

A Simple Device for Bedside Measurement of Carbon Dioxide Percentage in Expired Air. (26444)

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One aspect of our long-term study of domiciliary members with emphysema has been the development of relatively simple bedside methods of sufficient accuracy for clinical determination of ventilatory function and expiratory gas exchange(1). Measurement of carbon dioxide content of alveolar air is very valuable but its value to the clinician decreases with the time required to procure a determination by the exacting micro Scholander technic. Three years ago we found a report of a simple device for measuring carbon dioxide content of flue gases which was said to be accurate within $2\frac{1}{2}\%$. On using the apparatus we found that the results obtained were sufficiently comparable for clinical purposes to those obtained by the more refined laboratory technics. The apparatus, known as the Fyrite CO₂ Indicator Model CND* consists of 2 conical chambers connected by a glass tube. The bottom chamber is filled with a 10% potassium hydroxide solution so that some fluid rises in the tube. The upper conical chamber has a valve through which the gas sample enters. An adjustable scale allows reading of the height

of the fluid before and after addition of the gas sample.

The apparatus measures CO₂ saturated with water. To check its accuracy using tank gas, which is dry, it is necessary to run the gas through water to saturate it. Respiratory gases, being water-saturated, need no such treatment.

Expired air may be collected in a bag and subsequently passed into the Fyrite, or preferably, the patient expires through a mouthpiece and tube. The last portion of the expired breath is discharged directly into the apparatus. No special valves are required. The entire operation requires no more than 5 minutes. The following chart shows comparative values of the same expired air gas samples in 20 normals, using the micro Scholander and the Fyrite methods.

The results obtained by the simple Fyrite method vary from those obtained by the micro Scholander technic by an average of .216. This is certainly accurate enough for clinical purposes.

Recently, Ravin and Stein(2) have described an instrument which seems to be ade-

TABLE I. Comparative Studies on Determination of CO₂ in Expired Air on Normal Control Group Using the Scholander Analyzer and Fyrite CO₂ Indicator - Model CND.

Subject	Scholander	Fyrite	Subject	Scholander	Fyrite
1	3.10	3.00	11	3.47	3.25
2	3.77	4.00	12	3.52	3.75
3	3.29	3.50	13	2.26	2.70
4	3.38	3.50	14	3.22	3.00
5	3.31	3.60	15	3.11	3.25
6	3.21	3.40	16	3.15	3.00
7	3.23	3.10	17	2.83	2.75
8	3.41	3.50	18	3.55	3.25
9	3.29	3.00	19	2.80	3.00
10	3.18	3.50	20	3.12	2.75

Avg difference .216

Scholander analyzer			Fyrite CO ₂ indicator - Model CND		
No. of cases	Mean and S.E. of mean		No. of cases	Mean and S.E. of mean	
20	3.20 ± .078		20	3.24 ± .055	(p > 0.5)

* Bacharach Industrial Instrument Co., Pittsburgh, Pa.

quate for bedside studies. It requires much more care and time for its use than the apparatus described above.

We have also used the Fyrite Oxygen Indicator, which is similar in design and use to the CO₂ Indicator. In general, we have not found it as satisfactory when checked against the micro Scholander. This is of little importance since alveolar O₂ concentrations are of much less value to the clinician than the CO₂ concentration.

Summary. The Fyrite CO₂ Indicator Model CND is a satisfactory instrument for bedside measurement of carbon dioxide content of expired air.

1. Cole, M. B., Hammel, J. W., Manginelli, V. W., Lawton, A. H., Meindersma, M. S., *Dis. Chest*, 1960, v38, 519.
2. Ravin, H. A., Stein, M., *New Eng. Med. J.*, 1958, v259, 811.

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Correlations Between Active Eyelid Closure and Depletion of Brain Biogenic Amines by Reserpine.* (26445)

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In mice and rats active eyelid closure (blepharospasm) is used to evaluate the degree of sedation induced by reserpine(1). Neither guanethidine (15 mg/kg i.p.), which depletes norepinephrine stored in peripheral nerve endings(2), nor Bretyllium (20 mg/kg i.p.), which prevents its release(3), induces active eyelid closure in rats. This suggests that catecholamines stored at peripheral nerve endings are not directly involved in active eyelid closure induced by reserpine. The purpose of this paper is to elucidate the role played by depletion of brain biogenic amines in the mechanism of reserpine induced blepharospasm.

Our results suggest a positive correlation between active eyelid closure and depletion of serotonin (5HT) in the brain but fail to demonstrate a similar correlation with regard to brain norepinephrine.

Method. Male Wistar rats of 150 to 200 g were used in these experiments. Increase of motor output induced by drugs was evaluated with revolving cages. The animals were placed in the cage immediately after drug injection and kept there for 2 hours. A crossover design was used; the interval be-

tween retesting of each animal was one week. Eyelid closure was evaluated according to the method of Rubin *et al.*(1). Brain stem norepinephrine was determined according to Bertler *et al.*(4) using perchloric acid extracts. Brain stem 5 HT was measured by a bioassay technic using the rat uterus(5). Brain stems of 2 to 3 animals were combined and homogenized at 0°C with 19 volumes of 0.3 M ice cold sucrose containing 2.5×10^{-4} M of tranlylcpromine. An aliquot (5cc) of the homogenate was removed for total 5 HT analysis, a second aliquot (10cc) was used for perchloric acid extraction and a third aliquot (5cc) of the homogenate was centrifuged in a Spinco Model L ultracentrifuge with rotor No. 40 for 40 min. at 20,000 rpm, equal to 26,360 g. Serotonin analysis was then performed separately in supernatant and precipitate. The monoamineoxidase inhibitor (MAOI) d-l tranlylcpromine (SKF 385), Reserpine (Serpasil), and d-amphetamine sulfate were given alone or in combination to groups of at least 6 rats. Doses, route of injection, and time elapsed between treatments are indicated in Results.

Results. The effect of tranlylcpromine on brain amines of rats is illustrated in Fig. 1. Two mg/kg i.p. of the drug caused an increase of brain 5 HT which reached its peak

* A preliminary report of these experiments was presented at Fed. Am. Soc. Exp. Biol., Chicago, April, 1960 (*Fed. Proc.*, 1960, v18, 279.).