Detection of Ryles Tube Position by Capnograph

To,
The Editor, IJA.

Dear Sir,

Adding to the communication “Inadvertent entry of Ryle’s tube in Trachea” by Drs. C. Jayachandraiah and R.K.Jain, published in Indian Journal of Anaesthesia 41:214(1997), we further describe a method of detection of inadvertent Ryle’s tube placement in the trachea. A 65 year old obese female was undergoing laparoscopic cholecystectomy. A Ryle’s tube was inserted post intubation, which went in smoothly. On superficial examination in the oral cavity, no coiling of tube was seen. However, when stomach was not deflated on applying suction, tube position was checked by attaching the sampling line of the endtidal CO2 monitor to the Ryle’s tube. A capnogram trace similar to that seen in a normal intubated ventilated patient was seen, which confirmed the placement of the Ryle’s tube in the trachea which was taken out and repositioned correctly.

Insertion of Ryle’s tube in an intubated patient is a blind and tricky procedure. Very frequently, the tube gets coiled in the oral cavity and has to be inserted with the aid of Magills forceps. Placement in the trachea is also a probability, specially with the use of disposable PVC endotracheal tubes with low pressure cuff as was used in our case. We thus describe a fullproof method of detecting inadvertent tracheal placement of Ryle’s tube in the intubated patient.

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Editor Response : When attempting for insertion of Ryle’s tube in the intubated patient the trachea along with the endotracheal tube can be brought forward away from the oesophagus anteriorly by chin lift and jaw thrust. In addition, direct laryngoscopy and guiding the Ryle’s tube with the help of Magill’s forceps is certainly helpful.

Delayed recovery in Congenital Rubella Syndrome

To,
The Editor, IJA.

This is in response to letter to editor by Dr. Meemu Chadha, Consultant Anaesthetist, Choitram Hospital, Indore.


ii) Ind.J.Anaesth.2002;46(1) : 64

Sir,

I thank Dr. Chadha for her comments and fully agree with her regarding the golden rule. I want to point to a few more facts. Since this case happened, we are serially observing several children who are presented for congenital cataract surgery. It is said that intrauterine infection like rubella, toxoplasma, cytomegalovirus, chlymidia or herpes contribute for development of congenital cataract.

Recently, two children with congenital cataract posted for surgery without any other congenital abnormality had reacted indifferently to general anaesthesia. One child took more than 2 hours to recover from effects of relaxant Atracurium without any apparent cause. In the second child, who also had a delayed recovery, we strongly suspected that N2O in the fresh gas flow may have caused hypoxia and bradycardia. This doubt was confirmed after the surgery was over, when we added Nitrous Oxide intentionally to the fresh gas flow, SpO2 continued to fall significantly leading to hypoxia and bradycardia inspite of good ventilation. Of course, this is not the standard ethical method of confirming the cause of hypoxia ethical and to subjecting the patient for such a experiment.

I therefore like to bring it to the notice of all concerned and invite their valuable opinions.

Thanking you,

Dr. Prasanna Kumar Mishra  
SCB Medical College,  
Cuttack.
‘Complete Flaccid Paralysis following SA’

To,
The Editor
Indian Journal of Anaesthesia
Sir,

This has reference to the article “Complete flaccid paralysis following spinal anaesthesia – a case report” by Dr. Raje S.A. et al in IJA 46(1) 2002: 58-60.

I have read with dismay the above article and am perturbed to note that spinal anaesthesia has been blamed for the flaccid paralysis; although in fairness to matters, the authors have mentioned that, they could not find the exact causative mechanism of the paralysis. There are, however, some flaws in the article, viz.

i) Upgoing toe sign cannot manifest in a patient with flaccid paralysis below T10 level

ii) Being a case of acute appendicitis, the patient should have had leukocytosis, fever and vomiting. Yet there was no mention of such finding in the article.

iii) Whether the bupivacaine solution contained 8% dextrose (to make it hyperbaric) and if so whether the ampoule was autoclaved or not, is unclear.

From the case report that is furnished, I am inclined to conclude that the patient developed acute necrotizing myelitis (rather than viral meningitis). Necrotising myelitis could be from:-

1. Infection (anastomotic veins of Batson) – and in such a type, the myelitis will be of an ascending type.
2. Autoimmune reaction from a small cell type carcinoma of lung or
3. From an intraneural (cord) injection.

Thus, in the interest of medical science in general and for the practice of neuraxial block in particular, a follow up and a repeat MRI scan of spine around T9-T10 level is mandatory on the part of the authors of the article.

Dr. T.A.Koshy
Emeritus Professor of Anaesthesiology
Mangalore.

Response from author:
To,
The Editor, IJA


Respected Sir,

I am grateful to Dr. Koshy, who has read the article in detail. We would like to highlight that this article is not meant to blame the spinal anaesthesia or the anaesthesiologist, but to bring out to the notice of medical faculty that this complication though rare, can happen if adequate precautions are not taken.

Our patient presented with classical features of acute appendicitis and investigations such as ultrasonography and plain abdominal radiograph ruled out any differential diagnosis of right iliac fossa pain. At our institute, we had no facility of estimation of total leukocyte count on emergency basis and with clinical diagnosis of acute appendicitis, emergency appendicectomy was done, which was subsequently confirmed on the OT table and on histopathological examination also. The bupivacaine solution used contained 8% dextrose, which is available in presterilised ampoule as available on our hospital schedule and it was not autoclaved as this practice is obsolete. Our patient developed flaccid paralysis with extensor plantar reflexes. This clinical sign points to upper motor neuron paralysis and all investigations suggested were done which could not lead to a confirmatory diagnosis. We agree that a follow up MRI scan would have definitely benefited the academic point; however patient was lost to follow up after such a heart breaking complication at an young age.

Dr. Sameer Rege
Mumbai

Comments of the Neurophysicists:

To,
The Editor
Indian Journal of Anaesthesia
Sir,

The recent article ‘Complete Flaccid Paralysis Following Spinal Anesthesia’ and Prof. Koshy’s response to the same raise few points for clarification.a) The authors have mentioned, flaccid paraplegia, clinical level being at T10 with sphincter involvement and extensor plantar response. Whether the sensory loss was for all modalities or only for spinothalamic sensory loss, which would have been useful in identifying the pathology.

b) The proposed etiology was ‘aseptic meningitis’ for which there is no clinical and/or laboratory evidence of meningeal inflammation in the case report.'
c) It is not mentioned whether the MRI study was with or without contrast, as the contrast enhanced MRI may have higher sensitivity in identifying myelitis.

d) Available clinical data support the diagnosis of acute thoracic myelopathy and the evidence for inflammation/ischaemia is lacking from the available investigations.

e) Extensor plantar response is a time honoured sign of upper motor neuron lesion above lower lumbar segment\(^2\)\(^3\). With the clinical level being at T\(_{10}\) upgoing plantar response is a expected finding. The simultaneous presence of hypotonia and extensor plantar response is probably due to partial recovery from spinal shock.

Reference:


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‘Complete Flaccid Paralysis following SA’

To,
The Editor, IJA.
Ref: Indian J. Anaesth. 2002;46 (1) : 58

Sir,

The title of the above referred article is no doubt fascinating but is shocking too; such a title has been given by the authors without any concrete evidence to substantiate the title

1) The authors themselves have come out with the diagnosis of viral/aseptic and this too without any signs and symptoms of these, leave aside the investigative evidence. All investigations including M.R.I were on but electromyography which could have exonerated poor S.A. was not done.

2) We all know that over 60% of operations are done under Centrel Neuraxial blockade, and that such articles, at this juncture are bound to send wrong signals not only to our budding anaesthesiologists but to public at large.

3) We know, some such vague charges against S.A in early 50’s marred its popularity for nearly three decades

4) More over, in the wake of consumer protection act of recent years I am afraid such article will certainly attract untoward, uncalled for complications. Every one of us must exercise caution in such issues

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