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## Reliability of the ASA physical status scale in clinical practice: methodological issues

Editor—I was interested to read the paper by Sankar and colleagues[1] published in the September 2014 edition of the *British Journal of Anaesthesia*. The purpose of the authors was to evaluate ASA physical status (ASA-PS) inter-rater reliability and validity in clinical practice. They reported that the ASA-PS score had moderate inter-rater reliability ($\kappa$ 0.61), with 67.0% of patients being assigned to the same ASA-PS class in the clinic.[1]

It is crucial to know that there is no value of $\kappa$ that can be regarded universally as indication of good agreement. Two important weaknesses of *k* value to assess agreement of a qualitative variable are as follow: it depends upon the prevalence in each category which means it can be possible to have different $\kappa$ value having the same percentage for both concordant and discordant cells. Table 1 shows that in both (a) and (b) situations, the prevalence of concordant cells are 80% and discordant cells are 20%; however, we get different $\kappa$ value (0.38 and 0.60), respectively. $\kappa$ value also depends upon the number of categories which means the higher the categories, the lower the amount of $\kappa$ value.[2 3]

Based on their results, ASA-PS score had moderate ability to predict in-hospital mortality (receiver-operating characteristic curve area 0.69). For prediction studies, we need two different cohort data sets or at least split one cohort data set to develop our prediction model and then validate it.[2 3]

As a take-home message, for reliability and validity analysis, appropriate tests should be applied by researchers. Otherwise, misdiagnosis and mismanagement of the patients cannot be avoided.

**Table 1** Comparison of two observers' diagnosis with different prevalence in the two categories

| (a) | Observer 1 | | |
|---|---|---|---|
| | **Positive** | **Negative** | **Total** |
| Observer 2 | | | |
| Positive | 70 | 10 | 80 |
| Negative | 10 | 10 | 20 |
| Total | 80 | 20 | 100 |
| $\kappa$ =0.38 | | | |
| **(b)** | **Observer 1** | | |
| | **Positive** | **Negative** | **Total** |
| Observer 2 | | | |
| Positive | 40 | 10 | 50 |
| Negative | 10 | 40 | 50 |
| Total | 50 | 50 | 100 |
| $\kappa$ =0.60 | | | |

## Declaration of interest

None declared.

S. Sabour
*Tehran, I.R. Iran*
*E-mail: s.sabour@sbmu.ac.ir*

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## Cardiac output decrease and propofol: what is the mechanism?

Editor—I read with interest the correspondence regarding the haemodynamics of induction agents and bispectral (BIS, Covidien, USA) index-guided induction of general anaesthesia.[1 2] As far as I am aware, the paper by Moller Petrun and Kamenik[3] is the first to look at this period of change in haemodynamics using BIS-guided anaesthesia and a minimally invasive cardiac output monitoring device (LiDCOrapid, LiDCO Ltd, UK).

It is interesting to note that the paper referred to by Kakazu and Lippmann relates only to an abstract published nearly 30 yr ago.[4] However, they are quite correct in their proposition that the decrease in cardiac output (CO) may be quite substantial in high-risk patients after induction of general anaesthesia. Indeed, after the abstract published in 1986, Lippmann and colleagues[5–9] have been very keen to highlight the deleterious cardiac effects of propofol in a number of articles (mainly correspondence) to this and other journals.

Most anaesthetists continue to regard the decrease in mean arterial pressure (MAP) on induction as due either to cardiac depression, as Kakazu and Lippmann propose, or due to decrease in systemic vascular resistance. However, we believe that the decrease in CO has little to do with an effect of reduction in cardiac contractility as they suggest, but rather on an effect on preferentially reducing venous rather than arterial tone.

Elegant studies in the early 1960s demonstrated that venous tone was significantly raised in precisely those patients who would be most affected by venodilation and included patients with cardiac failure and the anaemic.[10] Although alluded to in some publications,[11] little attention has been directed to the known effects of propofol on venous smooth muscle. This pioneering work was carried out nearly 30 yr ago by Colin Goodchild and his colleagues at Leeds.[12 13] They clearly demonstrated that veno relaxation and an increase in venous capacitance resulted in diminished venous return and stroke volume leading to a decrease in CO and MAP. Venous relaxation due to propofol can very easily be offset by the simple expedient of administering a low-dose phenylephrine infusion immediately before the administration of propofol. We have shown, albeit in abstract form only, that this simple manoeuvre will markedly reduce the decrease in CO and MAP during induction with propofol.[14] Indeed, it may well be that most of the effects on reduction in stroke volume, CO, and MAP and increase in stroke volume variation seen post-induction may be due to venous relaxation. Although the effects of venous capacitance can also be counteracted by the use of fluids,[13] it is surely not the correct strategy in elective patients who are not fluid depleted and have not been subjected to many hours of fluid restriction.[15]

Moller Petrun and Kamenik are to be congratulated for pointing out the decrease in CO drives the decrease in MAP post-induction and is evident with both propofol and etomidate. Further work is being done in this area to see whether our supposition that phenylephrine will markedly reduce this decrease by its administration prophylactically during surgery in high-risk patients is correct. It also emphasizes the importance of measuring CO preinduction and getting a baseline reading. As Professor Jean Louis Vincent and Dr David Fagnoul have elegantly pointed out 'The main reason why reliable cardiac output monitoring can be useful during surgery is to be able to establish a baseline for high-risk patients in whom complications, such as hypoxemia, tachycardia or oliguria, arise after the immediate postoperative period, and therapeutic interventions become more complex. Some would argue that there is still time to introduce a cardiac output device at this point, but most would agree that it is preferable to be able to make a trend evaluation when such a problem occurs'.[16]

## Declaration of interest

D.W.G. has received subsistence and travelling expenses from LiDCO Ltd and also honoraria for speaking at meetings from Covidien.

David W. Green
*London, UK*
*E-mail: davidgreen1@nhs.net*

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