Mask CPAP and minitracheotomy, a cautionary tale

Sirs,

Iapichino et al. [1] report three cases of combined use of mask CPAP and minitracheotomy without complications. However, there must be a risk of surgical emphysema if an incision is made into the airway while there is a positive pressure within it. I report a case which shows that this is a potential complication.

Case

A 26 year-old-female with severe pulmonary cystic fibrosis was admitted to intensive care because of respiratory fatigue and failure to clear secretions. She was being supported prior to admission with nasal intermittent positive pressure ventilation (NIPPV). In view of the underlying diagnosis I hoped to avoid committing this patient to conventional endotracheal intubation and elected to insert a minitracheotomy tube (Portex Mini-Trach II) through the cricothyroid membrane to provide access for airway suction and possibly jet ventilation.

The patient continued on NIPPV. Under local anaesthesia with lignocaine and adrenaline I made a horizontal incision in the cricothyroid membrane but was unable to pass the tube as surgical emphysema formed rapidly. NIPPV was discontinued, the patient breathed O_2 at atmospheric pressure, the incision was reopened, and a minitracheotomy tube was successfully passed through the cricothyroid membrane into the trachea. A further attempt at NIPPV was quickly abandoned as it worsened the surgical emphysema. High frequency jet ventilation (Bromsgrove ventilator) at 120 breaths per minute via the minitracheotomy tube also worsened surgical emphysema, so it was necessary to induce emergency anesthesia and intubate the patient with a cuffed oral endotracheal tube in order to gain control.

Comment

Caution should be exercised when using a minitracheotomy in patients with raised airway pressure. Iapichino et al. do not give surgical details of the minitracheotomy procedure; was CPAP continued during minitracheotomy? Did they place the cannula through the cricothyroid membrane or trachea? Did they open the airway with a scalpel incision alone or use a Seldinger-type guide wire and dilate the opening?

While I agree that the technique holds promise I would suggest that

(1) the surgical procedure should only be performed after temporary discontinuation of CPAP or NIPPV
(2) the Seldinger guide wire system may be preferable in that the surgical incision is dilated to the size of the tube and may form a tighter fit
(3) that the patient be observed carefully for the development of emphysema when raised airway pressure techniques are used concurrently.

Yours faithfully,

T. Woodcock

Reference

1. Iapichino G, Gavazzeni U, Mascheroni D, Bordone G, Solca M (1991) Combined use of mask CPAP and minitracheotomy as an alternative to endotracheal intubation. Intensive Care Med 17:57–59

Dr. T. Woodcock, Southampton General Hospital, Intensive Care Unit, Tremona Road, Southampton S09 4XY, UK

Author's reply

Dear Sir,

We agree with Dr. Woodcock that caution is mandatory when combining the use of minitracheotomy and high airway pressure (such as during NIPP). Indeed, neck and facial subcutaneous emphysema has been reported [1] if forceful coughing occurs during or after the placement of a minitracheotomy, even in the absence of artificially raised airway pressure. However, we believe that this complication is unlikely to present when combining minitracheotomy with CPAP (as we suggested in our paper [2]) since no high peak pressure occurs (maximum airway pressure 10–15 cmH_2O). We thank Dr. Woodcock for this opportunity to better clarify the surgical details employed: after local anesthesia we perform a vertical skin incision in excess of 1 cm over the cricoid membrane; we then introduce the cannula using a Seldinger technique aided by a very small scalpel incision of the cricoid membrane. This approach may offer the advantages of a tight fit of the cricoid membrane around the cannula with a larger escape through the skin should an air leakage occur. In conclusion, we believe that the possibility of subcutaneous emphysema, although unlikely, should be kept in mind when combining the use of CPAP and minitracheotomy and we cannot underscore the necessity of close monitoring after every procedure particularly in critical patients.

Yours faithfully,

G. Iapichino

References

1. Pedersen J, Schurizek A, Melsen C, Juhl B (1988) Minitracheotomy in the treatment of postoperative sputum retention and atelectasis. Acta Anaesthesiol Scand 32:426–428
2. Iapichino G, Gavazzeni V, Mascheroni D, Bordone G, Solca M (1991) Combined use of mask CPAP and minitracheotomy as an alternative to endotracheal intubation. Intensive Care Med 17:57–59

G. Iapichino, Istituto di Anestesiologia e Rianimazione, Università degli Studi di Milano, Via F. Sforza 35, I-20122 Milano, Italy

Abrupt hemodynamic improvement in late septic shock with physiological doses of glucocorticoids

Dear Sir,

It is now clear the use of high-dose corticosteroids provides no benefit in the treatment of early septic shock [1]. In the past, adreno-cortical insufficiency might have been masked by the routine administration of corticosteroids during septic shock [1, 2]. We administered physiological doses of glucocorticoids in 7 patients with late septic shock, needing administration of high doses of catecholamines to maintain blood pressure since we suspected a relative adreno-cortical insufficiency. The dosage of glucocorticoids administered; duration for which we employed them; and the patients we selected to treat all differ from those utilized in studies arguing against their value. Patients were studied if they met at least 5 of the 7 classic criteria of septic shock [1]. The source of the sepsis was the lung in 4 patients and the abdomen in 3 patients. Except one, all patients had positive blood cultures and all patients were mechanically ventilated, 5 patients had continuous arterio-venous hemofiltration and dialysis (CAVHD). None of the patients suffered from adreno-cortical insufficiency prior to hospitalization. The time from onset of sepsis to drug administration was at least 6 days. Each patient received fluid loading and catecholamines (dopamine and/or norepinephrine and/or dobutamine) to restore the blood pressure and cardiac output. Two patients received all 3 catecholamines. Each patient was given 100 mg of hydrocortisone intravenously followed by 100 mg every 8 hours with dose tapering with improvement. Cortisol levels were measured in serum taken before the first dose of hydrocortisone. For statistical analysis the ANOVA for repeated measurements was applied and a p-value < 0.05 was considered statistically significant. Six patients survived the septic shock, however 2 died during their stay in the ICU. Serum cortisol levels were within the normal range (250–750 nmol/l). After the administration of hydrocortisone blood pressure increased

($p<0.01$). Heart rate and cardiac output remained essentially unchanged, filling pressures of the heart tended to decrease. Within 24 h dopamine could be tappered ($p<0.01$), norepinephrine could be stopped ($p<0.01$) and dobutamine could be decreased in the 3 patients.

Adjunctive glucocorticoids therapy was given in a seemingly intractable situation suggesting the presence of a relative adreno-cortical insufficiency [3]. Of relevance is that we administered physiological and not pharmacological doses of glucocorticoids late in the course of septic shock. Why was the cardiovascular system unable to maintain a normal vascular tone and what might have been responsible for the clinical improvement? There are probably multiple mechanisms. Endotoxin release can promote inappropriate vasodilatation leading to hypotension. In late septic shock vasodilation is probably the most important pathophysiologic mechanism [4]. In our patients this abnormality may have been ameliorated by glucocorticoids. The unchanged cardiac output in our patients may indicate that glucocorticoids may enhance the responsiveness of the peripheral circulation to pressor amines. Besides, the relative lack of cortisol itself may impair the ability of the arterial wall to maintain an appropriate tone. The problem remains how to define adreno-cortical insufficiency. We assume that in our observation the normal values of cortisol under severe stress may be indicative for relative adreno-cortical insufficiency [5]. Our data would seem to indicate, at a point in time of shock, relative adreno-cortical insufficiency may have developed. This preliminary observation does not support a strong conclusion. However, the results suggest a beneficial effect of the glucocorticoids on the peripheral circulation. We believe that irrespective the mechanisms and the difficulties in the diagnosis of adreno-cortical insufficiency, consideration of the use of physiological doses of glucocorticoids in late septic shock should be given if the clinical situation is seemingly intractable. Controlled studies are needed to define the patient group with septic shock which may benefit from a course of physiologcal doses of glucocorticoids

Yours faithfully

A. J. Schneider and H. J. Voerman

References

1. Bone RC, Fischer CJ, Clemmer TP et al. (1987) A controlled clinical trial of high-dose methylprednisolone in the treatment of severe sepsis and septic shock. N Engl J Med 311:653–658
2. Finlay WE, McKee JI (1982) Serum cortisol levels in severely stressed patients. Lancet I:1414–1415
3. Sibbald WJ, Short A, Cohen MP et al. (1977) Variations in adrenocortical responsiveness during severe bacterial infections. Unrecognized adrenocortical insufficiency in severe bacterial infections. Ann Surg 186:29–33
4. Groeneveld ABJ, Bronsveld W, Thijs LG (1986) Hemodynamic determinants of mortality in human septic shock. Surgery 99:140–152
5. Rothwell PM, Udwadia Z, Lawler PG (1991) Cortisol response to corticotropin and survival in septic shock. Lancet 337:582–583

A. J. Schneider, Surgical Intensive Care Unit, Free University Hospital, P.O. Box 7057, NL-1007 MB Amsterdam, The Netherlands

Haemofiltration/haemodialysis in patients with heparin-associated thrombocytopenia

Dear Sir,

We read with interest the paper by Dr. M. P. Shelly and co-workers entitled "White clot syndrome and continuous arteriovenous haemofiltration" [1]. They reported a patient with acute renal failure following septicaemia. He developed heparin-associated thrombocytopenia (HAT) during continuous arteriovenous haemofiltration with unfractionated heparin as the anticoagulant. Attempts to substitute prostacyclin for heparin as the anticoagulant in haemofiltration failed due to clot formation in the extracorporeal circuit and hypotension experienced by the patient. Unfortunately, the heparin-dependent antibody in the patient cross-reacted in vitro with the low molecular weight heparin (Fragmin, Kabivitrum, Uxbridge, UK). As a last resort, intermittent haemodialysis with unfractionated heparin was carried out. According to the report, the patient did not suffer serious thrombocytopenia, although the patient's platelet counts during intermittent haemodialysis were not reported. It was not mentioned whether he suffered any thrombotic complications (presumably he did not). Severe and extensive thromboses which characteristically affect patients with HAT, often lead to disastrous sequelae such as limb gangrene requiring limb amputation, stroke, acute myocardial infarction and even death.

This patient was indeed fortunate that he did not develop these potentially disastrous complications of HAT when he had intermittent haemmodialysis with heparin. It cannot be assumed that other patients with HAT when rechallenged with heparin, even though the drug is given intermittently, would be equally lucky and would not suffer severe thrombocytopenia and serious thrombosis. In HAT patients who require haemodialysis/haemofiltration, it would be much safer to use alternative anticoagulants. One of these alternative anticoagulants is Lomoparan (Organon, International BV) a low molecular weight glycosaminoglycan mixture of heparan sulphate, dermatan sulphate and chrondroitin sulphate. Unlike the low molecular weight hep*arins*, it is neither fractionated nor derived from commercial heparins. In contrast to the high cross-reactivity rate of low molecular weight heparins with the heparind-dependent antibody in patients with HAT, the cross-reactivity rate of the HAT antibody with Lomoparan is low [2–5]. We have recently used Lomoparan for haemodialysis/haemofiltration in 21 patients with HAT in Australia with encouraging results. Lomoparan provided adequate anticoagulation for the procedure in most of these patients without troublesome blood clot formation in the tubings of the machines. We now check whenever possible for cross-reactivity of the HAT antibody with Lomoparan in vitro before the drug is given to the patient.

Yours faithfully,

B. H. Chong and T. Jacques

References

1. Shelly MP, Majer RV, Perkins C, Pierce T, Nielsen MS (1990) White clot syndrome and continuous arteriovenous haemofiltration. Intensive Care Med 16:334–335
2. Chong BH, Ismail F, Cade J, Gallus AS, Gordon S, Chesterman CN (1989) Heparin-induced thrombocytopenia: studies with a new low molecular weight heparinoid, Org 10172. Blood 73:1592–1596
3. Makhoul RG et al. (1986) Heparin associated thrombocytopenia and thrombosis: a serious clinical problem and potential solution. J Vasc Surg 4:522–528
4. Borg JY et al. (1986) Severe heparin and pertosan polysulphate-induced thrombocytopenia: Clinical aspects, in vitro effects of pentosan polysulphate, low molecular weight (LMW) heparins and oligosaccharides. Thromb Res [Suppl VI] 4:96
5. Messmore HL, Fareed J, Corey J, Griffin B, Miller A, Zuckerman L, Parrez Z, Seghatchian J (1984) In vitro assessment of low-molecular weight heparin in patients with thrombocytopenia induced by standard heparin. Blood 64:5 [Suppl 1]

B. H. Chong, Haematology Department, Prince of Wales Hospital, Randwick, NSW 2031, Australia