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Is difficult or failed intubation a confounder or an effect modifier for hypoxaemia? Comment on *Br J Anaesth*; 125: e81-7

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Keywords: Caesarean delivery; desaturation; hypoxaemia; obstetric anaesthesia; rapid sequence induction; tracheal intubation

Editor—Bonnet and colleagues[1] reported the results of an observational study that showed that parturients who encountered difficult or failed intubation were at increased risk for hypoxaemia after intubation (adjusted odds ratio =19.1 [8.6–42.7]). Baseline predictors for difficult intubation were collected: it is possible that these identified risk factors[2] attributed to difficult or failed intubation prolonged the intubation time leading to hypoxaemia. It is important to know whether difficult or failed intubation is a potential confounder or an effect modifier (in other words, the interaction effect). A variable is considered a confounding variable if it is associated with both the exposure and outcome variables, but is not associated with the causal pathway between the exposure and the outcome (Fig. 1a). As shown in Figure 1b, it is likely that the occurrence of difficult or failed intubation might be the mediator between predictor variables of difficult intubation and hypoxaemia. If so, then, it would be appropriate to test the interaction effect of the covariate (i.e. the presence or absence of difficult/failed intubation in the regression model).

The authors mentioned that 9.6% of the parturients needing non-elective Caesarean section had severe pregnancy-induced hypertension. It is understood that the authors used rapid sequence induction of anaesthesia and tracheal intubation to secure the airway. However, in this group of patients, the transient, but severe hypertension that accompanies tracheal intubation can result in fatal neurological complications. As a result, guidelines recommend administration of agents such as short-acting beta blockers, opioids, or vasodilators to blunt the intubation stress response. The authors need to justify not using the above medications. Further, if the authors had used these agents, it would have been interesting to see the effect on hypoxaemia, especially with the short-acting opioids.

The authors cited an article suggesting that head-up positioning does not prolong the safe apnoea time in the obstetric population.[3] This unexpected result may be attributed to several factors: small sample size (10 parturients in supine *vs* 10 parturients in head-up position), lack of a reliable tool to assess lung denitrogenation (i.e. monitoring of end-tidal concentration of expired oxygen), and no information on parturient BMI. On the contrary, a study by Hignett and colleagues[4] on healthy term parturients showed a significant increase in functional residual capacity with the 30° head-up position in comparison with the supine position. In addition, the head-up position improves the glottic view at laryngoscopy.[5] In one survey, the majority of respondents preferred head-up or ramped positioning before induction of anaesthesia in obstetric patients.[6] It is necessary to conduct a multicentre randomised clinical trial to determine whether head-up position in comparison with supine position prolongs the time to desaturation during the apnoea phase in rapid sequence induction for Caesarean section.

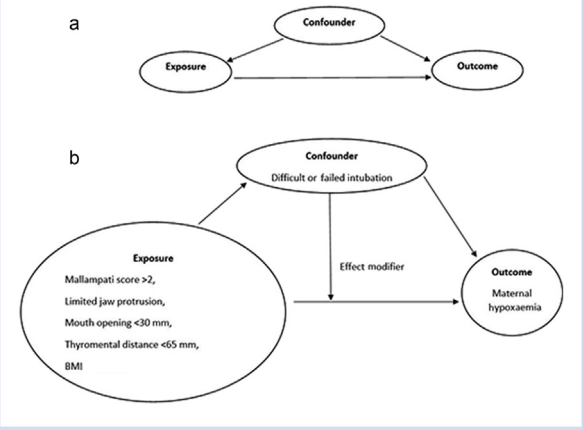

Fig 1. Confounder and mediator relations in exposure–outcome associations.

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Declarations of interest

The authors declare that they have no conflicts of interest.

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Not another requiem for succinylcholine. Comment on *Br J Anaesth* 2020; 125: 423–5

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Keywords: monitoring; neuromuscular blocking agents; postoperative pulmonary complications; succinylcholine; tracheal intubation

Editor—We read the recent editorial in *British Journal of Anaesthesia*[1] entitled 'Another nail in the coffin of succinylcholine' with considerable interest. The authors' analysis of the observations of Schäfer and colleagues[2] on the association between succinylcholine administration and postoperative pulmonary complications (POPC)[2] was scholarly, as expected from these well-regarded experts. But despite their cogent arguments, we are not yet ready to accept their conclusion that there is no role for succinylcholine in modern anaesthesia practice.

We concur that succinylcholine is not the ideal neuromuscular blocker, as the list of its potential side-effects is lengthy. However, we cannot agree that its pharmacodynamic profile is less than unique. After a 1.0 mg kg^{-1} dose (~3–4 times its effective dose for 95% depression of baseline twitch, ED_{95}), complete twitch suppression at the adductor pollicis muscle usually occurs in slightly more than 60 s. Of greater importance is that spontaneous recovery to 90% of control twitch height typically requires 10 min.[3] A reduced initial dose of 0.6 mg kg^{-1} will speed recovery by 1.5–2 min while still achieving 100% block in <2 min.[3] This lower dose (0.5–0.6 mg kg^{-1}) is as effective in producing good intubating conditions as a 1.0 mg kg^{-1} dose.[4] A small defasciculating dose (10% of ED_{95}) of a non-depolarising neuromuscular blocking agent preceding administration of succinylcholine 1.0 mg kg^{-1} will also reduce offset times.[5] Thus, although no antagonist to the neuromuscular blocking effects of succinylcholine is readily available,[6] one is rarely indicated.

Before the introduction of short and medium duration neuromuscular blocking agents, succinylcholine was widely used to facilitate tracheal intubation, followed by maintenance of neuromuscular block with non-depolarising drugs. This combination is rarely used clinically today; if it is used, we agree the practice may be suboptimal. However, some uniquely evanescent effects of succinylcholine still make it the drug of choice in several clinical situations: (1) when an episode of laryngospasm develops under sedation or mask anaesthesia, a small dose of succinylcholine ($\leq$0.4 mg kg^{-1}) can quickly abort laryngospasm leading to full recovery in <10 min[3]; (2) a patient undergoing a 30–40 min ambulatory procedure (e.g. sinus surgery) who needs tracheal intubation to protect the airway, but does not require further muscle relaxation; and (3) electroconvulsive therapy, when succinylcholine can mitigate tonic–clonic motor activity and potential injuries.

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