

3. Rosner M, Daughton S (1990) Cerebral perfusion pressure management in head injury. *J Trauma* 30:933–941
4. Chan KH, Miller JD, Dearden NM, Andrews PJD, Midgley S (1992) The effect of changes in cerebral perfusion pressure upon middle cerebral artery blood flow velocity and jugular bulb venous oxygen saturation after severe brain injury. *J Neurosurg* 77:55–61
5. Dearden NM (1992) Brain edema and raised intracranial pressure after head injury. In: Vincent JL (ed) *Update in intensive care and emergency medicine*. Springer, Berlin Heidelberg New York Tokyo, pp 537–552
6. Chesnut MR, Marshall LF, Klauber MR, Blunt BA, Baedwik N, Eisenberg A, Jane JA, Marmarou A, Foulkes MA (1993) The role of secondary brain injury in determining outcome from severe head injury. *J Trauma* 34:216–222

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Does PEEP-ventilation cause a humorally mediated cardiac output depression in pigs?

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Sir: In 1978 Patten et al. [1] applied PEEP to one of two dogs connected by cross circulation. In this dog cardiac output decreased 36%. In the other dog, which did not receive PEEP, cardiac output decreased 18%. It was hypothesized that the CO decrease by PEEP was partly due to a release of a humoral factor. Recently Berglund et al. [ICM (1994) 20:360–364] performed similar experiments in pigs and did not observe a decrease in cardiac output in the recipient pig during application of PEEP to the donor pig.

Two mechanisms could be valid to explain the different results between the two studies. Either pigs do not produce a humoral factor which decreases cardiac output additionally to the mechanical effect of PEEP on venous return, or pigs produce a relatively stronger counteracting control mechanism during application of PEEP than dogs. In this latter case the catecholamines should balance a negatively acting hypothetical humoral factor in the recipient pig.

If one of my hypothetical explanations should be true, we would expect to find about half the negative effect of PEEP on cardiac output in pigs compared to that in dogs. However, as far as I know, we have no evidence of such striking differences in responses to PEEP between dogs and pigs. Schreuder et al. [2] reported a decrease in cardiac output in pigs by PEEP₁₅ (= 15 cmH₂O) to about 60% of the control value at ZEEP (PEEP₀). Cassidy et al. [3] observed in dogs at PEEP₁₅ a decrease in cardiac output to 58% of the value at ZEEP and Scharf and Ingram [4] to about 50%.

The reasons of their different results from those of Patten et al. were not given by Berglund et al. So far, I have the impression that we will end at present in a 'tis-tisnt conclusion.

References

1. Patten MT, Liebman PR, Manny J, Shepro D, Hechtman HB (1978) Humorally mediated alterations in cardiac performance as a consequence of positive end-expiratory pressure. *Surgery* 84:201–205
2. Schreuder JJ, Jansen JRC, Bogaard JM, Versprille A (1982) Hemodynamic effects of positive end-expiratory pressure applied as a ramp. *J Appl Physiol* 53:1239–1247
3. Cassidy SS, Robertson Jr CH, Pierce AK, Johnson Jr RL (1978) Cardiovascular effects of positive end-expiratory pressure in dogs. *J Appl Physiol* 44:743–750
4. Scharf SM, Ingram Jr RH (1977) Effects of decreasing lung compliance with oleic acid on the cardiovascular response to PEEP. *Am J Physiol* 233:635–641

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Authors' reply

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Sir: The sharp-sighted comments on our cross circulation by Prof. Versprille are much appreciated. We believe, however, that the first of his two alternatives to explain the different outcome of our study and that of Patten et al. needs a complementary addition. Not only should the pig lack a humoral factor decreasing cardiac output (CO) but also a detectable counteracting mechanism.

Since we also believe that the pig – as well as the dog and his master – does have a counteracting mechanism, this would incline us to support the second of his two alternatives.

But for two reasons we don't believe the negative effect of PEEP on CO in the pig to necessarily be half of that in the dog.

First, we would like point out the hazards in simply comparing figures on CO depression due to PEEP reported from different laboratories. Variant conditions beside the specific PEEP-level have a great impact. Second, it is not a matter of course that depression due to some negatively acting humoral factor act in strictly additive fashion. It might well be that the effect of the humoral factor "hides", in part or entirely, within the effect of reduced venous return. Actually, the cross circulation model is suited to disclose such a phenomenon.

On the other hand we find it rather unlikely that a negative humoral factor and its counteracting mechanism would exactly outbalance each other – unless both were of diminutive magnitude and far below the order reported by Patten et al.

So far we have the impression that the reason for the difference in results between Patten et al. and us should be attributed the different species of our laboratory animals.

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