SHALLOW WATER BLACKOUT

by

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**Glossary**

C.A.B.A. Compressed air breathing apparatus - equipment which supplies compressed air to a diver. This may be either a portable gas cylinder strapped to the diver (SCUBA, Aqualung etc.) or a gas supply from the surface (SCBA, compressors etc.).

C.C.B.A. A closed circuit breathing apparatus. The equipment discussed in this project refers only to the Patt 5561, i.e. a closed circuit apparatus with pendulum flow of gas between the subject and counterlung, viz. the CO₂ absorbent canister. The gas is either 100% O₂ or a O₂/H₂ mixture.

Hypercapnoea - increased carbon dioxide tension.

Hypocapnoea - decreased carbon dioxide tension.

Hypoxia - inadequate oxygen tension.

Multifactorial Etiology - refers to the concept that any result has multiple causes, and that each of these must be appreciated. It is the antithesis of a cause/effect relationship.

Oxygen Syncope - a term devised by Miles to explain the occurrence of Shallow Water Blackout (SWB) in divers using oxygen equipment (CCBA).

Shallow Water Blackout (SWB) - loss or impairment of consciousness occurring at shallow depths (less than 25' - 30').
INTRODUCTION

Unconsciousness in divers using a breathing apparatus, and occurring at shallow depths (30ft. or less), was an enigma during the early days of CEEA diving. It was recognised as a syndrome by the divers, but was placed under diagnostic labels by physiologists and doctors (references 1, 10; 11 & 15). We find such cases listed under "Syncope", "Unconsciousness", "Shallow Water Blackout" and "Oxygen Syncope" - depending on the etiological bias of the observer.

Over the last decade physiologists and underwater medicine specialists have focussed their attention on the more esoteric fields of deep diving, inert gas narcosis, precise decompression schedules and sea-bed habitation. This shift in interest is not to be criticised, as it is here that new frontiers are being forged. The unfortunate corollary is that the problems of the run-of-the-mill diver are less attractive than those of the fashion orientated aquanaut.

It is too common, when dealing with diving incidents, to meet the comment "We do not seem to have that problem any more", from people who have lost contact with both the problems and the divers. Many towers also descend under the sea.

In carrying out an informal survey in the RAN it was noted that there were many more diving incidents in the water, than there were records in the appropriate file. Problem solving relies on problem identification and this depends on good communication. When one hears "there is no problem", the accurate translation may well be "there is no communication".

In the writers opinion, Shallow Water Blackout (SWB) is an example of such a communication breakdown. The incidence of this problem shows a marked variation and depends on the source of information - whether one speaks to an experienced diver, an instructor or a doctor. The source which appears most incomplete is the official records. These comments seem applicable to the RN and RAN. In the USN, incidents of "syncope" have increased in number (reference 2).

In this paper the possible causes for SWB have been reviewed. Some of the concepts held with venerable esteem have been refuted and others supported. The aim has been to test some of the major etiologies which have been proposed. The investigation covers fields of respiratory physiology, equipment, statistics and conjecture. This selection was dictated by those who have committed their ideas on etiology to paper.

The subject is discussed under the etiological headings:

(i) Multifactorial etiology.
(ii) Hypercapnic SWB.
(iii) Oxygen Syncope.
(iv) Hypoxic SWB.
(v) Hypocapnic SWB.
(vi) Physiological Sequelae of Ascent.
I MULTI-FACTORIAL ETIOLOGY.

The protagonist of this theory in Underwater Medicine is Miles (reference 10). Miles considers that much confusion results from an attempt to find a single common cause for this problem and believes that the final syncope is due not to one factor but to the summation of two or more which separately, though reducing the syncope threshold, would not produce unconsciousness. Together, it is claimed, they would result in syncope. Miles calls on the work of both Franks and Howell, (references 7, 13) as well as his own extensive experience in Underwater Medicine, and documents many such factors. "Oxygen Syncope will only occur if there is some predisposing factor".

The two or more factors which may summate to produce the syncope are derived from a whole group of conditions mentioned by Miles, namely

Breathing O₂ at a high partial pressure.  
Hypoxia.  
Hunger (or hypoglycaemia).  
Hyperventilation (or hypocapnic alkalosis).  
Alcohol.  
Over indulgence in food.  
Increased intra-pulmonary pressure (with Valsalva manoeuvre).  
Relaxation after effort.  
Anxiety (psychological stress).  
Acute infections.  
CO₂ retention.  
Pain.  
Fatigue.

The objections to a multi-factorial approach are twofold:

1. The inadequacy of a multi-factorial approach in any scientific field, is that unless the factors can be quantitatively assessed the approach is heuristically sterile. The corollary of this is that whenever a result is observed, one has so many causes from which to select, that there are satisfactory answers and explanations available without further inquiry, thought or investigation.

A single and simple hypothesis can and must be regarded with suspicion, as it will almost certainly prove inadequate. However, if one does investigate a single hypothesis one can then quantify its importance. A multi-factorial approach will exonerate us from scepticism and from advancement of knowledge; a single working hypothesis of causation will encourage further investigation and testing. Shallow Water Blackout should be considered a symptom of one or more diagnostic entities. Each diagnosis should be named and this implies a judgement of causation and a plan of action i.e. treatment.
MULTI-FACTORIAL ETIOLOGY (Cont.)

There is no limit to the analysis of causes; genetic, anatomical, physiological, psychological and even sociological, many of which are not mentioned by Miles. Accepting the multi-factorial etiology gives a feeling of omnipotence and wisdom - we can always give an impressive answer. The only lasting value of a multi-factorial approach is in suggesting a series of causes each of which may be assessed independently and their interactions investigated.

If knowledge is to advance, it is necessary to quantify the relative significance of various causes. If, as in our present example of SWB, alcohol is one causative factor, then it could be related to the type of alcohol taken, the period over which it was ingested, the relative absorption rates under different conditions, the patient's individual susceptibility to alcohol, his psychological response, hepatic function, dietary indiscretions etc. However, before investigating all these factors, it is essential to verify the extent to which alcohol per se is a factor in the SWB. To accept this belief because it has face validity, can only retard our diagnostic approach and final knowledge of SWB.

2. There is a specific flaw in the application of the multi-factorial approach to this disorder. Miles points out that it is significant that all cases of syncope occurred in men breathing 100% O₂, and no cases were found where air was breathed, even among the very large numbers of civilian recreational divers using aqualungs (CABA). The observation that it is essentially found in subjects breathing O₂ from rebreathing apparatus (CABA) and is rarely observed with open circuit breathing apparatus (CABA) has received support (references 1, 6, 11 and 15). However, many of the factors mentioned e.g. acute infections, alcohol, hyperventilation, valsalva techniques, fatigue, panic reactions etc. are as much applicable to CABA as to the O₂ supplying CABA. If any two of these factors may combine to give syncope than they seem selectively lenient to the CABA divers. When it is also considered that there must be many hundreds, if not thousands, of hours spent by amateur, Naval and professional divers with CABA, compared to each hour spent with CABA, one would imagine that the prevalence of SWB should be much greater with the CABA than it in fact is. Miles could find no such cases. For this reason it is hard to believe that the multi-factorial etiology is appropriate.

This criticism in no way detracts from the common sense recommendations made by Miles, and which presumably reflect his extensive experience in the field of Underwater Medicine.
Barlow and Macintosh in 1944 (reference 1) described "shallow water blackout" a syndrome occurring after a time, at depths less than 25 feet, while breathing 100% O₂ in a closed circuit. It was usually preceded by hard physical work. The subjective symptoms were vague e.g. haziness, dizziness, sleepy feeling. The diver, unless he was aware of the risk of SWB, usually had no immediate desire to stop work. The sensation was not an unpleasant one. The objective appearances were; differing degrees of impairment of consciousness, flushed appearance, rapid respiration, shock, headache and nausea occurred with, or followed, the syndrome and lasted an hour or more.

After considering the possible causes, circulatory disturbances and anoxia were dismissed, as were O₂ intoxication and hypercapnia. After carrying out some experiments on the effects of hypercapnia on land, it was concluded that SWB was a manifestation of CO₂ toxicity. It was stressed that some canisters with CO₂ absorbers were inadequate when the subjects performed heavy work. If was noted that an inefficient canister would be functionally equivalent to increasing the dead space.

Surgeon Lieutenant Commander H.M. Balfour (reference 1) suggested a test whereby the diver rebreathes from his counterlung, without absorber in the canister. The diver would rebreathe the CO₂ until the CO₂ accumulated to a sufficient level to produce symptoms, he would then presumably be aware of the symptoms of the CO₂ intoxication, and recognise these under operational conditions.

A Superintendent of Diving Memorandum (reference 15) claimed that SWB, continued to occur in spite of improvements of canister design and it was found that the CO₂ percentage in the expired air could rise to 6-8% when the percentage in the inspired air was less than 0.2%. It seemed to agree with Barlow and Macintosh, that the evidence for CO₂ intoxication was fairly convincing and it was thought that this may occur because of the presence of a high tension of O₂. With this high O₂ pressure, the body was said to lose its normal response to CO₂, thus the respiratory centre, desensitized by the high O₂ tension, might allow CO₂ to build up to an anaesthetic level. A request was made to the Underwater Physiology Sub-Committee to investigate the mechanism of SWB and the possible measures to be taken in reducing or preventing the incidence of this disorder.

The RAM School of Underwater Medicine was instructed by the Naval Board to investigate the CO₂ absorber effectiveness in the O₂BA. The results of those investigations are recorded in SUM Project 2/68 (reference 6). A summary of the conclusions is as follows:
HYPERCAPNOEIC S.W.B. (Cont.)

The effectiveness of the equipment was related to the deteriorating efficiency of the CO₂ absorber and SWB would be likely to occur any time after 30 minutes, if the diver was performing heavy work. The time at which the set became ineffective varied with the CO₂ clearance requirement e.g. a set which was effective for one activity could become ineffective when that activity was increased. Thus the set would produce hypercapnoeic SWB at the very time when the diver exerts his greatest effort i.e. in an emergency.

On land the subject notices hyperpnoea, headaches, ataxia, spatial disorientation and dizziness, restlessness, sweating of his forehead and hands, visual distortion and colour distortion. His face feels "full" and warm and he appears somewhat bloated (as if he had indulged himself in alcohol). Halitosis and headache follow the episode and may last some hours. Muscular fasciculation and inco-ordination is demonstrable. Gross jerking movements may occur in the limbs. Respiration is increased in both rate and depth. The subject will often continue his job despite instructions to the contrary. Gross tremor and convulsions may occasionally occur. Stupor and coma may occur and under loss controlled conditions may result in death.

In the water, inco-ordination and ataxia is much less noticeable - as this environment minimises the effects of gravity and one is seldom performing delicate movements such as picking up small objects. Sweating, and the uncomfortably hot feeling, is countered by the cool environment. Hyperventilation is not noted by the diver doing hard work, or a purposeful task. Three of the subjects losing consciousness in the RAN series complained of transitory ambyopia and one complained of light headedness. These prodromal symptoms occupied only a few seconds. Unconsciousness is a likely result of CO₂ toxicity, before the diver can take appropriate action.

The concept of hypercapnoeic SWB, introduced by Barlow and MacDonald, has ample experimental evidence to support it. It is a predictable and consistent syndrome and can be verified by analysis of the expiratory gas. The possible causes for the hypercapnoeic state may be considered as:

(a) Failure of CO₂ absorption because of absorber inefficiency.
(b) Increased functional dead space due to deep respirations, less time for CO₂ to be absorbed and a greater volume of non-functioning CO₂ absorber in the canister.
(c) Physiological CO₂ build-up despite adequate ventilation, in the presence of high CO₂ tensions and the resultant interference with CO₂ transport.
III OXYGEN SYNCOPE

The neurological effects of oxygen toxicity are well known and documented (reference 4, 14). When the partial pressure of $O_2$ is in excess of 2 ats absolute, consequences may occur which are reproducible. These include: facial pallor, fasciculation of the lips, facial perspiration, salivation, nausea and vertigo, mood changes, aurae of many types, constriction of the visual fields, irregularities of respiration, and epileptic convulsions.

When faced with cases of unconsciousness occurring with divers who breathed $O_2$ through a CGBA at less than 2 ats absolute there was a natural tendency to wonder whether $O_2$ had a sub-lethal effect, producing unconsciousness. No support for this concept has been obtained by ECG studies (Reference 4, 16).

A concept of "oxygen syncope" was devised by Miles (reference 10). He noted that unconsciousness in underwater swimmers was especially related to:

1. Oxygen breathing.
2. Depths less than those acceptable for $O_2$ toxicity.
3. Use of CGBA.
4. Inexperienced divers.

The first 3 points were noted by Barlow and MacIntosh (reference 1) many years previously. On a priori grounds, the unconsciousness is unlikely to result from a specific result of the depth, as divers using compressed air do not have such complications at the same depth. Although one hypothesis for SWS could have incriminated the equipment and its interference with respiratory function, Miles chose $O_2$ as the likely cause - despite the knowledge that $O_2$ was at pressures not normally considered to have deleterious effects on the central nervous system, or the cardiovascular system. He suggested the term "oxygen syncope" to replace that of SWS.

Syncope, by definition a loss of consciousness due to transient cerebrovascular insufficiency, has not been demonstrated in underwater swimmers. It seems unlikely because of 3 factors.

1. Oxygen causes vaso-constriction, not vaso-dilation, which is a common association of syncope.
2. The diver is usually in the horizontal position, and relatively unaffected by gravity. Gravity is a contributing factor to most syncope.
3. A far more important criticism of the "oxygen syncope" concept lies in the actual experiments performed by Miles. His series of experiments were devised to prove the syncope inducing effects of $O_2$. It is on the basis of these experiments that the justification for this concept rests. The concept received widespread acceptance and is now an integral part of reviews on the subject of SWS. The results given by Miles require careful assessment. This is done in Appendix A.
One is faced with the ironical situation of using Miles' results to refute his conclusions and explain his findings. The syncope could, under his conditions, be due to the breath-holding time. As such it is not without precedence in general medicine. Micturition syncope and cough syncope are thought to be related to a maintenance of raised intra-thoracic pressure obstructing venous return, resulting in diminished cardiac output and insufficiency of the cerebral blood supply.

These conclusions were easily tested. In Appendix B experiments are described which tested the hypothesis that:

1. Syncope is related to O₂ inhalation.
2. Syncope is related to hyperventilation.
3. Syncope is related to the mechanics of equipment used, with hyperventilation.

Only (1) need be considered here. Of the 80 tests performed, the number who developed unsteadiness were:

29 breathing air (out of a possible 40).
28 breathing O₂ (out of a possible 40).

These groups were tested under identical conditions, but in an alternating sequence.

Three of the 80 lost consciousness (and these were all breathing air). The breath holding time was kept constant for each group (60 seconds) and thus we avoided Miles' error of including this as an uncontrolled, unassessed variable.

By this experiment we supported the suggestion that, if the breath holding time is kept constant, the incidence of syncope is not increased when breathing O₂. There was no support for the hypothesis that O₂ inhalation, per se, was related to syncope.
IV HYPOXIC SWB

The objective clinical sequelae of hypoxia include alterations of mood (euphoria, depression), of memory and intellect, of physical competence, and of consciousness (excitement, stupor, coma). Death may occur. Man is not endowed with any sensory perception of $O_2$ deficiency, and so has no emergency system to instigate appropriate avoidance reactions (such as with hypercapnia).

An increase in the functional dead space, noted with $CO_2$, tends to increase ventilation relative to the percentage of $CO_2$ of the rebreathed gas, and so offsets somewhat the possibility of hypoxia. Factors which may cause hypoxia, and SWB following from it, are:

(i) Incorrect breathing gases.
(ii) Shallow respirations with increased dead space.
(iii) Mechanical resistance to respiration.
(iv) Dilution hypoxia.
(v) Carbon monoxide poisoning.
(vi) Oxygen consumption exceeding supply.

(i) Incorrect Breathing Gases. Miles (reference 11) describes how 2 divers lost consciousness within a few minutes of entering the water, and how an experienced diving officer, using one of their sets, also lost consciousness within 8 minutes. After examination of the equipment it was found that the bottles had been incorrectly filled with air, instead of $O_2$. Unfortunately we are not informed whether gas analyses were done on the cylinders of any other divers, and possibly others in this series could have developed hypoxic SWB under similar conditions. This cause of hypoxic SWB is likely to have its effects within a few minutes of using the set, although this could be prolonged if 40% $O_2$ was used instead of 60% or 100% $O_2$.

It is axiomatic that incorrect breathing gases could result in hypoxic SWB.

(ii) Shallow Respiration with Increased Dead Space.

Theoretically if one has a low tidal volume and a rebreathing set with a large dead space (see Appendix D) then one could rebreathe from the dead space of the breathing tube and canister. This would allow the $CO_2$ to be partially absorbed, the $O_2$ to be utilised, and $N_2$ from both lungs and body to be the dominant tidal gas. The possibility of SWB being related to a shallow tidal respiration into a large dead space was supported by the discovery of Donald (reference 4, 5). He showed that subjects could become tolerant of high $CO_2$ tensions if breathing high pressure $O_2$. This made more feasible the belief that the rise of $CO_2$ need not stimulate the subjects to change from shallow to deeper respiration and to avoid the hypoxic SWB or hypercapnic SWB.
HYPOXIC S.W.B. (Cont.)

There are two major problems in accepting this explanation. Firstly it has little or no experimental evidence to demonstrate the "Shallow respiration" association with S.W.B., and secondly Donald's work has received little support from respiratory physiologists who accept increased inspired CO₂ as a definite respiratory stimulant (reference 9). Attempts were made to reproduce unconsciousness under these conditions. See Appendix C (Exp. D 2).

We could not obtain subjects who had enough control over their tidal volume, once the expired CO₂ started to rise. Thus we were unable to demonstrate the applicability of Donald's work (showing tolerance to high levels of CO₂ with O₂) to the use of this equipment. Until this applicability is demonstrated, and unconsciousness does occur under these conditions, it would seem unwise to use this explanation for S.W.B. If the subjects exercised, the likelihood of low tidal volume would be much less, irrespective of the arterial CO₂ (reference 9), and thus the tidal volume/dead space ratio would be much greater. Under even mild exercise conditions S.W.B. could not conceivably be due to this explanation. Hyperventilation with exercise exceeded that required to remove the extra CO₂ produced, (reference 9) and would offset this cause of S.W.B.

(iii) Resistance to Respiration.

If the equipment is such as to cause resistance to the flow of gas, or if the flow of gas is impeded by increased density, hypoxia may be produced. The latter is most unlikely, as at 30 feet the maximum breathing capacity is reduced only to 75% of normal. The possibility of the equipment producing a resistance to the respiration was considered, and is discussed in Appendix D. It is unlikely, per se, to be a cause of S.W.B.

(iv) Dilution Anoxia

This is aptly described by Miles (reference 12). Even though the diver may be breathing from a supply of 100% O₂, this is contaminated with N₂ in the counterlung by the following methods:

(a) Not adequately clearing the counterlung of air before use. This may leave a litre or more N₂ in the counterlung.

(b) The diver breathing from atmosphere prior to using the equipment i.e. not clearing his lungs with O₂. If he starts breathing from his set, from a position of inspiration, this could add another 3 litres of N₂.
HYPOXIC SWB. (Cont')

(c) Using $O_2$ "on demand" may result in the counterlung having $N_2$ (from (a) and (b)). This $N_2$ build up in the counterlung is intensified if $N_2/O_2$ mixtures are used.

(d) Approximately 1 litre of $N_2$ is dissolved in the body, and if one breathes from 100% $O_2$, the dissolved $N_2$ shall partly diffuse from body to lungs to counterlung.

It is difficult to understand how (d) could significantly contribute to anoxia. If there is one litre of $N_2$ dissolved in the body, and if one breathes from 100% $O_2$, the dissolved $N_2$ shall partly diffuse from body to lungs to counterlung.

It was considered prudent to ensure that the explanations given for (a) and (b) causing dilution anoxia, and therefore SWB were able to be supported. See Appendix C. Results demonstrated the validity of hypoxic SWB.

(v) Carbon Monoxide Toxicity

The combination of carbon monoxide with haemoglobin to form a stable compound (carboxy-haemoglobin) results in a reduction of the $O_2$ carrying capacity of the blood. Symptoms include decreased exercise tolerance, headache, irritability, impaired judgment and memory, confusion and unconsciousness. The time that this occurs will be directly related to the pressure and exposure time of carbon monoxide. Although this condition may occur with EEGA used from a petrol driven compressor, in which the intake is adjacent to the exhaust, it has been recorded in the use of EEGA when bottles with CO contamination are used to charge sets. It is rare when adequate precautions are taken, and when gas cylinders, both routinely and following incidents, are consistently checked for CO concentration. The specification in use by the RAN allows a maximum of 0.01%.

(vi) Increased Oxygen Consumption.

It is of interest that the flow of $O_2$ of the EEGA is usually quite inadequate for strenuous swimming with fins (reference 3). Thus, if the $O_2$ consumption is in excess of the $O_2$ supply, the counterlung is likely to contain $N_2$, as explained above or from the gas mixture supplied. This may remain unnoticed by the diver, who has no warning of hypoxia. CO$_2$ concentration may be kept low by the activity of the absorbent, especially in the early part of the dive when the absorbent is functioning at its maximum. Most flow rates of $O_2$ allow for a consumption of 1.5 litres per minute, the actual consumption can be double this.
The possibility of hypocapnesia following the hyperventilation of anxiety or panic has been used to explain the increased tendency of SWB among trainees using CABA. One could become involved in an assessment of the basic assumption (i.e. that SWB has a high incidence in trainees), because there seems no relevant controlled data available, to show this incidence.

If there is a significant causative relationship between hyperventilation and SWB, then the CABA (CABA or CABA) which allowed the greater hyperventilation would be the case most likely to be associated with SWB. On these grounds, it would be reasonable to assume that hyperventilation would be greater with the CABA than the CABA since SWB occurs only rarely with the CABA.

Appendix D describes the investigations carried out on each breathing apparatus. It was clearly demonstrated that hyperventilation was very likely with the CABA, and most unlikely with the CABA. As SWB is so rare with CABA it is difficult to see how one can incriminate hyperventilation as a likely cause.

In retrospect, when one considers:

1. the resistance and dead space of the CABA, as opposed to the CABA;
2. the increased tissue CO₂ tension when breathing high concentrations of O₂, and
3. the possible interference by oxygen with CO₂ sensitivity of the respiratory centre (reference 4),

it is difficult to understand how hypocapnesia could occur with the CABA. Response to hyperventilation as measured by the symptoms of paraesthesia (see Appendix D), was compared in the CABA and CABA.

The results were as follows:

<table>
<thead>
<tr>
<th></th>
<th>CABA</th>
<th>CCBA</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>Air</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>21</td>
<td>13</td>
</tr>
</tbody>
</table>

These supported the findings of Appendix D viz. hypocapnesia in subjects breathing O₂ from a CABA must be accepted with considerable scepticism. The effect of hyperventilation is more marked with CABA, and this contrasts with the rarity of SWB.

Despite these criticisms of the association of hyperventilation and SWB, there still remained the possibility that the mild hyperventilation which was possible using the CABA, in some way influenced the functioning of the equipment, or the CO₂ absorbent, so as to produce "syncope" or SWB.
This was tested as an hypothesis (see Appendix B). The result gave little support to it. The number of subjects with symptoms of syncope were:

<table>
<thead>
<tr>
<th></th>
<th>CABA</th>
<th>CCRA</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO₂</td>
<td>13</td>
<td>16</td>
</tr>
<tr>
<td>Air</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>31</td>
</tr>
</tbody>
</table>

These results are not significant and do not disagree with the Null hypothesis (that they both came from the same population). If they reflected an important syncope producing effect of hyperventilation with both sets, one would expect a much higher prevalence, with a slightly lower incidence of SWS with CABA.
VI  PHYSIOLOGICAL SEQUELAE OF ASCENT.

(i) Increased Intra Thoracic Pressure.

The mechanical resistance to breathing with the CGBA could be a contributory cause of SUB (see appendix D). It is not of such an order that it could, per se, result in diminished venous return and syncope.

Syncope has been noted by the author to occur in some subjects doing free ascents from 60 ft. It is recorded by the subjects as a "faint", "light headedness", "dopey" sensation as they approach the surface. As many divers under training tend to over distend their lungs, and are also likely to exhale too slowly as they ascend, they may well achieve physiological pressures of the order described by Liles in his "Oxygen Syncope" experiments (i.e. in excess of 50 mm Hg). It may well be that his work has more relevance to these cases than to SUB or syncope.

Presumably some divers with CGBA could develop syncope by ascending a few ft. (50 mm Hg) with a closed glottis - however as the same is likely to happen with CABA divers, it is not a cause to be considered likely. As well as ascending, one would have to keep the glottis closed for longer than a few seconds, for cerebral vascular insufficiency to occur. This does not seem any ethical way of testing this concept at shallow depths, as when this pressure gradient develops, pulmonary barotrauma is a possible complication.

(ii) Diminished Alveolar Oxygen Tension.

Schaefer (reference 8) has documented fully the lowering of alveolar oxygen pressures. This finding is partially explained by the rapid release of nitrogen during the change from higher to lower pressures. Although this condition may occur to some degree in divers with either CABA or CGBA, its main relevance is to skin divers using breathholding and hyperventilation techniques.
DISCUSSION

Unconsciousness in a shallow water diver may be due to:

(a) General clinico-pathological causes.
(b) Specific physiological responses to the hyperbaric environment.
(c) Occupation hazards from the equipment used.

(a) Clinico-pathological causes of unconsciousness, which are co-
incidental to the diving environment, must be considered. These
include cerebrovascular accidents, epilepsy, head injury, cardiac lesions
etc. The incidence of these diseases is probably no higher when diving
to 30 ft. than with any other activity, and so although they are
important for the individual differential diagnosis, they would not be
considered as occupational diseases.

(b) Specific physiological responses to the hyperbaric environment
encountered in the upper 30 ft. of water. The pressures of oxygen
and carbon dioxide are no greater in this upper layer, than they are in
the 30-300 ft. layer -- irrespective of the equipment used. Breathing
oxygen is equivalent to breathing air from 165 to 300 ft., as far as
the oxygen pressure is concerned. It is difficult then to understand
how the oxygen pressure could be more harmful in the upper layer.
Considering the difficulty in obtaining 100% \(O_2\) in the counterlung of the
CCBA even when using 100% \(O_2\) in the cylinders, then the incompatibility
of oxygen inducing SWB becomes obvious. The basis of the "Oxygen Syncope"
etiology has already been questioned.

Carbon dioxide toxicity would be enhanced at greater depths, where
\(CO_2\) levels in the tissues are increased -- so again we are faced with the
paradox of explaining why the blackouts occur in shallow water.

Resistance to respiration likewise will result in effects only
at greater pressures or depths.

Thus to explain SWB, it is difficult to incriminate physiological
factors due to the hyperbaric environment. Mechanical effects -- the
physiological sequelae of ascent -- are possible causes in some divers
using a breathing apparatus, and are likely causes in skin divers using
breath holding and hyperventilation techniques.

(c) Occupational hazards from the equipment used. The equipment
(CCBA) has associations which are consistent with the dominant clinical
characteristics noted in SWB viz; CCBA is usually used with 100% \(O_2\)
and so has an operational limitation of 30 ft. depth. This is also the
commonest depth for operations and training with this equipment. The
probable methods of producing SWB with this apparatus are:

(1) CCBA uses a rebreathing system and so hypoxia is possible, and
(2) CCBA has a \(CO_2\) absorbent, which may become functionally
inadequate and result in the hypercapnic syndrome.

Both these tendencies would be accentuated by exercise (utilising
more \(CO_2\) and producing more \(CO_2\)). The recorded association of exercise
with SWB (reference 1) is thus explained.
DISCUSSION (Cont.)

For these reasons, and on the basis of the above reports incriminating CSBA and supported by previous work (reference 1, 6, 11), it is believed that most cases of S.W.B are attributable to the hazards of this apparatus or its misuse. These may be given diagnostic categories of:

(i) S.W.B due to hypercapnea,

(ii) S.W.B due to hypoxia (which should be qualified by the reason for the hypoxia).
CONCLUSION

The etiological concepts of Shallow Water Blackout, Oxygen Syncope and Unconsciousness due to breathing apparatus, have been questioned.

Under the conditions of this review, and referring only to CABA and COBA equipment as defined in the glossary, the following conclusions were drawn:

1. The multifactorial (multiple cause) etiology is of minimal heuristic value.
2. Hypercapnic Shallow Water Blackout is an established entity.
3. The original work, suggesting the concept of "Oxygen Syncope" is questioned. Experiments designed to support it, failed to do so.
4. Hypoxic Shallow Water Blackout has many causes, and some of these were considered in this report.
5. Hypercapnic Shallow Water Blackout with divers using COBA, is refuted.
6. Shallow Water Blackout due to the physiological sequelae of ascent is left in doubt, pending further information.
REFERENCES

4. Donald K.W., 1947 "Oxygen Poisoning in Man" AEDU Report No. XVI.
APPENDICES

A. Statistical assessment of Miles' Results,
by P. Scully-Power & Carl Edmonds.

B. Modified Oxygen Syncope Test,
by Carl Edmonds.

C. O.C.S.A. Model Experiments,
by Daniel Quick & Carl Edmonds.

D. The Mechanical Performance of CCBÄ & CABA,
by J. Colebatch.
APPENDIX A.  MILES' OXYGEN SYNCOPE EXPERIMENTS.

The experiments were carried out on 36 young divers. Each subject breathed gas (either air or O₂) while horizontal on a tilt table. After 5 minutes he hyperventilated for 1 minute and was then tilted quickly to an upright position. He then exchanged the mouthpiece for a tube attached to a mercury manometer, and blew a column of mercury as high as he could, for as long as he could.

The results which Miles stressed were:

<table>
<thead>
<tr>
<th></th>
<th>Air (No)</th>
<th>Oxygen (No)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of consciousness</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Evidence of Symptoms</td>
<td>2</td>
<td>14</td>
</tr>
</tbody>
</table>

Results which he did not use in his discussion, but which were recorded, support the knowledge that subjects after breathing O₂ can hold their breath longer than after breathing air (reference 9).

i.e.

<table>
<thead>
<tr>
<th></th>
<th>Air</th>
<th>O₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean breath holding time (secs)</td>
<td>63.4</td>
<td>82.4</td>
</tr>
<tr>
<td>Mean pressure held (mm Hg)</td>
<td>52.9</td>
<td>50.0</td>
</tr>
</tbody>
</table>

It was also noted that 2 patients who lost consciousness on both air and O₂ were removed from the trial after its completion because, in retrospect, it was elicited that they had had a heavy meal and 3 pints of beer prior to the tests. There was no suggestion that all the other subjects underwent the same retrospective enquiry, and even had they done so, removing results after an experiment is not a statistically sound manoeuvre.

The correct results are therefore:

<table>
<thead>
<tr>
<th></th>
<th>Air</th>
<th>Oxygen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of consciousness</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>Incidence of Symptoms</td>
<td>2</td>
<td>14</td>
</tr>
</tbody>
</table>

A statistical assessment of the results do not support Miles conclusions that O₂ breathing and syncope are related.

Statistical Evaluation of Miles' Experiments.

1. Consider two samples of AIR and OXYGEN. Consider the Null Hypothesis that they could have come from the same population.
VI. Describing the similarity of two gases, O2 and Air:

<table>
<thead>
<tr>
<th></th>
<th>Syncope</th>
<th>No Syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>5</td>
<td>31</td>
</tr>
<tr>
<td>O2</td>
<td>9</td>
<td>27</td>
</tr>
</tbody>
</table>

Applying the Yates correction:

<table>
<thead>
<tr>
<th></th>
<th>Syncope</th>
<th>No Syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>5½</td>
<td>30½</td>
</tr>
<tr>
<td>O2</td>
<td>8½</td>
<td>27½</td>
</tr>
</tbody>
</table>

Then using equation \( \chi^2 = \frac{(O-E)^2}{E} \)

We have \( \chi^2 = 1.12 \)

With one degree of freedom, the probability (P) that, in random sampling, the value of this statistic will occur greater than the value actually obtained is:

\( P = 0.3 \)

We therefore have no grounds to reject the Null Hypothesis.

2. Despite these arguments, it is still possible that the degree of O2 in the two gases (Oxygen = 100%, Air = 20%) may be significantly related to the degree of syncope. This possibility was tested as follows:

Consider a population defined by the results obtained for Air (20% O2).

Consider the hypothesis that the occurrence of syncope is directly proportional to the amount of O2.

Then the results for Oxygen are:

<table>
<thead>
<tr>
<th></th>
<th>Syncope</th>
<th>No Syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expected</td>
<td>25</td>
<td>11</td>
</tr>
<tr>
<td>Observed</td>
<td>9</td>
<td>27</td>
</tr>
</tbody>
</table>

Then using the equation \( \chi^2 = \frac{(O-E)^2}{E} \)

We have \( \chi^2 = 33.51 \)

With one degree of freedom, the probability (P) is less than 0.01. Since this probability is extremely small, the value of the expected statistic is significantly large and we have good grounds to reject the hypothesis.
APPENDIX A. (Cont.)

3. There is, however, one factor which seems to have been ignored by Miles – the extent of impairment of venous return during increased intrathoracic pressure. Oxygen breathing allows for longer breath holding than air breathing (reference 10), and this is supported by Miles' results. Longer breath holding results in a greater impairment of venous return to the heart. It is possible, and predictable, that the group who can hold their breath longer, will have the greater tendency to reduced cardiac output following inadequate venous return.

Putting this hypothesis to the statistical test, we get our first encouraging hint as to the cause of the syncope.

Consider a population defined by the results obtained for AIR (mean breath holding time 63.4 secs).

Consider the hypothesis that the occurrence of syncope is directly proportional to the breath holding time.

The results for OXYGEN (mean breath holding time 82.4 secs)

<table>
<thead>
<tr>
<th>Syncope</th>
<th>No Syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expected</td>
<td>6.5</td>
</tr>
<tr>
<td>Observed</td>
<td>9</td>
</tr>
</tbody>
</table>

Then the results for OXYGEN (mean breath holding time 82.4 secs)

Then using equation $\chi^2 = \frac{(O-E)^2}{E}$

We have, $\chi^2 = 1.17$

In this case of one degree of freedom, the probability (P) is given by

$P = 0.3$

We therefore have no grounds to reject this hypothesis.
APPENDIX B. MODIFIED CABOT SYNCOPE EXPERIMENTS.

An "oxygen syncope test" was modified from that of Miles (reference 10). Twenty subjects were used from the Diving School of HUSS RUBCON. Each subject was given a schedule to allow him to undergo a set order of tests. The schedules were:

<table>
<thead>
<tr>
<th>5 Subjects</th>
<th>5 Subjects</th>
<th>5 Subjects</th>
<th>5 Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>CCBA O₂</td>
<td>CCBA Air</td>
<td>CCBA Air</td>
<td>CCBA Air</td>
</tr>
<tr>
<td>CCBA Air</td>
<td>CCBA O₂</td>
<td>CCBA O₂</td>
<td>CCBA Air</td>
</tr>
<tr>
<td>CCBA O₂</td>
<td>CCBA Air</td>
<td>CCBA O₂</td>
<td>CCBA O₂</td>
</tr>
</tbody>
</table>

The equipment was not attached to the patient, but was set up on an adjacent table.

The subject would lie in a horizontal position and hyperventilate, as per his schedule, for 5 minutes.

He would then inhale fully, stand upright, and hold his breath for 60 seconds. He would then report whether he had noticed any symptoms. If there was any confusion in his reply (e.g., when he claims he feels "faint") he was asked by his attendant whether he noted (a) unsteadiness or (b) numbness or tingling feelings. Unsteadiness was usually obvious to the attendant, who often had to steady the subject, and was taken to be an early feature of syncope. Collapse with unconsciousness was also recorded. Parasthesia was taken to indicate the effects of hyperventilation induced hypocapnea.

The hypotheses being tested were:

1. O₂ breathing would produce more symptoms of syncope - unsteadiness and unconsciousness - than air breathing.

This hypothesis, if verified, would demonstrate the importance of O₂ as a cause of S-B, if the S-B could be equated with "syncope".

2. Hyperventilation would be easier with the CCBA, and thus would show an increased incidence of parasthesia, and also of syncope.

3. Syncope was in some way related to the mechanics of the CCBA sets when the subject was hyperventilating.
Subject Unsteadiness Paraesthesia Unconsciousness  
No.  \( U \) \( P \)  \( U \) \( C \) \( F \)  \( U \) \( C \) \( P \)  \( U \) \( C \) \( P \)  
1  u  -  u  -  u  -  u  -  
2  u  -  -  -  u  -  u  -  
3  -  +  u  -  uc  -  u  -  
4  -  +  -  +  u  -  u  -  
5  -  +  u  +  u  +  u  +  
6  -  +  -  +  -  -  -  -  
7  u  +  u  +  u  -  -  -  
8  -  -  u  -  uc  +  u  -  
9  -  -  -  -  -  -  -  -  
10  u  -  -  -  u  +  -  -  
11  -  +  u  -  -  +  u  -  
12  u  -  u  +  u  -  u  -  
13  u  -  u  +  u  -  u  -  
14  u  -  u  +  u  +  u  -  
15  u  -  u  -  u  -  u  -  
16  u  -  -  -  -  -  -  
17  u  -  u  -  u  -  u  -  
18  u  +  -  +  u  -  u  +  
19  u  +  u  +  u  +  u  +  
20  u  +  u  +  u  +  u  +  
Total  13  10  16  10  15  3  

1st Hypothesis.
To support Oxygen Syncope. The numbers exhibiting unsteadiness were:

<table>
<thead>
<tr>
<th></th>
<th>Air</th>
<th>( O_2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>CABA</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>CCBA</td>
<td>16</td>
<td>15</td>
</tr>
<tr>
<td>Totals</td>
<td>29</td>
<td>28</td>
</tr>
</tbody>
</table>

\( \chi^2 = 0.01471 \)

Results not significant.
Collapses and unconsciousness occurred in 3 cases, all breathing air - 2 on CCBA and one on CABA.

CONCLUSION

There was no evidence to support the hypothesis that O₂ had a syncope producing effect, from either CCBA or CABA.

2nd Hypothesis.

Hyperventilation more likely with CCBA. The number of subjects with paraesthesia were:

<table>
<thead>
<tr>
<th></th>
<th>CABA</th>
<th>CCBA</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>Air</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>21</td>
<td>13</td>
</tr>
</tbody>
</table>

\[ \chi^2 = 2.64787 \text{ (i.e. not significant)} \]

CONCLUSION

The hypothesis had no support from these experiments. The opposite was noted i.e. hyperventilation symptoms were more likely with CABA sets. They were also more likely with air than O₂, and this may be related to reduced CO₂ transport from the tissue with O₂ breathing (ref. 9). The combined protection of O₂ and the CCBA made hyperventilation effects most unlikely.

3rd Hypothesis.

Syncope is more likely with the CCBA than CABA under these conditions:

The incidence of unsteadiness was as follows:

<table>
<thead>
<tr>
<th></th>
<th>CABA</th>
<th>CCBA</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂</td>
<td>13</td>
<td>16</td>
</tr>
<tr>
<td>Air</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>31</td>
</tr>
</tbody>
</table>

CONCLUSION

The probability of these results being due to chance, makes it unlikely that this type of experiment will be of any use in determining which characteristic of the CCBA makes it related to SWB.
APPENDIX C.

Experiment D. (2).

To test the possibility of hypoxia due to shallow respirations, it was decided to attempt to produce unconsciousness by having subjects respire from a small plastic bag, of fixed volume, through a CO₂ absorbent, tube and mouthpiece — of the same type as in the C3A. An O₂ supply was connected to the plastic bag, to top up with O₂, if required following the CO₂ absorption. This use of O₂, on demand, varied the gas measurements in an irregular manner, but was required by most subjects. See figure I.

The plastic bag was distended with O₂, which was drawn through the tube and mouthpiece, and then exhaled to the atmosphere once, through the mouthpiece cock. The subjects then attempted to breath from the plastic bag, via the rebreathing system. They were told to breath as often as they wished, but the tidal gas was limited to approximately 350 mls.

Under these conditions the experiment was never completed. Dyspnoea occurred with the subjects attempting to increase the tidal volume and being obstructed in these attempts by the limitation of the plastic bag. The hypoxia occurred after approximately 5 minutes, coinciding with a rise of CO₂ in the expired gas. Subjects could tolerate this restriction of their tidal volume for approximately 15 minutes.

<table>
<thead>
<tr>
<th>Min.</th>
<th>Inspiration</th>
<th>Expiration (ex Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>O₂</td>
<td>CO₂</td>
</tr>
<tr>
<td>5</td>
<td>515</td>
<td>&lt;10</td>
</tr>
<tr>
<td>10</td>
<td>440</td>
<td>&lt;10</td>
</tr>
<tr>
<td>15</td>
<td>585</td>
<td>&lt;10</td>
</tr>
<tr>
<td>20</td>
<td>580</td>
<td>&lt;10</td>
</tr>
<tr>
<td>23</td>
<td>525</td>
<td>&lt;10</td>
</tr>
</tbody>
</table>

Under these conditions the O₂ from the bag would fill the mechanical dead space of the equipment (approx. 150 mls) before it mixed with the functional dead space and exchangeable gases in the lungs. We failed to produce unconsciousness due to shallow respirations.

Experiment D (iv).

Using the same Canister/breathing tube/mouthpiece as in figure I, but with a larger plastic bag (volume 1.5 litres), the bag was filled with O₂ and was allowed to be topped up by the subject as required. There was no instruction given re clearing the lungs with O₂.
APPENDIX C. (Cont.)

The subjects became cyanotic and lost consciousness, under these conditions (in practice the time would depend on the size of the bag and the amount of $\text{CO}_2$ added to replace the $\text{CO}_2$ absorbed).

\(\text{a). (a) Inspiration - Aspiration}\)

\begin{tabular}{|c|c|c|c|}
\hline
\text{Mins.} & \text{$\text{O}_2$} & \text{$\text{CO}_2$} & \text{Mixed $\text{CO}_2$} \text{mm Hg} \\
\hline
5 & 375 < 10 & 230 & 45.5 \\
10 & 195 < 10 & 160 & 48.0 \\
15 & 190 < 10 & 120 & 25.0 \\
20 & mixed gas $\text{O}_2$ = 50 mm Hg & Subject lost consciousness, appeared $\text{CO}_2$ = 27.5 mm Hg; cyanotic. No warning symptoms noted. \\
\hline
\end{tabular}

\(\text{b) Inspired - Expired}\)

\begin{tabular}{|c|c|c|c|}
\hline
\text{Mins.} & \text{$\text{O}_2$} & \text{$\text{CO}_2$} & \text{Mixed $\text{CO}_2$} \text{mm Hg} \\
\hline
5 & 555 < 10 & 355 & 31 \\
10 & 355 < 10 & 235 & 27 \\
15 & 185 < 10 & 135 & 29 \\
20 & 95 < 10 & 30 & 27 \\
\hline
\end{tabular}

Increase resp. rate.
Subject noticed a ringing in the ears, dizzy, dreaming. He became cyanotic and lost consciousness at 22 minutes.

\text{Inspiration}

\text{22 Tube air Bag air}

60 < 10 105 16 mm Hg

\text{N.B. The tube contained gas derived from the bag and the canister (expired air with $\text{O}_2$ removed). This explains how the "dead space" of the canister may contribute to hypoxic $\text{CO}_2$.}

Using the U.M. similar results occurred when the counterlung was squeezed empty and the set placed on, with $\text{O}_2$ used by demand.

\(\text{b) Inspired - Expired}\)

\begin{tabular}{|c|c|c|c|}
\hline
\text{Mins.} & \text{$\text{O}_2$} & \text{$\text{CO}_2$} & \text{Mixed $\text{CO}_2$} \text{mm Hg} \\
\hline
5 & 210 < 10 & 345 & 45 \\
10 & 300 < 10 & 130 & 46 \\
15 & 205 < 10 & 45 & 38 \\
20 & 105 < 10 & 45 & 32 \\
\hline
\end{tabular}

Increased respirations noted.

The patient became cyanotic and stuporous at 24 minutes. U.M. had to be removed rapidly.
APPENDIX C. (Cont.)

Controls were carried out, using a wash-out of the lungs by 2 maximal inhalations of $O_2$ from the counterlung and exhaled to the atmosphere (i.e. the lungs were cleared of a large quantity of $N_2$, but the $N_2$ content of the body was not significantly influenced): $O_2$ was still used on demand.

Results showed a small drop in $O_2$ pressures, but this was not clinically significant.

<table>
<thead>
<tr>
<th>e.g. Time (mins)</th>
<th>Inspired $O_2$ (mm Hg)</th>
<th>Inspired CO$_2$</th>
<th>Expired $O_2$ (mm Hg)</th>
<th>Expired CO$_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>695 &lt; 10</td>
<td>680</td>
<td>34</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>705 &lt; 10</td>
<td>670</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>680 &lt; 10</td>
<td>640</td>
<td>34</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>565 &lt; 10</td>
<td>525</td>
<td>39</td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>555 &lt; 10</td>
<td>510</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>431 &lt; 10</td>
<td>415</td>
<td>38</td>
<td></td>
</tr>
<tr>
<td>70</td>
<td>420 &lt; 10</td>
<td>360</td>
<td>41</td>
<td></td>
</tr>
</tbody>
</table>

The above is considered adequate evidence to support the concept of dilution anoxia as a cause of SIDS.
APPENDIX D.

The Mechanical Performance of Breathing Aids Used with Diving.

Occasional episodes of unconsciousness during diving have been reported when the closed circuit apparatus (C.C.A.) was used. These episodes have occurred some time after commencing the dive, but not when the open circuit apparatus (O.A.B.A.) was used. The mechanical performance of these two types of breathing aid have been studied to determine how they might influence breathing and the state of consciousness.

Methods.

Mouth pressure was measured with an Elema Schonander pressure transducer EMT 35, gas flow rate with a Pfeizsch pneumotachometer No. 2 and an Elema Schonander transducer EMT 32, and tidal volume by electrical integration of the gas flow rate signal. The integrator had an effective time constant of more than 10,000 seconds, allowing an integrator error of less than 1%. After amplification the mouth pressure, gas flow rate and tidal volume were recorded on a Mingograph 34 Jet recorder. The breathing aids were tested with the same subject in each case.

Results and Discussion.

Open Circuit Apparatus.

With the first type of apparatus (C.I.B.A.), gas is supplied from a high pressure reservoir cylinder and inspiration is activated when mouth pressure is reduced 6 cm of water below ambient pressure (Fig. 2). For comparison a control record breathing room air is shown in Figure 1. The expiratory valve of the C.I.B.A. provides a relatively small resistance to gas flow of 3.6 cm H$_2$O/L/sec (Table 1). Tidal volume and total ventilation were increased during breathing with the C.I.B.A.

During voluntary hyperventilation total ventilation was more than 100 L/min, and inspiratory gas flow reached a level in excess of 300 L/min (fig. 3). After triggering gas flow, inspiratory resistance was negligible. During expiration, mouth pressure reached 16 cm water, but expiratory resistance was 4.8 cm H$_2$O/L/sec only a little greater than during quiet breathing.

These results show that the C.I.B.A. open circuit apparatus imposes little impediment to breathing. It would permit ventilation to be increased to any level likely to be required during heavy work. In addition the apparatus has the advantage of a low dead space. The cause of the increased total ventilation during quiet breathing was not clear from these studies. Ventilation may have increased because of the relatively high inspiratory flow rate that followed opening the inspiratory valve.
APPENDIX D. (Cont.)

Closed Circuit Apparatus (C.C.A.)

With the closed circuit apparatus the subject rebreathes through a sodalime canister to a gas reservoir or "counterlung". This system imposes an increased dead space estimated to be about 150 - 200 ml, that is, approximately equal to that already present in the respiratory system. During quiet breathing the gas reservoir exerted a pressure of 5-8 cm of water depending on the level of inflation (Fig. 4). During inspiration, mouth pressure decreased to the ambient level, increasing again during expiration. Inspiratory resistance for the subject was therefore negligible. In expiration resistance to gas flow was about three times that present in the normal lung and airways. In addition, an elastic resistance was imposed by the gas reservoir of about 1 cm of water per 100 ml tidal volume. This elastic resistance is approximately equal to that of the lung and chest wall together.

During quiet breathing, total ventilation was increased compared to breathing without the apparatus. The increase in ventilation was due to an increase in tidal volume which was approximately equal to the added dead space of about 200 ml. These measurements were made about 1 minute after starting breathing with the closed circuit apparatus when tidal volume appeared to be at a steady level. However, it is impossible to establish whether a steady state of gas exchange had been achieved.

Results obtained during hyperventilation show mainly the effects of a high resistance to gas flow both in inspiration and expiration (Fig. 5) and (Table 2). The mouth pressure change from inspiration to expiration reached 50 cm of water and is nearly in phase with gas flow. This high mouth pressure is due to a relatively high dynamic resistance both in inspiration and in expiration. The measured resistance was equivalent to breathing through a tube with an internal diameter of 9 mm. During hyperventilation elastic resistance appeared to be less than during quiet breathing.

During voluntary hyperventilation, total ventilation reached 66 L/min, which is considerably less than that found when using the open circuit apparatus. On the other hand, work done by the subject on the equipment to achieve increased ventilation was more than four times greater in the case of the closed circuit apparatus. The lower total ventilation achieved with the closed circuit C.C.A. is consistent with the fact that maximum ventilation is limited to a greater extent by inspiratory resistance than by an equivalent expiratory resistance (1).

Conclusions.

The results make it unlikely that hyperventilation due to anxiety could be the cause of unconsciousness (syncpe) during diving. If this were the case such a problem would occur with the open circuit (O.C.A.) rather than with the closed circuit apparatus. Besides, vasovagal syncpe is virtually excluded because of the absence of gravitational effects on the circulation when immersed.
The outstanding differences between these two types of breathing aid are the higher resistance, higher dead space and high oxygen concentration in the closed circuit equipment. Increase in effective ventilation with the closed circuit C.C.B.A. is limited both by dead space and resistance to gas flow. Furthermore increased inspiratory resistance, besides limiting maximum ventilation, also affects the control of breathing by diminishing the ventilatory response to arterial CO$_2$ tension (2). An increase in CO$_2$ tension might be expected when using the closed circuit C.C.B.A.

A reduction in effective ventilation would also occur with the closed circuit C.C.B.A. if the tidal volume did not increase in proportion to the added dead space. During quiet breathing with this breathing aid, increase in elastic resistance provided by the resistance of the gas reservoir to distention predominated over dynamic resistance. The normal effect of added elastic resistance is to cause a reduction in tidal volume (3). In some subjects this effect may cause difficulty in adjusting to breathing with the apparatus.

Analysis of the finding therefore leads to the conclusion that a likely cause of difficulty in using the closed circuit C.C.B.A. relate to an inadequate ventilation. The attempt to increase ventilation is opposed by the resistance to gas flow especially at high levels of ventilation. The sensation of difficulty with breathing which would result may lead to marked anxiety, whether this results in unconsciousness is not known.

A possible cause of unconsciousness which must be considered in this situation is the effect of oxygen at increased pressure on the central nervous system. Oxygen toxicity is augmented by moderate increases in arterial CO$_2$ tension (4). Tolerance of high oxygen is also said to be reduced by exercise and water immersion.
APPENDIX D. (Contd.)

Suggested Modifications to the Closed Circuit U.B.A.

The most important modifications are to reduce dead space and inspiratory resistance; expiratory resistance is less important. This could be achieved by connecting a separate inspiratory tube to the gas reservoir thereby by-passing the CO₂ absorber; a one-way valve or valves would be required to direct expiration through the soda lime. As this modification would overcome major disadvantages of this breathing aid as at present used, while retaining the closed circuit, I recommend that it be given serious consideration.

(H.J.M. Collebatch) M.R.A.C.P.
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MAY 1966.
### TABLE 1. (Appendix D.)

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>C.A.B.A.</th>
<th>C.A.B.A. Hyperventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tidal Volume</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>litres</td>
<td>615</td>
<td>.904</td>
<td>2.950</td>
</tr>
<tr>
<td><strong>Breaths/min</strong></td>
<td>14.8</td>
<td>14.5</td>
<td>35</td>
</tr>
<tr>
<td><strong>Total ventilation</strong></td>
<td>9.1</td>
<td>13.1</td>
<td>104</td>
</tr>
<tr>
<td>litre/min BTPS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Inspiratory resistance</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>on H$_2$O/L/sec</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Expiratory resistance</strong></td>
<td>-</td>
<td>3.8</td>
<td>4.8</td>
</tr>
<tr>
<td>on H$_2$O/L/sec</td>
<td></td>
<td>at 25 L/min</td>
<td>at 200 L/min</td>
</tr>
<tr>
<td><strong>Elastic resistance</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
### TABLE 2 (Appendix D)

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>C.C.S.A.</th>
<th>C.C.S.A. Hyperventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tidal volume</strong>&lt;br&gt;litres</td>
<td>3.160</td>
<td>3.330</td>
<td>3.160</td>
</tr>
<tr>
<td><strong>Breaths/min</strong></td>
<td>14.2</td>
<td>14.7</td>
<td>21</td>
</tr>
<tr>
<td><strong>Total ventilation</strong>&lt;br&gt;litres/min BTPS</td>
<td>8.66</td>
<td>12.2</td>
<td>66.4</td>
</tr>
<tr>
<td><strong>Inspiratory resistance</strong>&lt;br&gt;on H₂O/L/sec</td>
<td>-</td>
<td>0</td>
<td>7.8</td>
</tr>
<tr>
<td><strong>Expiratory resistance</strong>&lt;br&gt;on H₂O/L/sec</td>
<td>-</td>
<td>6.0</td>
<td>10.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>at 50 L/min</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>at 170 L/min</td>
</tr>
<tr>
<td><strong>Elastic resistance</strong>&lt;br&gt;on H₂O/L</td>
<td>-</td>
<td>10.6</td>
<td>5.5</td>
</tr>
</tbody>
</table>
REFERENCES TO APPENDIX D.


ACKNOWLEDGMENTS.

1. To Ship's divers HMAS RUSHCUTTER, for assisting in the experiments.

2. To ROYAL J. Manley and the staff of the School of Underwater Medicine, for organising the experiments and assisting in this project.

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Dan Quick, Technical Officer, School of Underwater Medicine, H.M.A.S. PENGUIN.
Figure 1: Breathing room air through pneumotachometer at rest. Mouth pressure remains at the ambient level.

APPENDIX D.
Figure 2: Quiet breathing with open circuit apparatus (C.A.B.A.). Inspiration is initiated by reducing mouth pressure 6 cm H₂O below the ambient level. Inspiratory gas flow reaches a higher level than during control state in Fig. 1.

APPENDIX D.
Figure 1: Hyperventilation with O₂, N₂, A. The upper curve shows the percentage of N₂ before and after the hyperventilation period. After hyperventilation, the curve shows a pressure during inspiration in contrast to an increase.
Figure 4: Quiet breathing with closed circuit apparatus (CCS). At end expiration mouth pressure is 6 - 7 cm H₂O and decreases during inspiration.
Figure 2: Hyperventilation with C.C.B.A. Mouth pressure is nearly in phase with gas flow and the mouth pressure swing is greatly increased due to a high resistance to gas flow both in inspiration and in expiration.

APPENDIX B.