorespiratory complications (9,10).

In summary, with the advent of routine sedation in digestive endoscopy, we must not overlook those complications that may arise during endoscopy procedures as a result of autonomic nervous system stimulation, most particularly in patients with prior cardiopulmonary disease. Whether cardiorespiratory complications result from endoscopy *per se*, patient preparation, or sedation cannot be easily differentiated.

**REFERENCES**


