

Carbon Dioxide Changes in Hyperventilation and Breath-hold Diving

P. G. LANDSBERG

SUMMARY

Breath-hold dives to a depth of 10 metres (33 ft) in fresh water at 1640 metres (6000 ft) above sea level, at atmospheric pressure (P_a) 625 mmHg, were performed by 5 male divers. The water pressure at 10 metres at this altitude is approximately 1.83 ATA (1368 mmHg). Peripheral venous blood from the right antecubital vein was analysed for combined CO_2 content ($^c\text{CO}_2$) by the Van Slyke method and expressed in $^c\text{CO}_2$ vol.%. The CO_2 content of peripheral venous blood is lowered by hyperventilation and increased by repeated breath-hold diving. Individual divers had different rates of CO_2 elimination from blood to alveolar air. CO_2 elimination is also not the same in any one diver from day to day. It is postulated that the effects of hyperventilation are unpredictable in different divers under the same diving conditions. This may have contributed to the deaths of several divers in South Africa.

S. Afr. Med. J., **48**, 18 (1974).

Under conditions of normal atmospheric pressure, breath-holding results in important changes in the mechanism whereby the CO_2 is transported and exchanged in the lungs, but a reversal of CO_2 from the lungs to the blood does not occur unless the inspired air contains a high concentration of CO_2 .¹

Under conditions of raised atmospheric pressure, as occurs in breath-hold diving, the whole aspect of CO_2 transport and exchange is markedly altered and cannot be equated with apnoea at normal atmospheric pressure. Prolonged apnoea under raised atmospheric pressure results in a reversal of CO_2 exchange from the lungs to the blood.²

Arterial pCO_2 will increase and exceed venous pCO_2 during apnoea, simply as a result of the oxygenation of haemoglobin in the face of falling CO_2 output to the lungs (Haldane effect). During apnoea a shrinkage of lung volume occurs, suggesting that CO_2 output decreases, while O_2 uptake is continuous.² Schaeffer² showed that the flow of CO_2 into the lungs during ascent from a dive is regulated in part by the speed of ascent. Mithoefer¹ offered an explanation for the 'aberrations in CO_2 transport and exchange' which occur during apnoea with O_2 . Craig² in 1961, showed that unconsciousness following

hyperventilation and breath-hold diving, was probably caused by hypoxia.

In 1963, Lanphier,¹ working with 4 divers under simulated breath-hold diving conditions in a compression chamber, established the course of alveolar gas exchange under pressure and also determined the time course of venous CO_2 content during simulated dives. He worked in a pressure chamber in order to determine accurately the pressure-time relationships and to facilitate the taking of blood samples. He concluded that 'CO₂ narcosis' may cause unconsciousness in prolonged, deep breath-hold dives. Hong,² in a field study of Korean diving women, noted that they did not hyperventilate to any great extent and followed the relatively safe pattern of the 60-second dive to 10 metres in which neither hypoxia nor CO_2 accumulation is a serious hazard.

A South African study by Smit³ in 1967, determined the alveolar CO_2 % and O_2 % after breath-holding in 2 divers in a swimming-bath no blood CO_2 levels were determined.

The first South African field study was conducted in 1968 and is described in this article. The time course of peripheral venous CO_2 in divers during hyperventilation and breath-hold diving is illustrated and an attempt is made to explain the unpredictable hazards of this practice in human divers.

METHOD

Five healthy, experienced male divers participated in the experiment. All divers were trained spearfishermen or SCUBA (Self-Contained Underwater Breathing Apparatus) members of the Transvaal Sub-Aqua Club in Benoni. Divers were kitted up on land in wetsuits, weight belts, masks, snorkels and flippers. All venous blood samples were taken from the right antecubital vein. The divers then forcibly hyperventilated on land for 30 seconds and the second blood sample was taken immediately thereafter. The divers then rowed themselves in a rubber dinghy for about 100 metres to the middle of Cinderella Dam, Boksburg, Transvaal, taking about 150 seconds. Each diver, after adequate rest, then hyperventilated forcibly on the surface and dived to 10 metres 3 times in rapid succession, as occurs during spearfishing conditions when spearfishermen dive repeatedly after short rest periods. The third blood sample was taken immediately on surfacing; the diver's right arm was placed over the side of the dinghy to facilitate venepuncture. The blood samples were each taken in a sterile plastic syringe and sealed under a layer of liquid paraffin in a 5-ml bottle. The blood

Department of Physiology, University of Pretoria

P. G. LANDSBERG, M.B. B.CH.

Date received: 6 February 1973.

was taken between 1600 and 1800 on 31 March 1968, and analysed on 1 April 1968, in the SAIMR laboratory at the Boksburg-Benoni Hospital.

RESULTS

The divers did not over-exert themselves and surfaced as soon as the desire to breathe was apparent. All divers managed the three 10-m dives within about 60 seconds. All divers became dizzy during and immediately after forced hyperventilation for 30 seconds on land.⁶ Hyper-

ventilation before diving was just as forced as on land and 3 divers (B, C, D) were still dizzy on surfacing. At no time were any of the divers distressed. The CO₂ content of the peripheral venous blood was determined by the Van Slyke method and expressed in volume % (Table I). Means were determined for each group of blood samples and the standard deviations were calculated (Table I). The time course of CO₂ content (°CO₂) in vol. % for resting, hyperventilation and 3 dives to 10-m levels, is shown in Fig. 1.

TABLE I. AVERAGE VALUES AND STANDARD DEVIATIONS OF °CO₂ LEVELS IN DIVERS

	Resting	Hyperventilate	Hyperventilate and dive
	47,5	44,7	55,1
	46,5	47,5	58,9
	51,3	49,7	55,1
	52,2	52,2	54,1
	54,1	55,1	47,5
Mean	50,3	49,7	54,1
SD	1,27	1,62	1,66

DISCUSSION

Interpretation of Results (Fig. 1)

When considering the mean values °CO₂ vol. % ± SD it was found that: (i) the resting °CO₂ = 50.3 vol. % in this group of divers; (ii) this decreases to 49.7

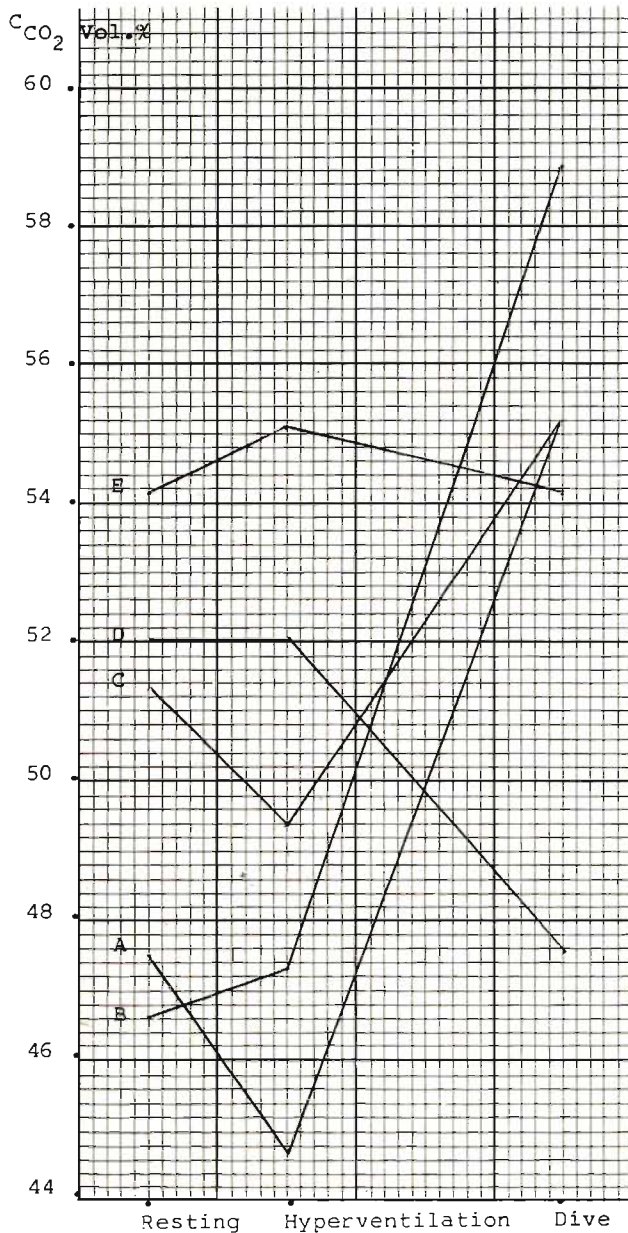


Fig. 1. CO₂ content of peripheral venous blood during hyperventilation and breath-hold diving.

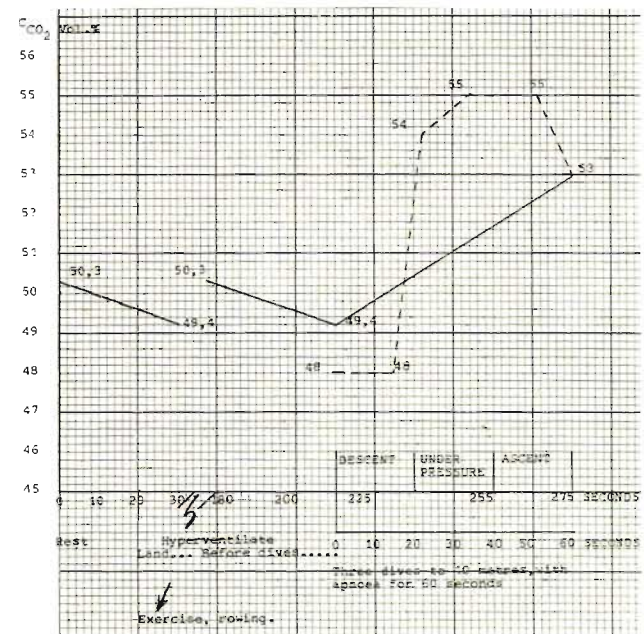


Fig. 2. Comparison of the results of this study on hyperventilation and breath-hold diving with those of Lanphier on simulated breath-hold diving without hyperventilation.

vol. % after forced hyperventilation, (iii) this increases to 54,1 vol. % after forced hyperventilation and repeated breath-hold diving; (iv) because of the relationship between °CO₂ vol. % and the pCO₂ in the peripheral venous blood of divers, it may be concluded that in general the peripheral venous blood CO₂ levels in trained divers are reduced by hyperventilation and increased by repeated breath-hold diving; (v) this is not true in all cases as divers show a variation in the diffusion of CO₂ in and out of the blood

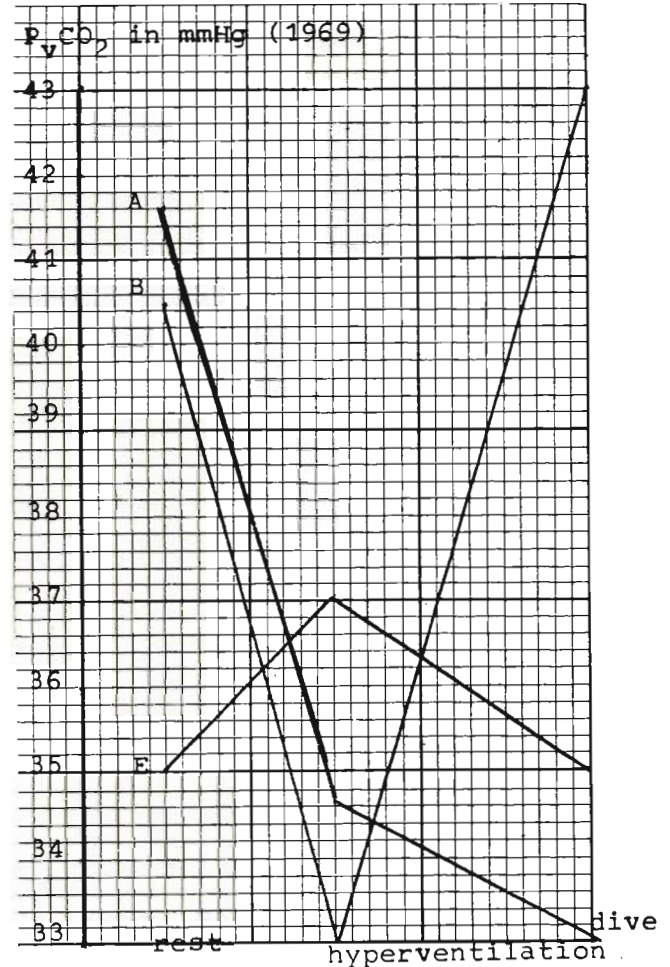
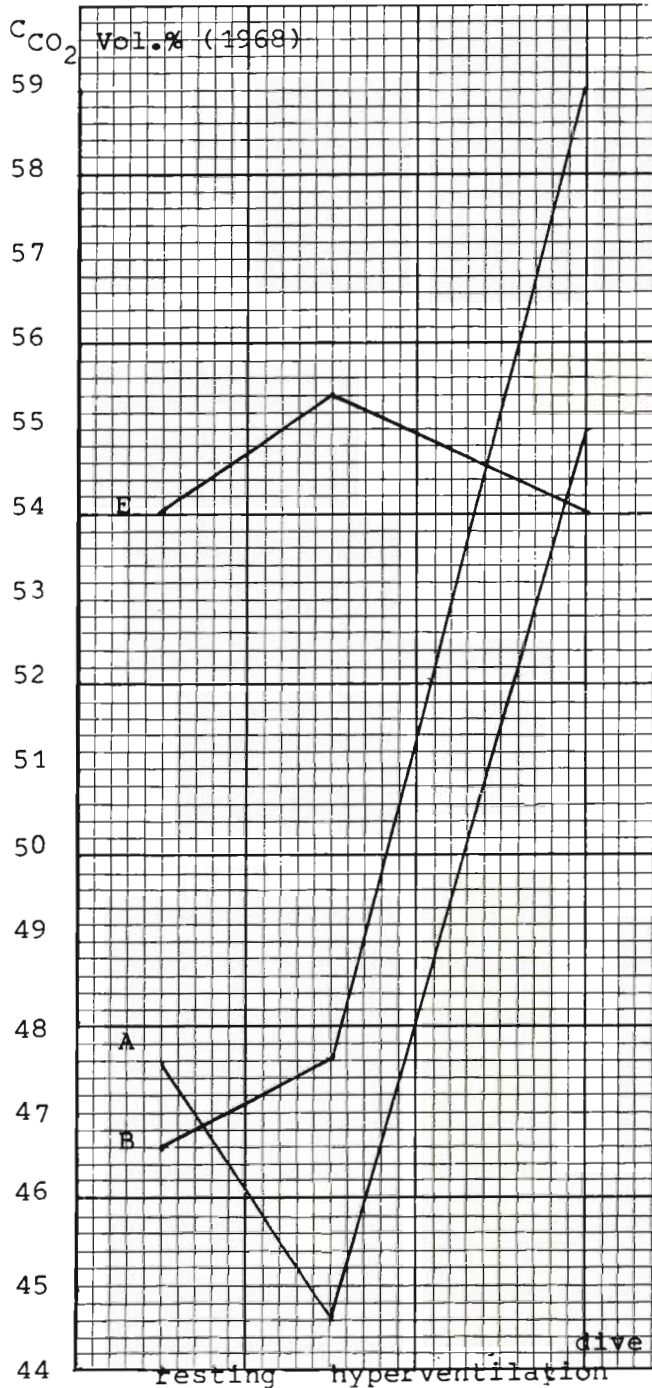
stream. This may possibly explain the unpredictable nature of the effects of hyperventilation in divers.

Time Course of °CO₂ in Breath-hold diving (Figs 2 and 3)

Lanphier¹ was able to make more CO₂ estimations working under dry simulated dive conditions in a compression chamber without prior hyperventilation and his graph is superimposed on the graph representing the present finding (Fig. 2).

The graph in Fig. 3a is the same as the analysis graph in Fig. 1 and shows 3 different patterns of CO₂ content in the peripheral venous blood. The graph in Fig. 3b shows the same divers on a different occasion, when the peripheral venous CO₂ was measured in mmHg by the Astrupp method. The data obtained under the field conditions did not permit conversion of the graphs to the same units.

A comparison of the 2 graphs shows that only diver



Figs 3 a and b. Comparison of fluctuating CO₂ levels in the peripheral venous blood of divers under the same diving conditions but on different occasions.

E has the same pattern of peripheral venous CO₂ change during the hyperventilation breath-hold dive cycle, showing that it is impossible to predict the changes that take place during a breath-hold dive. The factors responsible for this are:

1. The different tidal volumes in divers.⁷
2. The variable rates of CO₂ diffusion in and out of the lungs; this cannot be measured at present.³
3. The physical degree of hyperventilation.
4. The amount of CO₂ produced by the exercise of hyperventilation which is added to the CO₂ produced during the dive.³
5. The activity of the diver while submerged; this may be considerable in a spearfisherman.
6. The time that a diver is submerged; this may be up to 90 seconds.^{1,2}
7. The rate of ascent.³
8. The length of recovery period on the surface before the next dive; adequate time must be allowed to blow off the accumulated CO₂ from a large reservoir.³
9. The diver may wilfully 'shut-off' or ignore the physiological alarm set up by the high blood CO₂: stimulating the respiratory centre and urging him to breathe.¹

Individual Objective Observations

Two divers (A and C) had cyanosis of the lips on surfacing, indicating low blood O₂ levels. Divers A and C showed a drop in °CO₂ after hyperventilation on land. This is the expected result. Divers B and E showed a rise in °CO₂ after hyperventilation on land (Fig. 1). Increase of CO₂ in the peripheral venous blood may not be readily determined after a dive because of the CO₂ buffering action of the blood and the decrease in peripheral blood flow during diving⁹ and the variable rates of ascent in the diver.³ Their hyperventilation may not have been sufficiently forceful to affect the buffering systems in the blood. Also the rate of diffusion of CO₂ in or out of the blood may differ from diver to diver—a physiological 'fluctuation'.¹ The CO₂ diffusion process also takes time and may not be reflected after 30 seconds.³ Hyperventilation also increases respiratory muscular work with increased CO₂ production.³ The diver's tidal volume will also affect his hyperventilation and CO₂ diffusion rate which is impossible to measure at present.^{7,8}

These points illustrate why hyperventilation, prior to breath-hold diving to increase the period of apnoea underwater, is a physiologically dangerous practice, as the CO₂ decrease so produced may not regain a sufficiently high concentration during the period of apnoea to stimulate the respiratory centre to initiate breathing. They also explain why some divers are adversely affected by hyperventilation, and that the effects of hyperventilation are unpredictable at any time during the hyperventilation breath-hold dive cycle. Divers A, B and C all showed a marked rise in CO₂ content after apnoea. This is probably owing to the accumulation of metabolic CO₂ and the re-

versed CO₂ gradient that occurs during underwater breath-hold diving.¹ Divers D and E, however, show a drop in °CO₂. This paradoxical result may be explained by variable alveolar CO₂ accumulation rates found in different divers on ascent after a breath-hold dive,³ depending on the rate of ascent, e.g. fast ascent—low alveolar CO₂, high blood CO₂. Divers D and E may have had a rapid diffusion of CO₂ from blood to alveolar gas,¹ e.g. slow ascent—high alveolar CO₂, low blood CO₂—and may also not have produced large amounts of metabolic CO₂ during their underwater stay. This paradoxical finding may also be explained by the decrease in peripheral blood flow that occurs during breath-holding, i.e. the °CO₂ change will not be shown as readily in the peripheral as in the central mixed venous blood.⁹

It may be important to determine the variability of CO₂ diffusion gradients in the selection of personnel for diving training, especially naval divers¹⁰ and submariners.

CONCLUSIONS

This experiment was initiated in part by the drowning of an expert, world-class South African spearfisherman on 8 March 1968. What were the factors which contributed to the tragic and unnecessary death of a superbly fit and very experienced diver? Spearfishermen admit that they hyperventilate, otherwise their deep diving in search of fish becomes physiologically impossible. Hyperventilation gives them 10-20 seconds extra 'bottom time', as shown by Lanphier,¹ but increases the danger of hypoxic 'black-out'. They appear to understand the dangers of hypoxic 'black-out' on ascent. They control their hyperventilation prior to breath-hold diving and appear to heed the physiological alarms set up in their bodies urging them to the surface to breathe. In spite of this, several have arrived on the surface unconscious, automatically breathing a mixture of water and air through their snorkels until they overcome their hypoxia and regain consciousness, coughing, cyanosed and bewildered, yet diving again after a period of rest. Some 'dive on their watches', taking 75 seconds to dive to ± 20 metres (surface to surface). They have worked out their individual dive times by experience with previous hyperventilation and near-'black-outs'. The effects of hyperventilation in divers are unpredictable, as we cannot predict a high or low level of CO₂ at any point in the hyperventilation breath-hold dive cycle. It is therefore very difficult for an experienced diver who has been hyperventilating prior to breath-hold diving for many years, to be dogmatic about his ability to heed the physiological alarms that he may experience during a dive. One day he may have a perfect CO₂ accumulation and stimulation of his respiratory centre with rapid diffusion of CO₂ from blood to alveolar air taking place long before hypoxia causes unconsciousness. The next day CO₂ accumulation may be slow due to excessive hyperventilation, or CO₂ diffusion into the alveolar air may be slow due to rapid ascents, causing excessive CO₂ accumulation in the blood, which produces 'CO₂ narcosis'.¹ I tend to agree with Lanphier¹ that hypoxia, which occurs owing to the breath-holding and underwater exercise combined with these factors, will cause

unconsciousness and drowning. Another factor involved is the overriding competitive spirit that prevails at spear-fishing competitions, which causes the diver to wilfully shut off his physiologically protective alarm systems.⁸

REFERENCES

1. Lanphier, E. H. and Rahn, H. (1963): *J. Appl. Physiol.*, **18**, 471.
2. Craig, A. B. jun. (1961): *Ibid.*, **16**, 583.
3. Schaefferr, K. E. in Bennett, P. B. and Elliot, D. H. eds (1969): *The Physiology and Medicine of Diving and Compressed Air Work*, p. 144. London: Tindall & Cassell.
4. Mithoefer, J. C. (1959): *J. Appl. Physiol.*, **14**, 706.
5. Hong, S. K., Rahn, H., Kang, D. H., Song, S. H. and Kang, B. S. (1963): *Ibid.*, **18**, 457.
6. Smit, P. J. (1967): *Voeding*, **28**, 380.
7. Miles, S., Dean of Post-graduate Medical Studies, University of Manchester, England (1971): Personal communication.
8. Donald, K. W., Professor of Medicine, Royal Infirmary, Edinburgh, Scotland (1971): Personal communication.
9. Dahn, I. and Pauley, P. (1966): *Acta physiol. scand.*, **68**, 277.
10. Deppe, A. H. (1971): 'A study of psychological factors relevant to the selection of Scuba divers', p. 44, D. Litt. et Phil. thesis, University of South Africa.