with the levels estimated in the untreated group (mean 2·9 mgm. per cent *S.D.* ± 0·63 mgm. per cent). By calculating a common variance, comparison of the mean values for each group reveals a significant difference at the 0·1 per cent level. The elevated plasma magnesium-level in the treated group is associated with a mean plasma salicylate-level of 51 mgm. per cent (*S.D.* ± 16·8 mgm. per cent).

Taken with the finding of decreased excretion of Mg^{++} under similar conditions of intoxication[1] it is apparent that a retention of magnesium ions is produced by the administration of salicylate.

Since the early observations of Winter and Barbour[5] magnesium has been used both experimentally and clinically to reduce body temperature under a wide variety of conditions[6]. The rise in plasma magnesium which is associated with high plasma salicylate-levels may therefore explain the antipyretic effect of this drug.

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Protective Action of γ-Aminobutyric Acid against Oxygen Toxicity

The effects of the inhalation of oxygen at greater than normal pressures have assumed greater importance as man's penetration into the depths of the sea has increased. It was Bert[1] who for the first time revealed that, although oxygen at low pressures sustains life, the gas is toxic at high pressures. In their review of the subject, Stadie *et al.*[2] conclude that the dominant symptoms of the action of oxygen at 0·8–2·0 atmospheres are respiratory, while at higher oxygen pressures central nervous system symptoms predominate. Several theories have been put forward to explain the toxic effects of oxygen at high pressures[3], but no one theory is completely satisfactory.

Of the animal tissues and fluids which have been examined for γ-aminobutyric acid (GABA), only those from the central nervous system contain the amino-acid in detectable amounts[4], and it has been suggested that GABA may play an important part in some aspects of the regulation of physiological activity in the brain[5]. More recent investigations have indicated that treatment of animals with GABA affords some protection against convulsions induced by hydrazides[6] and that human beings with various types of seizures experience significant improvement in their condition as a result of treatment with oral doses of GABA[7].

In view of these findings it was deemed worth while to investigate the influence of GABA on the toxic effects of oxygen at high pressures. The results of this investigation are presented here.

Table 1. Effect of an Oral Dose of γ-Aminobutyric Acid on the Resistance of Rats to Oxygen Toxicity

Exp. No.	No. of rats convulsing		No. of rats with severe convulsions		No. of rats dying within 24 hr.	
	Control	GABA	Control	GABA	Control	GABA
1	3	0	2	0	2	1
2	2	3	1	1	0	1
3	3	3	2	0	3	0
4	3	2	2	0	2	0
5	3	1	0	0	0	0
6	3	2	3	0	2	1
7	3	2	1	0	1	0
8	3	1	2	0	2	0
9	3	1	1	1	1	1
10	3	3	1	1	1	0
11	3	3	2	2	3	3
12	3	3	2	1	2	1
13	3	2	2	1	2	2
14	3	2	0	0	2	1
15	3	2	2	0	2	0
16	3	3	2	1	2	1
Total	47	33*	25	8†	27	12*

* Probability that difference from control rats is significant, $P < 0.01$.
† Probability that difference from control rats is significant, $P < 0.001$.

Male Wistar rats weighing 190–220 gm. were used in the experiments. Two ml. of 0·9 per cent aqueous sodium chloride containing 2 m.moles of GABA were injected intraperitoneally 30 min. prior to exposure of the animals to oxygen at high pressure. Control rats were treated similarly except that the GABA was omitted from the saline solution. Three control and three treated rats were studied simultaneously using the procedure of Walker[8], which entails placing the animals in lucite compartments contained in a pressure chamber. After the apparatus had been flushed well with oxygen for approximately 1 min. the pressure was increased steadily during a 1-min. interval to a gauge pressure of 75 lb./in.^{2}. This pressure was maintained for 40 min. followed by decompression to atmospheric pressure over a period of 2 min. During the exposure to oxygen at high pressure a fan outside the pressure chamber circulated the oxygen and passed the respired gas through a screen of soda-lime to remove the carbon dioxide exhaled by the animals.

The effect of oxygen at high pressure on control and GABA-treated animals is shown in Table 1. A convulsion was scored when an involuntary movement of the body occurred and the seizure was considered to be severe when there was gross and violent movement of the body as a whole. It is evident that GABA reduced significantly the effects of oxygen at high pressure on the treated rats, not only in the number of deaths resulting from oxygen at high pressure but also in the number and severity of the convulsions. Of the control rats having seizures, 53 per cent suffered violent convulsions whereas the corresponding figure for the treated rats was 24 per cent.

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