CORRESPONDENCE

Rigid bronchoscopy-induced bacterial translocation: is it a real threat?

To the Editor:

I read with great interest the report by NAYCI *et al.* [1], regarding rigid bronchoscopy-induced bacterial translocation in rats. However, several of their speculative comments are misleading and merit comment.

First, the animal model used in their study does not completely reproduce all the features of rigid bronchoscopy in humans. Bronchoscopy means examination of the tracheo-bronchial tree through a bronchoscope. In the current study, the inserted angiocath did not have similar physical properties of a rigid bronchoscope. Furthermore, this examination was performed outside the tracheobronchial tree through a lateral neck incision. For this reason, it would be better to entitle the article "intratracheal insertion of an angiocath induces bacterial translocation" rather than "rigid bronchoscopy induces bacterial translocation".

Secondly, the authors found a decrease in pH and arterial oxygen tension (P_{a,O_2}), and an increase in arterial carbon dioxide tension values following insertion of the angiocath. They have concluded that bacterial translocation may be related to the hypoxaemia-induced mucosal damage of the intestines. However, SAARNIVAARA *et al.* [2] have found that instrumentation with a telescope during rigid bronchoscopy ameliorates rather than impairs the ventilation and oxygenation of the patients. During the course of the bronchoscopy, as well as after 1 h in the recovery room, the arterial acid-base status was within normal limits; the mean P_{a,O_2} value during bronchoscopy ranged 23–32 kPa (175–240 mmHg) and was 18 kPa (138 mmHg) in the recovery room when the patients breathed ~35% oxygen in the air. The authors suggested that the increased P_{a,O_2} value might be the result of increased oxygen concentration as well as of an increased inflation pressure at the distal end of the bronchoscope [2]. Furthermore, it has been suggested that the term rigid bronchoscopy should be replaced with "open ventilating bronchoscopy", because the property of splinting open the airway and providing a side-arm for delivery of oxygen or anesthetic gases is an inherent feature of all present rigid bronchoscopes. In contrast, the flexible bronchofibrescope inevitably fills a portion of the airway and diminishes ventilation [3]. These findings suggest that rigid bronchoscopy is a safe procedure regarding hypoxaemia.

Thirdly, what was the average internal tracheal diameter of the rats? This is an important point, since if the diameter of an angiocath is close to the tracheal diameter of a rat this may lead to a much tighter insertion and the latter situation may cause tracheal mucosal damage during the procedure. According to the authors, mucosal damage induced by the bronchoscope can promote bacteraemia and bacterial translocation. Nevertheless, in my opinion, the risk of mucosal damage is minimal for a human trachea during procedure performed by a well-trained and skilled bronchoscopist, because anteroposterior and transverse internal diameters of the adult human trachea are 16×14 mm, and the rigid bronchoscopes commonly used have an external diameter that varies 2–9 mm [4, 5].

In summary, one should be cautious of making a conclusion about a direct link between rigid bronchoscopy and

bacterial translocation, because the results obtained using this animal model may be significantly different than those obtained in clinical studies.

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References

1. Nayci A, Atis S, Talas DU, Ersoz G. Rigid bronchoscopy induces bacterial translocation: an experimental study in rats. *Eur Respir J* 2003; 21: 749–752.
2. Saarnivaara L, Tarkkanen J. The effect of instrumentation with a telescope during bronchoscopy on arterial oxygen tension and acid-base balance. *Acta Anaesthesiol Scand* 1983; 27: 242–244.
3. Helters RA, Sanderson DR. Rigid bronchoscopy: the forgotten art. *Clin Chest Med* 1995; 16: 393–399.
4. Scothorne RJ. The respiratory system. In: Romanes GJ, ed. *Cunningham's textbook of anatomy*. New York, Oxford University Press, 1995; pp. 491–529.
5. Prakash UBS, Diaz-Jimenez JP. The rigid bronchoscope. In: Prakash UBS, ed. *Bronchoscopy*. Philadelphia, Lippincott-Raven, 1997; pp. 53–69.

From the authors:

We would like to thank A. Zamani for his comments concerning our article [1].

It seems that our methodology regarding the bronchoscopic procedure has been misunderstood. We intended to develop a bronchoscopic instrument that was basically a rigid, straight and hollow metallic tube, and followed the main bronchoscopic procedures. The instrument was connected to a three-way stopcock. An open system, one opening was then connected to the oxygen source, while the other accessed the atmosphere. The latter orifice precluded any unforeseen risk of excessive airway pressure and allowed to aspirate tracheobronchial secretion. The bronchoscopic instrument was inserted into the rat airway *via* the transoral route, certainly not outside the tracheobronchial tree. The lateral neck incision was performed to confirm whether the bronchoscopic instrument was in the trachea.

During the planning phase of this project, the upper airway anatomy, the trachea and the main stem bronchi of the rat had been carefully examined to construct our experimental design thoroughly. Meticulous dissection provided an excellent view of the anatomic structures of the airway. The length of the bronchoscope was 70 mm and the external diameter was 3 mm. The length of the bronchoscope was sufficient to reach the lower trachea and main stem bronchi. The bronchoscope was marked every 1 cm to estimate the anatomic localisation of the tip of the bronchoscope. The bronchoscope was inserted into the rat airway *via* the transoral route. The distance from the mouth to the epiglottis was approximately 30–32 mm. The larynx began at the opening bounded

anteriorly by the free border of the epiglottis. The length of the larynx was ~4–5 mm. The cricoid cartilage formed a complete ring at the inferior aspect of the larynx and was fixed to the tracheal rings. The trachea was entered after passing through the cricoid cartilage and ended at the division into the right and left main stem bronchi at the carina. The bronchoscope was introduced through the larynx and directed to the trachea. The length and internal diameter of the trachea was ~25–27 mm and 3–4 mm, respectively. The angle between the longitudinal axis of the trachea and the bifurcation of the main bronchi was approximately 15–17°. The angle of the right main bronchus was only slightly less than that of the left main bronchus. Once the trachea was entered, the tip of the bronchoscope was gently slid along the right and left lateral wall of the trachea. This manoeuvre allowed both the right and left main bronchi to be entered easily. In addition, to enter the right and left main bronchi, the rat's head was turned and flexed slightly to the ipsilateral side and the proximal portion of the bronchoscope moved toward the contralateral corner of the mouth. The main stem bronchi was ~6–7 mm in length and its internal diameter was 2–3 mm. The tip of the bronchoscope was advanced 4–5 mm into the right and left main bronchi. At this point, the distal progression of the instrument was halted. Overall, these attempts provided convincing results on proceeding tracheo-bronchial endoscopy. Thus, we strongly believe that our "simulated bronchoscopy" had the main physical properties of a rigid bronchoscope. The rat airway was not visualised in these experiment, yet, we think that this would not have changed the pathophysiological sequence leading to bacterial translocation following bronchoscopy.

There may some reports indicating that rigid bronchoscopy does not impair ventilation and oxygenation [2]. However, what about clinical studies that do show that rigid bronchoscopy induces alterations in arterial blood gases, including hypoxaemia, respiratory acidosis and hypercarbia [3–5]? GODDEN *et al.* [3] measured serial arterial blood gases in 10 patients during rigid bronchoscopy and showed carbon dioxide retention in nine patients even though ventilation was adequate in all. MATHISEN and GRILLO [4] observed 19 complications of 56 patients after rigid bronchoscope, in whom two of those complications were hypoxia and/or hypercarbia. It may intuitively appear that associated procedures during rigid bronchoscopy will increase resistance to ventilation and result in derangements in oxygenation [6]. Overall, can we insist that rigid bronchoscopy is a 100% safe procedure regarding alterations of arterial blood gas parameters?

As mentioned above, at the beginning of this study, we examined the upper airway anatomy, trachea and main bronchi, and then prepared the instrument to be safely inserted into the trachea. The internal diameter of the trachea was 4–5 mm. The external diameter of the bronchoscope was 3 mm. In the present study, the bronchoscope was gently slid along the trachea. It was crucial not to damage the mucosa of the trachea during bronchoscopy, which was later also confirmed by histopathological examinations. As was stated in our article, bacterial translocation occurred from the damaged intestinal mucosa, not the tracheal mucosa.

In conclusion, the merit of our study is that it addresses the phenomenon of bacterial translocation in the specific settings of bronchoscopy. Bronchoscopy leading to impairment of arterial blood gas parameters may induce intestinal mucosal barrier dysfunction and bacterial translocation. However, further investigations aimed at understanding the clinical consequences of this phenomenon are warranted.

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References

1. Nayci A, Atis S, Talas DU, Ersoz G. Rigid bronchoscopy induces bacterial translocation: an experimental study in rats. *Eur Respir J* 2003; 21: 749–752.
2. Saarnivaara L, Tarkkanen J. The effect of instrumentation with a telescope during bronchoscopy on arterial oxygen tension and acid-base balance. *Acta Anaesthesiol Scand* 1983; 27: 242–244.
3. Godden DJ, Willey RF, Fergusson RJ, Wrigth DJ, Crompton GK, Grant IW. Rigid bronchoscopy under intravenous general anesthesia with oxygen Venturi ventilation. *Thorax* 1982; 37: 532–534.
4. Mathisen DJ, Grillo HC. Endoscopic relief of malignant airway obstruction. *Ann Thorac Surg* 1989; 48: 469–473.
5. Rah KH, Salzberg AM, Boyan CP, Greenfield LJ. Respiratory acidosis with the small Storz-Hopkins bronchoscopes: occurrence and management. *Ann Thorac Surg* 1979; 27: 197–202.
6. Prakash UB, Diaz-Jimenez JP. The rigid bronchoscope. In: Prakash UB, ed. *Bronchoscopy*. Philadelphia, Lippincott-Raven, 1997; pp. 53–69.

Inflammatory response phases and their hypothetical trophic meaning

To the Editor:

A recent review by WOOD *et al.* [1] emphasises the role of oxidative stress, specifically lipid peroxidation, in the pathophysiology of asthma.

We have also found that oxidative stress is involved in the pathophysiology of post-traumatic inflammation [2–4]. In order to integrate the different alterations that are produced after injury by mechanical energy, we consider that a response based on the successive functional predominance of the nervous, immune and endocrine systems would be produced. This hypothesis implies that the final and prevalent functions

of these systems may represent the consecutive phases of the response to stress and all of them could have a trophic meaning.

Considering that these functions are expressed by the endothelium and, thus, by extension, by the vascular wall, the post-traumatic local inflammatory response could be divided into three phases. The first one is a nervous or immediate phase, with vasoconstriction and vasodilation that lead to the ischemia-reperfusion phenomenon. This, in turn, causes cellular oedema (ischemia) and interstitial oedema (reperfusion). The second phase is an immune or intermediate one, which is the cause of diapedesis or cell migration, especially of neutrophils