Antimicrobial activity of local anaesthetics

Dear Sirs,

I read with surprise in the interesting article by J. Pugin and P.M. Suter (Intensive Care Med [1992] 18:6–10) that the author instills 10–15 ml of 2% lidocaine into the tracheobronchial tree before performing bronchoalveolar lavage for quantitative bacterial cultures for the diagnosis of pneumonia. Perhaps the authors are not aware of the fact that lidocaine, like other membrane stabilising agents (e.g. beta-blockers and local anaesthetics like tetracaine, dibucaine and procaine) has potent antimicrobial activity as detected by effects on cell growth rates and viability of *E. coli* and *C. albicans* in vitro. The effect of lidocaine on suspensions of bacterial cells growing in a chemically defined medium has been compared with those of antibacterial agents such as ampicillin, chloramphenicol, puromycin and cationic surface active agents [1]. Fortunately for the results of the quantitative cultures of BAL fluid containing lidocaine, the local anaesthetic permits bacterial cell recovery when the agent is removed. Anyhow, if allowed to mix with 50 ml BAL fluid, 10–15 ml 2% lidocaine would attain a concentration of 4000–6000 µg/ml, enough to induce membrane damage in bacterial organisms and possibly compromize their successive growth in culture. The antimicrobial activity of local anaesthetics should be taken into consideration when provedures directed at the isolation and culture of microorganisms involve their local use.

Yours faithfully

F. de' Clari

Reference

1. Abanzukwe TC, Fazley Bazas BS, Salt WG (1991) Further studies on the antimicrobial acitvity of beta-adrenergic blocking agents and local anaesthetics. Abstract No 726, 17th International Congress of Chemotherapy, Berlin, June 23–28

Dr. F. de' Clari, Ospedale Civico, Via Tesserete 46, CH-6900 Lugano, Switzerland

Reply to Dr. F. de' Clari's letter to the editor

Dear Sirs,

Dr. F. de' Clari raises the controversial issue of using local anesthestics when performing bronchoalveolar lavage (BAL). So far, no in vivo studies have addressed the specific question of the effects of lidocaine or its derivates on BAL fluid bacteriologic yield. Like other investigators [1, 2], we have the feeling that the use of reasonable quantities of 2% lidocaine should not influence the growth of bacteria recovered by BAL. It is very unlikely that lidocaine will reach the concentrations cited by Dr. F. de' Clari in BAL fluid because the local anesthetics are instilled into the trachea and main bronchi, yet BAL sampling is performed in a subsegment in a wedged postion, i.e. distal to the sites of lidocain injection. If some lidocaine is present in our BAL fluids, it probably has been greatly diluted.

We would, however, agree with Dr. G.U. Meduri's guidelines for protected specimen brush (PSB) [3] to avoid the injection of lidocaine through the sampling channel of the bronchoscope. The volume of sample recovered by PSB is so small (0.001 ml) that we can guess that even a small contamination of local anesthetics will lead to a significant concentration of anesthetics and therefore influence bacterial growth.

Finally, this raises the question of the type of anesthesia to be used in inbutaed patients when performing BAL. Discomfort and coughing during bronchoscopy and BAL will result in a decrease in the yield of BAL fluid. The guidelines in our ICU include the use of intravenous sedation and local anesthetics to prevent discomfort and coughing and to keep paralyzing agents only for special indications.

Yours faithfully,

J. Pugin and P.M. Suter

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2. Kahn FW, Jones JM (1987) Diagnosing bacterial respiratory infection by bronchoalveolar lavage. J Infect Dis 155:862–869
3. Meduri GU (1990) Ventilator-associated pneumonia in patients with respiratory failure. A diagnostic approach. Chest 97:1208–1219

Prof. Dr. P.M. Suter, Division of Surgical Intensive Care, Hôpital Cantonal Universitaire, CH-1211 Genève 14, Switzerland

P50 in critical illness

Dear Sir,

We read with great interest the article of Myburgh et al. concerning reduced P50 values in critically ill patients, which in our opinion deserves some comments [1].

P50 was calculated from a single venous blood gas analysis and oxygen saturation measurement assuming physiologic heme to heme interaction resulting in a constant Hill's number [2]. We are not convinced that this assumption is valid in these patients. Changes in heme to heme interaction in fact can occur under physiologic and pathophysiologic conditions altering the oxygen binding kinetics of the hemoglobin molecule [3–5]. Depending on the magnitude of the Hill coefficient a reduced P50 value can be associated with a near normal oxygen unloading in tissues if the oxygen saturation is higher than 50% and vice versa. To our knowledge no investigations exist concerning heme to heme interaction, the magnitude of the carbon dioxide and the fixed acid Bohr coefficient and the magnitude of the temperature effect for certain groups of critically ill patients. We therefore strongly recommend a reinvestigation of erythrocyte function in critically ill patients using a more precise methodology.

In addition controversy still exists whether shifts of the oxygen dissociation curve influence tissue oxygenation and in particular oxygen consumption. Several studies favour both possibilities. The uneventful course of pregnancy and delivery in women with atypical hemoglobin or enzyme deficiency of erythrocyte metabolism, factors which both may be associated with extremely left shifted oxygen dissociation curves, support the view that the impact of the oxygen dissociation curve on oxygen supply to tissue is small compared to other factors.

Yours faithfully,

W. Hasibeder, H. Sparr, M, Haisjackl, G. Luz and R. Germann

References

1. Myburgh GH, Webb RK, Worthley LIG (1991) The P50 is reduced in critically ill patients. Intensive Care Med 17:355–358
2. Severinghaus JW (1979) Simple, accurate equations for human blood O2 dissociation computations. J Appl Physiol 21:1108–1116
3. Braumann KM, Böning D, Trost F (1979) Oxygen dissociation curves in trained and untrained subjects. Eur J Appl Physiol 42:51–60

4. Braumann KM, Kleemann W, Martens U, Maassen N, Maass U, Schmidt W, Böning D (1988) Hemoglobin oxygen affinity in patients suffering from arterial occlusive disease of the legs. Klin Wochenschr 66:397–403
5. Mairbäurl H, Humpeler E (1980) Diminution of the temperature effect on the oxygen affinity of hemoglobin after prolonged hypothermia. Pflügers Arch 383:209–213

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Author's reply

Dear Sir,

Thank your for the letter received from Dr. Hasibeder et al. regarding my paper "The P50 is reduced in critically ill patients". I wish to make the following comments in reply.

This study arose from an analysis of data comparing oxygen saturation measured directly by cooximetry and derived from standard equations. The statistically significant difference between these two measurements was postulated to be due an alteration of P50, the calculation of which is from oxygen tension based on the oxygen affinity of haemoglobin under standard conditions of pH, PCO_2 and temperature and quantifies the non-acute influences. The point is well made that haem to haem interactions under various conditions, particularly in critically ill patients, will alter oxygen binding kinetics of the haemoglobin molecule and may interfere with the calculated measurement of P50. The results of this study, however, do concur with the point made by Hasibeder et al. that erythrocyte function in critically ill patients requires further investigation. Our study postulates that the demonstrated reduction in P50 may be due to either an increase in RBC/ATP ratio or an increase in RBC/pH gradient, both of which reduce RBC 2,3 diphosphoglycerate levels. As suggested by Hasibeder et al., a subsequent study using precise methodology and concurrent measurement of 2,3 DPG and RBC pH levels in a chronically catheterised sheep model is underway.

In reply to the second point, our study contributes to the debate regarding the disposition of the oxygen dissociation curve in critically patients and we agree that haemoglobin affinity for oxygen in critically ill patients is multifactorial and that the impact on tissue oxygen delivery is relatively small.

Yours faithfully,

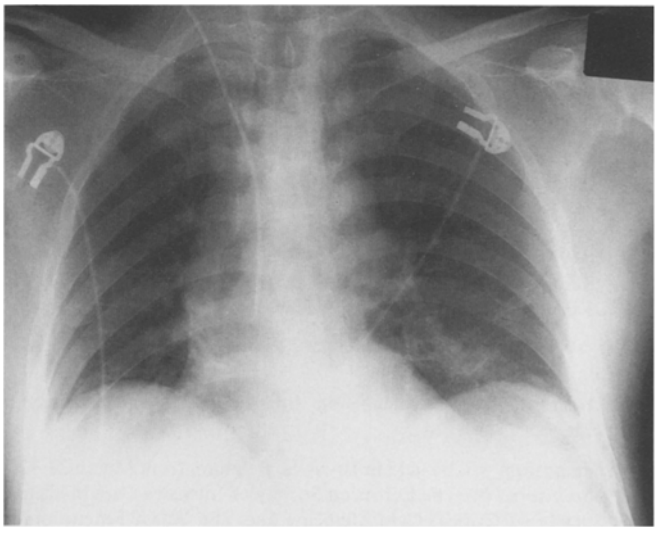
Dr J. A. Myburgh, Staff Specialist, Intensive Care Unit, Royal Adelaide Hospital, Adelaide, South Australia, 5000

Checking central venous catheter placement

Dear Sir,

In checking the site of central venous pressure catheters a lot of methods have been suggested includings: a chest X-ray, blood gases on the sample of blood, back flow of blood, and the fluctuations in pressure with respiratory movements. However, it is not always easy to be definite in cases of low blood pressure with congestive cardiac failure in whom the venous pressure approach the arterial, and the arterial oxygen saturation is low. I had a case of 68-year-old dehydrated man presenting with signs of shock. His blood pressure was 85/55 mmHg. Siting a triluminal catheter in the right internal jugular vein was attempted through the conventional approach but it was a failure. A rather lower approach (between the two heads of sternomastoid muscle) was used and it was a straightforward cannulation. However, the saline column in the mannometer did not drop down, though the saline infusion attached to it was dripping down upon turning the 3-way tap.

An X-ray taken showed the catheter in the right of midline and seemed in the right side of heart, with no significant rotation of the chest or shift in mediastinum. A transducer was used to measure the central venous pressure and showed an arterial waveform and a BP of 90/55 mmHg. The catheter was removed.



This case demonstrates the occasional difficulty in identifying the inadvertant arterial cannulation even when we have an X-ray and I think that a transducer should be used always in such cases.

Yours sincerely

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