# Correspondence

# Pessi's law and curve

### Dear Sir,

I read with great interest the letter on Pessi's law and curve in your journal [1]. I feel that I should point out that circa 430 BC, or 2359 years before Walter Cannon [2], Hippocrates presented a theory about the steady-state of body homeostasis and furthermore wrote a chapter on prognosis in which he states that mortality increases with the severity of the disturbance of vital functions [3].

I would therefore like to suggest that the statement of Pessi "mortality increases with the severity of disturbance of vital function on admission" should, more properly, be called the law of Hippocrates.

Yours sincerely, M. E. Sinclair

### References

- Harju E, Pessi T (1986) Mortality increases with the severity of disturbance of vital function on admission to intensive care. Pessi's law and curve. Intensive Care Med 12:58
- Cannon WB (1929) Organization for physiological homeostasis. Physiol Rev 9:399
- Hippocrates (circa 430 BC) Prognosis: the importance of being able to foretell the course of an illness and an account of the significance of various signs. In: Lloyd G (ed) (1983) Hippocratic writings. Translated by Chadwick J, Mann WN. Penguin, Middlesex, UK, pp 170-185

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# **APACHE II**

#### Dear Sir,

APACHE II, the severity of disease classification system proposed by Knaus and coworkers [1] improves our capacity to predict patient outcome compared with the APACHE prototype version [2] and the abreviated SAPS proposed by Le Gall and coworkers [3].

APACHE II incorporates the feasability of expected mortality rate prediction based on assigned factors corresponding to different diagnostic conditions.

Although APACHE II is as simple as its predecessor since the physiologic measurements have been reduced from 34 to 12, it is still time-consuming, firstly to assign physiologic measurement scores, and subsequently for the calculation of inhospital expected mortality rates.

To reduce to a minimum this time and to obviate errors in assignation and addition of scores or mortality rate, we have designed a microcomputer short programme that will possibly be useful in any ICU.

The programme is written in BASIC for an ATARI-800 XL microcomputer. It occupies 5.7 Kb (10.3 Kb post-initialization) in RAM and it can be adapted easily to any computer by simply chang-

ing the commands for alphanumeric strings treatment, because its handling is different for diverse BASIC dialects.

The programme works in two parts. The first, after the introduction of the values of the 12 parameters displayed by the microcomputer, shows the APACHE II score, APS points, age points and chronic health points, and the total score. Later it solves the exponential equation that determines the estimated risk of inhospital death.

The scores are presented on a monitor screen because it is the most common microcomputer peripheral device, and its treatment is similar in the diverse BASIC dialects, unlike printing devices, that require concrete commands for each different computer.

For those who are interested and would like to receive a copy of the programme please write to us for details.

### Yours sincerely,

J. Gil Cebrian, M. P. Bello Cámara and R. Diaz-Alersi

### References

- Knaus WA, Draper EA, Wagner DP, Zimmerman JE (1985) APACHE II: a severity of disease classification system. Crit Care Med 13:818
- Knaus WA, Zimmerman JE, Wagner DP, Draper EA, Lawrence DA (1981) APACHE – acute physiology and chronic health evaluation: a physiologically based classification system. Crit Care Med 9:591
- 3. Le Gall JR, Loirat P, Alperovitch A (1983) Simplified acute physiological score for intensive care patients. Lancet 2:741

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## Acute respiratory distress syndrome

#### Dear Sir,

The need for an accurate index to predict outcome is emphasized by Smith and Gordon in their article on "An index to predict outcome in adult respiratory distress syndrome." [1]. I feel, however, that their study has two conceptual inaccuracies and a significant methodological difficulty.

Of their 30 patients 16 had bacterial infection or toxaemia, and 9 had multiple injuries, yet neither the severity nor the extent of this pathology is reflected in the score. In multiorgan pathology outcome is not dependent on only one variable. Their assumption that outcome depends only on the respiratory parameter is therefore false.

The second conceptual difficulty entails the use of Paw. Peak upper airway pressure refers to the maximum pressure attained during air movement and is therefore affected by the inspiratory flow rate and airway resistance. A simple bronchial mucous plug or merely increasing the ventilator frequency will play havoc with the Paw reading and therefore with the calculated ventilator score. Conceptually what is required is a measure of the plateau pressure reflecting the patient's static lung compliance. Using compliance calculations obviates the need for a table of control values and allows prediction of PEEP effects (Dr. R. G. Clark's table does indeed show the increasing peak pressure with increasing flow rates. The tidal volumes divided by the mean pressure remain virtually constant, a finding to be expected in normal lungs with normal compliance).

A methodological limitation of this study is that it is retrospective. It is true that the authors state that they have embarked on a prospective study, but it should be remembered that no predictive score can be justified by the retrospective data from which it was calculated. Any attempt to promote this score without adequate prospective validation must be considered premature.

Yours sincerely, L. M. Pott

### Reference

 Smith PEM, Gordon IJ (1986) An index to predict outcome in adult respiratory distress syndrome. Intensive Care Med 12(2):86

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# **Replies from the authors**

Dear Sir,

Thank you for the opportunity to reply to Dr. Pott's comments. Our ventilator index is designed to predict outcome from the pulmonary lesion of ARDS and not to predict outcome from sepsis, trauma, etc. The natural history of patients who have ARDS tends to be one of progressive respiratory failure and death from hypoxaemia which occurs with or without other organ involvement. In addition it was possible to obtain complete separation of the survivors from non-survivors by analysis of the extent of the pulmonary lesion alone which indicates that it is unnecessary to include other organ failures.

The second "conceptual difficulty" that worries Dr. Pott is the use of the Paw. If we had relied on a single Paw measurement each day, then he would be right to say that it could be influenced by various factors which could give a false ventilator score. However, the Paw was noted hourly providing 24 readings per day from which the mean was calculated. Thus transient changes would not significantly influence the overall daily value. Secondly, the use of control values for peak Paw allows for changes resulting from varying the frequency of ventilation to be taken into account. We agree that it may well be possible to derive a ventilator score using compliance but disagree that our use of the peak Paw detracts from the accuracy of the present index. To date the index has been tested prospectively on 16 patients with 100% accuracy.

Finally, Dr. Pott feels that our method of deriving the index is a "methodological limitation" until tested prospectively. It was stated in the last sentence of the paper, "If this (the ventilator score) is *confirmed by prospective use* then the ventilator index will become a valuable aid in exercising triage in the intensive care unit."

#### Yours sincerely,

P. E. M. Smith and I. J. Gordon

Dear Sir,

Thank you for the opportunity to reply to Dr. Hickling's comments [1]. The oxygen gradient and upper airway pressure were chosen because on preliminary review of all the patients it was apparent that these two parameters closely reflected the severity of the lung lesion. This was confirmed by the fact that the survivors and non-survivors could be completely separated using these variables together with age. Although the oxygen gradient is affected by  $F_1O_2$ and PEEP, it is still a useful guide to the severity of the lung lesion provided the methods we described in the paper are adhered to. As far as we are aware these methods are standard practice in many intensive care units at the present time. Where alternative methods of ventilation are used then providing there is consistent management a score may still be calculated but the critical value above which survival is unlikely will differ.

Finally, we cannot dispute that patients with ARDS may die from causes other than respiratory failure but the object of our index is to quantify the pulmonary lesion regardless of other organ disease. Nevertheless, in the group of patients we studied progression of the pulmonary lesion was by far the commonest cause of death.

### Yours sincerely

I.J. Gordon and P.E.M. Smith

#### Reference

1. Hickling KG (1987) Acute respiratory distress syndrome. Intensive Care Med 13:83 (Letter)

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