Reviewer's report

Title: Sensitivity of intra-abdominal, intra-organ and intravascular pressures in detecting changes induced by external abdominal pressure with and without PEEP: an experimental study

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Reviewer: Manu Malbrain

Reviewer's report:

I read with interest the paper by Jakob et al. Entitled “sensitivity of intra-abdominal, intra-organ and intravascular pressures in detecting changes induced by external abdominal pressure with and without PEEP”

Any further characterisation on IAP, IAH and ACS is worthwhile and I congratulate the authors for having performed this study and bringing us more light on this topic. The article is short and to the point, well written in correct English. I only have some minor comments.

1) Introduction: please stick to the recently published definitions on IAH and ACS by the WSACS: IAH is defined as a SUSTAINED increase in iAP above 12 mmHg. While ACS is defined as a sustained increase in IAP above 20 mmHg (not 25 as stated by the authors) with NEW ONSET organ failure [1, 2].

2) Introduction page 5: some recent papers alluded to the interactions between intrathoracic and infrathoracic pressures. I don’t agree with the last paragraph that these pressure changes are not well defined [3-5].

3) Methods: what about the impact of ventilation and thus the respiratory changes on IAP and other pressures. Recent data show that this may be related to the compliance of the abdominal wall. Do you have data on end-expiratory, end-inspiratory and mean values of these pressures; if not this can be discussed as limitation and option for future studies.[6]

3) Methods: major drawback is that ITP or Peso were not measured – this needs to be addressed (cfr above)

3) Methods: another drawback is that the application of external pressure on the abdomen is far from a real life situation where the abdomen is either fluid filled (ascites) or gas filled (laparoscopy) or filled with edematous organs. Making it difficult to extrapolate these results to other studies or a real life situation. A recent publication also alluded to the optimal animal model and implications and guidelines for research on IAH and ACS [7, 8].

Methods: some studies alluded to the fact that morphinomimetics (like fentanyl) can increase IAP by abdominal muscle contraction. Also the level of sedation is important, the use or not of muscle relaxants and the positioning of the subject or
animal. This could be discussed a little bit more afterwards [9-11].

Methods: pigs were starved with free access to water, later on ringer solution was given to keep PCWP between 6-12mmHg. This may have introduced a bias since static barometric thresholds may not reflect true preload status in conditions of increased ITP or IAP, volumetric monitoring would have given some added value. What about SVV/PPV?

Methods: regarding zero reference, just a notice that ideally IAP should be zero-referenced at the mid-abdomen level. It is important to note that the level of the zero reference used clinically in IAP monitoring may have an impact [12].

Methods: instillation of 50ml into the pigs bladder probably is too much, in humans a maximal instillation volume of 20mls was recommended – this need to be discussed.

Methods: just a note regarding the application of external weight, this technique and its effects on IAP, ICP, … has been studied before and moreover it was in humans by Citerio, this reference needs to be added [13]

Methods: regarding peep: other studies showed an effect of PEEP on IAP, but this was related to the magnitude of PEEP (little effect at PEEP levels below 11) and the baseline IAP. Maybe the results could have been different if PEEP was increased to a level equal to IAP.

Page 9-10 are difficult to read. I understand the different stages of the study or given but it would make more sense to presnet the data otherwise: effects on IAP (stomach vs bladder vs direct); effects on vascular pressures (supradiafragmatic: CVP, PCWP, PAP – vs infradiafragmatic: IVCP)

Discussion: state definition of APP= MAP – IAP

Page 12 do you mean pulmonary artery oclusion pressure instead of diastolic? I don’t agree with the statement that your data do not support the use of higher filling pressure targets in IAH: please omit statement because in general static pressure targets are useless and volumetric indices or SVV/PPV were not measured

Conclusions: no data given to support statement that bladder, stomach and direct IAP correlate well: please add results on correlations and bland and altman analysis

Tables: since a swan ganz was in place can you provide data on SVR, CO, ...

Table 2: please also provide data on CVP, PAOP

Figures are difficult to read: I would replace by correlation plots and bland and altman plots comparing direct IAP as gold standard with IGP, IVP, IVCP and then look at correlations between IVP as gold standard vs IGP and IVCP and finally IGP vs IVCP. Correlations should be looked for for ALL comparisons, for PEEP 0 vs PEEP 10 and fro nl IAP vs IAP >12
Another interesting figure could be to look at delta IAP vs delta IGP/IVP/IVCP

References: a more recent review on “what is normal IAP” has been published [14], increased mortality in relation to IAH has also been shown in a recent and the only one so far multicentre study [15]

Regarding abbreviations: please expand all abbreviations when used for the First time and use them in a concise way afterwards (eg IAP, IAH, ACS,...)

IN CONCLUSIONS: again I want to congratulate the authors for this nice study that I enjoyed reading. If they take into account the comments above they can further improve their paper.

Suggested references


Level of interest: An article of importance in its field

Quality of written English: Acceptable

Statistical review: Yes, and I have assessed the statistics in my report.

Declaration of competing interests:

I declare that I have no competing interests, except as former founding president of the world society on abdominal compartment syndrome I just have a high intrest in this topic...