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CORRELATION BETWEEN THE PHYSICAL SIGNS OF HYPERCAPNIA AND THE MIXED VENOUS P_{CO_2}

BY

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Many patients with chronic non-specific respiratory disease have persistently raised P_{CO_2} levels, without the physical signs of hypercapnia, during periods of relatively good health. In an acute exacerbation the P_{CO_2} may rise still further and the physical signs of hypercapnia may appear. Therefore the significance of a raised P_{CO_2} in an acute exacerbation is in doubt unless the patient's usual P_{CO_2} level is known or unless the hydrogen-ion concentration is estimated.

It would be valuable to be able to assess on clinical grounds the amount by which the P_{CO_2} has been elevated during the course of an acute exacerbation. We have therefore sought a correlation between the physical signs of hypercapnia and the P_{CO_2} estimations of patients admitted to hospital in acute respiratory failure. We have also tried to correlate these signs with the rise in P_{CO_2} above each patient's usual level.

The Series.—A prospective study was made of all patients admitted in respiratory failure to the Central Middlesex Hospital during the winter of 1961–2. Many had previously been seen as out-patients either at that hospital or at the Willesden Chest Clinic.

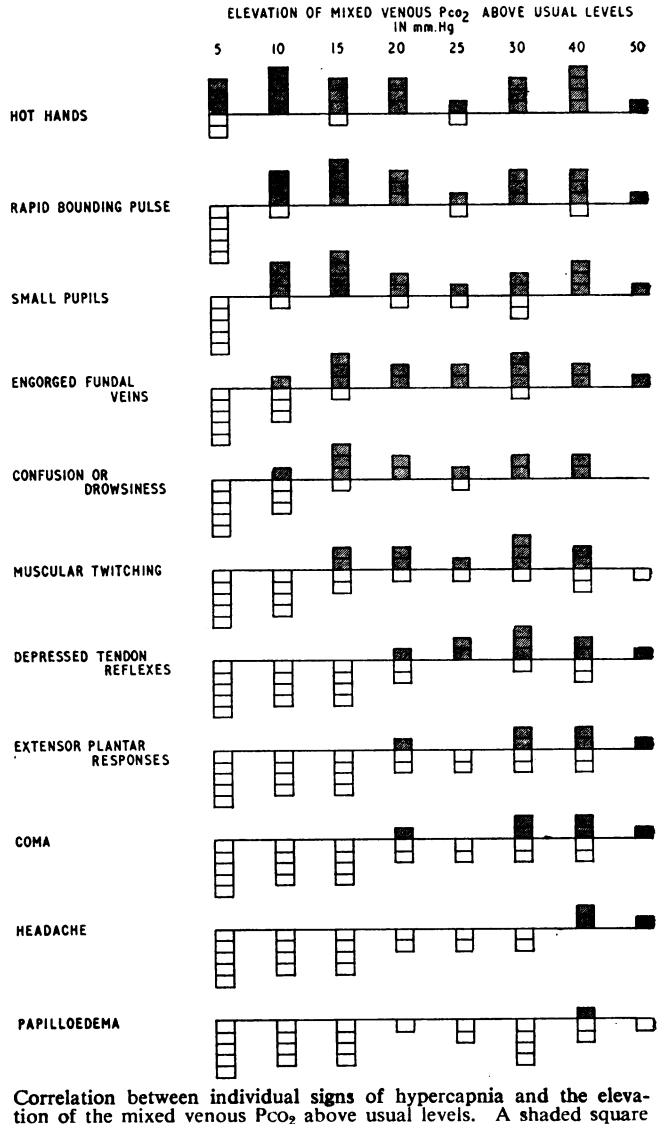
Methods

On admission to hospital of the patients we recorded the presence or absence of each of the physical signs which have been ascribed to CO_2 retention. These are peripheral vasodilatation; a rapid bounding pulse; tremor or twitching, most marked in the forearms; confusion or drowsiness; depressed tendon reflexes; extensor plantar responses; small pupils; engorged fundal veins; papill-oedema; and coma. Headache, although not a physical sign, was also recorded and is discussed as one for convenience. That these signs were not due to hypoxia was shown by the failure of oxygen administration to abolish them.

After the physical examination the mixed venous P_{CO_2} was estimated by the rebreathing method of Campbell and Howell (1960).

Finally, wherever possible we recorded the mixed venous P_{CO_2} during relative health, or the "usual mixed venous P_{CO_2}," as we shall call it. In some cases this was known from previous estimations; in others it was estimated when the patient had fully recovered. Unless it was possible to estimate the usual mixed venous P_{CO_2} level in one of these ways the patient was excluded from the results. Thus no patient in whom the usual mixed venous P_{CO_2} had not been previously estimated and who subsequently died or did not regain his previous state of health is included in this report.

We were able to collect this information for 26 episodes in 23 patients.



Correlation between individual signs of hypercapnia and the elevation of the mixed venous P_{CO_2} above usual levels. A shaded square above the line for each sign indicates its presence in one case; an unshaded square below the line indicates its absence in one case.

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Results

Like previous workers (Westlake, Simpson, and Kaye, 1955; and Sieker and Hickam, 1956), we were unable to find a constant relation between the presence or absence of individual signs and the absolute mixed venous P_{CO_2}. For example, one patient who was in coma on admission had a mixed venous P_{CO_2} of 75 mm. Hg, while another patient, with the same mixed venous P_{CO_2}, had almost no signs of hypercapnia.

However, we were able, in retrospect, to find a better correlation between the presence or absence of individual signs and the amount by which the mixed venous P_{CO_2} was elevated above the usual level for each patient. The details are shown in diagrammatic form in the Chart.

It can be seen from this diagram that hot hands, a rapid bounding pulse, and small pupils were often found with even small elevations of the mixed venous P_{CO_2} (5 to 10 mm. Hg) above usual levels. Engorged fundal veins, confusion or drowsiness, and twitching were found when the mixed venous P_{CO_2} had risen by 15 mm. Hg or more above the usual level. Depressed tendon reflexes, extensor plantar responses, and coma occurred with any frequency only when the mixed venous P_{CO_2} was raised by 30 mm. Hg or more above the usual level. It was only with the highest elevations of the mixed venous P_{CO_2} (40 mm. Hg or more) above usual levels that papilloedema and headache (if the patient was not in coma) occurred.

Discussion

With this information it should usually be possible on clinical examination to arrive at an approximate assessment of the amount by which the P_{CO_2} has risen during the acute exacerbation, this being one index of the severity of respiratory failure. This assessment has a further value: we have discussed in a previous paper (Hamilton and Gross, 1963) the protective effect which CO_2 retention may have on the cerebral oxygen supply. If the P_{CO_2} is to be controlled by respiratory stimulants this should be done with reference to the usual P_{CO_2} level for that patient, and not to the accepted normal level.

Furthermore, the appearance of hot hands, a rapid bounding pulse, and small pupils in a convalescent patient gives warning of increasing CO_2 retention before the neurological signs (which are usually taken as the warning signs) are manifest.

It is not possible to say from this study whether these signs are due to increases of P_{CO_2} or whether they are more closely related to the associated rise in the hydrogen-ion concentration, which seems more probable. A further study, estimating arterial *p*H, would be necessary to decide this.

Summary and Conclusions

It is suggested that there is a quantitative correlation between the appearance of individual signs of hypercapnia and the amount by which the P_{CO_2} has risen during the course of an acute exacerbation of respiratory failure.

Peripheral vasodilatation, a rapid bounding pulse, small pupils, engorged fundal veins, confusion or drowsiness, muscular twitching, depressed tendon reflexes, extensor plantar responses, coma, headache (if the patient is not in coma), and papilloedema, in approximately that order, are shown to occur with successive increases of the mixed venous P_{CO_2} above the patients' usual mixed venous P_{CO_2} levels.

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EFFECT OF BRONCHODILATOR AEROSOL ON VENTILATORY CAPACITY IN CHRONIC LUNG AND HEART DISEASES

BY

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During a review of the results of pulmonary function tests carried out in a diagnostic laboratory note was made of the effect on ventilatory capacity of a bronchodilator aerosol in three groups of patients: those with obstructive lung disease, those with diffuse interstitial pulmonary fibrosis, and those with mitral and aortic valve disease. The findings are here briefly recorded as they are of some therapeutic significance and interest.

Patients and Methods

The number of patients in each diagnostic group, with their age, sex, and ventilatory capacity, is set out in Table I.

Obstructive Lung Disease.—The series of 100 cases is a consecutive one from patients referred for investigation by clinicians from hospital and private practice, both in-patient and out-patient. The diagnosis is based on the clinical findings and the results of tests of ventilatory capacity. All the patients had asthma, bronchitis, or emphysema: those with industrial pneumoconiosis were excluded.

Diffuse Interstitial Pulmonary Fibrosis.—The 22 patients were referred for investigation and consultation from many sources in Victoria, and all were examined clinically by

TABLE I.—*Number of Patients, Age, Sex, and Ventilatory Capacity in the Diagnostic Groups*

		Obstructive Lung Disease		Diffuse Fibrosis	Aortic Valve Disease	Mitral Valve Disease
		Moderate (F.E.V.$_1$/V.C. >0·5)	Severe (F.E.V.$_1$/V.C. <0·5)			
No. of patients	Male	39	38	11	19	20
	Female	11	12	11	3	39
Age (years)	Mean	47·6	54·8	56·3	45·4	39·1
	Range	15–72	14–76	35–72	26–63	14–57
F.E.V.%V.C.	Mean	62·8	37·3	78·1	71·1	75·7
	Range	51·0–86·0	24·1–50·1	53·7–92·5	43·4–93·0	37·1–93·3
F.E.V.$_1$ (litres)	Mean	2·20	0·98	1·97	2·73	2·31
	Range	1·06–4·88	0·15–2·56	0·79–3·20	1·39–4·35	0·95–4·34

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