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THE EFFECTS OF HYPERCAPNIA ON COGNITIVE AND PSYCHOMOTOR PERFORMANCE IN THE HYPERBARIC ENVIRONMENT

by

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B.Sc.(Hons), Liverpool Polytechnic, 1985

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ABSTRACT

Although the impact of high partial pressures of CO_2 on diving performance and safety has been recognized for some time, few studies have obtained quantitative data concerning the interactions of CO_2 and compressed air narcosis. This study, examines the N_2 and CO_2 components of compressed air narcosis by comparing the effects of a range of end-tidal PCO_2 tensions in air at 1 and 6 ATA. At each pressure, twelve healthy male volunteers completed a number of cognitive and psychomotor tests under normocapnic and hypercapnic conditions. Carbon dioxide levels were regulated by a rebreathing circuit so that $P_{ET}CO_2$ tensions fell within the ranges 30-35 mmHg, 45-50 mmHg and 55-60 mmHg. Cognitive and psychomotor performances were examined on a variety of tasks including: the Stroop test, an arithmetic test, a paired association memory test (immediate and delayed recall), number comparison, a copying test and the Purdue pegboard test. Performance on all tasks demonstrated significant ($p<0.05$) decrements at 6 ATA. Cognitive and psychomotor performance further deteriorated at depth when $P_{ET}CO_2$ tensions rose above 47 mmHg ($p<0.05$), however, no global threshold for the onset of CO_2 narcosis was indicated by the test performance scores. High PCO_2 tensions ($P_{ET}CO_2$ >47 mmHg) also significantly impaired ($p<0.05$) cognitive and psychomotor performance at surface pressure. No statistically significant nitrogen-carbon dioxide interaction was found on the performance tests ($p>0.05$). The pattern of intratest results, were however, quite different for N_2 and CO_2. At high $P_{ET}CO_2$ tensions, performance deficits were due to a retardation of performance rather than a disruption of the accuracy of processing. Nitrogen narcosis, conversely, produced significant impairment through both decreases in accuracy and slowed processing. There was also evidence suggesting that neural structures, which support memory and decision-making, show greater functional impairment under compressed air narcosis than those supporting visual perception and fine manual dexterity. It was concluded that within the $P_{ET}CO_2$ ranges studied: (1) PCO_2 pressures are additive in their effects on impaired cognitive and psychomotor performance at depth; (2) the underlying physiological mechanism of CO_2 narcosis is different from that for N_2 narcosis; (3) the degree of performance impairment induced by elevated PCO_2 tensions is dependent on the end-tidal CO_2 partial pressure and the sensitivity of the particular task, or cognitive factor, to hypercapnic stress. The N_2 performance data were explained according to the slowed processing model of narcosis and this model was further developed, through the concept of arousal, to account for the performance decrements under CO_2 narcosis.

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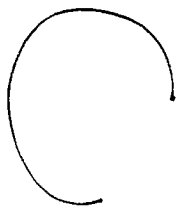
I would finaly like to thank all my subjects who kindly donated their time by willingly experiencing the pleasures and discomforts of compressed air narcosis and hypercapnia.

DEDICATION

In fondest memory

of

Thomas & Maggie Fothergill



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CHAPTER 1

INTRODUCTION

The importance of understanding man's working limitations in foreign environments has gathered momentum with technological progress over recent years. Frequent space flights by the Soviet Union and the United States have provided valuable physiological data on the effects of prolonged exposure to "outer space" (Yegorov, 1979; Johnson & Dietlein, 1977). Although dangers facing the astronaut are widely recognized due to the high profile and publicity of space missions, little credit has been given to the original research that enabled man to endure this harsh environment. Prior to manned space flight most of the data on human exposure to alien environments was derived from hyperbaric research where the hazards to which the diver is exposed are very similar to those encountered by the astronaut in space.

As yet, however, there are serious limitations which prevent man working beyond 7 ATA breathing compressed air, and 69 ATA breathing a gas mixture of He, N_2 and O_2. The primary objective of the present thesis was therefore to investigate one of the limitations of divers under the former condition, namely the effect of hypercapnia on cognitive and psychomotor performance.

When humans are exposed to greatly increased air pressures, as are found in the working environment of tunnel workers and divers, symptoms of euphoria, intoxication and narcosis are commonly exhibited. The confusion, euphoria and neuromuscular incoordination induced by compressed air narcosis can significantly reduce performance and lead to major concerns for these workers' safety.

Unfortunately the causes and mechanisms of compressed air narcosis are unclear. Past theories have suggested factors such as increased partial pressures of O_2 and N_2, CO_2 retention, pressure per se and the involvement of anxiety and claustrophobia (Bennett, 1966). Today, the majority of investigators view increased nitrogen pressure as the principal cause of the narcosis, and agree that increased O_2 pressure, CO_2 retention and other factors contribute to the narcosis either directly or indirectly by interacting with or modifying the N_2 effect (Hesser *et al.* 1978; Fowler *et al.* 1985).

The relative importance of high partial pressures of CO_2 on diving performance and safety has been recognized for some time. In 1939, Behnke and Willmon observed mental disturbances in divers working at 8.3 ATA (240 fsw) in the salvage of U.S.S. *Squalus*. These authors reported that the symptoms were of unusually high intensity and concluded that, at this depth, the accumulation of CO_2 in the divers' helmets may have augmented the narcotic action of nitrogen.

More recently, evidence of CO_2 accumulation has been obtained by an underwater gas sampler in divers using open-circuit breathing systems (Dwyer, 1977; MacDonald & Pilmanis, 1982) and end-tidal values of CO_2 partial pressure ($P_{ET}CO_2$) above 70 Torr have been found in experimental dives (Lanphier 1956, 1963; Morrison *et al.* 1981). Unfortunately, it is only recently that a number of experiments have been conducted in an attempt to evaluate quantitatively the role of CO_2 in compressed air narcosis.

1.1 OBJECTIVES

1. To determine quantitatively the effects of various levels of hypercapnia on cognitive and psychomotor performance in the hyperbaric environment.
2. To formulate a relationship between CO_2 and N_2 narcosis according to cognitive and psychomotor changes under varying levels of P_ICO_2 and P_IN_2.
3. To expand the current knowledge and data on human behavioural responses to hypercapnia at increased ambient pressure with reference to points 1 and 2.

1.2 ABBREVIATIONS AND DEFINITIONS OF TERMS

1. **Attempted score:** The number of problems or test units attempted on a performance test.
2. **Correct score:** The number of problems or test units correctly completed on a performance test.
3. **Error score:** The number of incorrect answers to problems or test units exhibited on a performance test.
4. **Immediate recall (IR):** The capacity to remember a series of word/number association pairs in the minute following presentation of the word/number list.
5. **Delayed recall (DR):** The capacity to remember a series of word/number association pairs, 13 minutes following the initial presentation of the word/number list.
6. **Additivity:** Used with reference to a factor that produces a combined effect with narcosis equal to the separate effects of narcosis and the factor.
7. **Synergy:** Used with reference to a factor that produces a combined effect with narcosis proportionately greater than the separate effects of narcosis and the factor.
8. **Potentiation:** Used with reference to a factor that is postulated to cause further impairments of narcosis through a common mechanism.

Symbols and abbreviations for pulmonary variables used within this thesis were based on the recommendations of Pappenheimer (1950) and Comroe *et al.* (1962).

CHAPTER 2

LITERATURE REVIEW

2.0.1 Assessment of the Narcotic Effects of High Pressure Gases

As the objective of this thesis was to quantify the effects of hypercapnia on cognitive and psychomotor performance, a review of the approaches to performance assessment in hyperbaric research is given below. This review does not attempt to reiterate the procedures and findings of individual studies, for which recent comprehensive reviews can be found elsewhere (see Bennett, 1982; Shilling, 1983; Biersner, 1985; Fowler *et al.* 1985). The review does, however, reflect on a number of approaches and problems associated with assessment and comparison of narcotic effects in the hyperbaric literature.

Many different tests have been developed and used to assess the narcotic action of high pressure N_2 on various aspects of human performance. Depending on the approach to the analysis of narcosis, performance decrements have been catagorized by their effects on different processes of behaviour. The different approaches have been outlined by Fowler *et al.* (1985) and include a descriptive model, the hierarchial organization hypothesis, the operant paradigm and the slowed processing model. The operant paradigm approach has been used solely as a technique for studying narcosis in animals. Although this technique may be useful in hyperbaric research where safety aspects exclude the use of human subjects, no further comment on this technique will be made.

The most common approach to the assessment of narcotic effects has been the descriptive model. The early dominance of this approach may be attributed to research conducted by physiologists or medical personnel who viewed narcosis primarily as a set of clinical signs and symptoms. This is illustrated by the classic description of narcosis which was characterised by a "euphoria, retardment of the higher mental processes and impaired neuromuscular coordination" (Behnke *et al.* 1935). Using this approach, Shilling *et al.* (1977) exemplifies a modern version of the descriptive model in terms of narcosis's differential effect on three categories of behaviour referred to as cognitive, reaction time, and dexterity, respectively. Performance tests used to explore cognitive function have included mental arithmetic (Moeller *et al.* 1981; Moeller and Chattin, 1975; Fothergill and Hedges, 1987; Schmidt *et al.* 1974; Bennett *et al.* 1969; Hesser *et al.* 1978), conceptual reasoning or spacial logic (Kiessling and Maag, 1962), sentence comprehension (Vaernes and Darragh, 1982), continuous freeword association (Adolfson and Muren, 1965), as well as, many different tests of short term memory (Moeller *et al.* 1981; Biersner *et al.* 1977; Fowler and Ackles, 1975).

The reaction time category has been defined in terms of one and two choice reaction time (Frankenhaeuser *et al.* 1963; Shilling and Willgrube, 1937; Kiessling and Maag, 1962; Moeller *et al.* 1981, Whitaker and Findley, 1977), card sorting (Bennett *et al.* 1967) and digit cancellation (Fothergill and Hedges, 1987).

Dexterity, or neuromuscular coordination, as defined in the early classic description of narcosis, has been determined from performance on the Purdue pegboard (Fothergill and Hedges, 1987; Kiessling and Maag, 1962), the Bennett hand tool dexterity test (Moeller *et al.* 1981; Schmitt *et al.* 1974), the screwplate test (Hesser *et al.* 1978), mirror drawing (Frankenhaeuser *et al.* 1963), two-dimensional tracking (Whitacker and Findley, 1977; Moeller *et al.* 1981; Moeller and Chattin, 1975; Schmidt *et al.* 1974) and the stabilimeter (Adolfson *et al.* 1972).

One of the main problems when comparing performance data across the experiments cited above was the variety of conditions and protocols under which the assessment of narcosis was made. For example, several contrasting tests may be used to assess the same behavioural activity and even if the narcotic effects are assessed on the same test, these effects may be expressed differently. Subjects, although in most studies males, also varied widely in age, education, intelligence, motivation and diving experience. Additionally, a wide range of practice was required of subjects on tests. While the majority of baseline measures were collected at 1.0 ATA before hyperbaric exposure, a number of studies collected baseline data at low hyperbaric pressure. Even if conducted at 1.0 ATA, some baseline testing was done inside the chamber, while in other experiments baseline data was collected outside the chamber. The interval between baseline and hyperbaric testing also varied widely among experiments.

Poor experimental design was found to be a common fault in hyperbaric performance research which often led to contaminated data. Many experiments failed to counterbalance baseline and test conditions or to randomize exposure among subjects when multiple hyperbaric exposures were used. These design problems and procedural differences resulted in a wide range of reported performance decrements between categories in the descriptive model, as well as, between the contrasting intracategory performance tests. For this reason Fowler *et al.* (1985) regarded the descriptive model as an unsatisfactory portrayal of narcosis. They did, however, point out that the majority of data indicated cognitive tests were more sensitive than dexterity tests.

A second approach to the analysis of narcosis has been based on the hypothesis that sensitivity of physiological systems to anaesthetics (or hyperbaric air) depends on their phylogenic age (Himwich, 1951).

This hierarchial organization hypothesis, has been used by a number of researchers to explain an assortment of performance decrements with a wide spectrum of anaesthetics including nitrous oxide and hyperbaric air (Steinberg, 1954; Kiessling and Maag, 1962; Biersner *et al.* 1978; Hesser *et al.*, 1978; Adam, 1973). The hypothesis is based on the concepts that the newer "higher centers" of the central nervous system (CNS) responsible for complex mental functions are more readily affected by narcosis than the "lower centers". Thus the hypothesis implies that the greater the complexity of a performance test, the more it will be affected by narcosis.

Fowler *et al.* (1985) point out, however, that due to an inadequate objective method for defining complexity, interpretation of the degree of complexity of performance tests can only be made on common sense a priori grounds. Using the hierarchial organization hypothesis they allege it is possible to differentiate between tests involving predominantly higher cognitive processes and those tests that involve relatively little conscious processing, or reasoning ability, such as dexterity tests. Unfortunately, the reliability of further propositions based on this hypothesis deteriorates beyond this point (Fowler *et al.* 1985).

Recently the slowed processing model, (a predominantly behavioralist approach to nitrogen narcosis), has been extensively used by Fowler and co-workers (Fowler, 1972; Fowler and Granger, 1981; Hamilton *et al.* 1984; Fowler *et al.* 1983). The slowed processing model, with its foundations in cognitive psychology, is based on the notion that narcosis acts by slowing the different stages of information processing. The narcosis performance deficits, reflected by alterations in the speed and accuracy of responding, are thought to be caused by disruption of structural, functional or strategic variables, (either alone or in combination) (Fowler *et al.* 1985) (See Figure 2.1 and explanation in Section 2.0.2).

In order to distinguish the individual contributions of these variables, to the overall performance decrement, several techniques described in detail by Fowler *et al.* (1985), have been employed. These included the additive factor method (AFM) for isolating the effects of narcosis on the speed of processing of various stages and signal detection theory for distinguishing between the effects of narcosis on structural and strategic variables. The latter technique has been used on performance tests where accuracy is of primary concern, and fast responses are not required. Using AFM logic, the majority of studies on reaction time have indicated that slowing, due to narcosis, is a functional deficit brought about by a decrease in the efficiency of the system as a whole (Fowler *et al.* 1983; Fowler and Granger, 1981; Hamilton *et al.* 1984). This has led to the view that, decreased levels of arousal or activation in the CNS produced by narcosis

may be responsible for the performance deficiency on cognitive performance tasks.

Conversley, decreases in the accuracy of processing in reaction time and movement time experiments, have been attributed to changes in the speed-accuracy trade-off setting (a strategic change) rather than a structural change (Fowler *et al.* 1985). One of the proposed reasons for the decrease in accuracy is that subjects prefer to divide the impact of narcosis between speed and accuracy so that the loss in speed is not as conspicuous. A second reason is that the euphoria experienced under narcosis may provide a disinclination in the subject to maintain a given speed accuracy setting (Fowler *et al.* 1982).

2.0.2 Human Memory Processing and Narcosis

Memory deficits caused by narcosis have been known for some time. Early investigators reported that hyperbaric air, in excess of 4 ATA, produced acute cases of amnesia as well as learning deficits (Shilling and Willgrube, 1937; Behnke *et al.* 1935; Damant, 1930). With the aid of classic theories of memory information processing, the effects of narcosis on long term and short term memory have been extensively investigated (See Fowler *et al.* 1985 for a review). An example of a classic, simplistic, model of the human processing system is shown in Figure 2.1.

In the first stage of information processing, the stimulus enters the system via one or more of the senses and is held briefly in original sensory form in the respective sensory register. For example, acoustic stimuli will be processed in the register for hearing, termed "the echo", while visual stimuli will be processed in the register for vision, called "the icon" (Neisser, 1967).

While information is in a sensory register a number of important processes occur. One of these is pattern recognition, a complex process that results in contact between the information in a sensory register and previously acquired knowledge that is stored in long term memory (LTM). Generally, pattern recogntion may be thought of as assigning meaning to a stimulus (Klatzky, 1975). At this stage, in information processing, selective attention assures that important information is focussed on and not lost due to the system's limited capacity. Once an input to the system has been recognized and attended to, it can be passed on to the next stage of the system, namely short term memory (STM).

Manipulation and temporary storage of information in STM is only partly understood (Baddeley, 1983; Hitch, 1980) however, rehearsal has been shown to play an essential part in maintaining information in STM. It has been suggested, for example, that a verbally coded item will last less then a half-minute in STM if it is not rehearsed, and that about a half dozen such items can be held in STM at one time

(Klatzky, 1975). A second function of rehearsal relates to the transfer of information to LTM. It was proposed, by Atkinson and Shiffrin (1968), that the more an item is rehearsed in STM and the longer it stays in STM, the more likely it is to be remembered later. Consequently, rehearsal processess can provide an opportunity for strengthening the representation of information in LTM, so that later it is more amenable to recall (Klatzky, 1975).

Despite the limitations of the model shown in Figure 2.1, similar models to this have provided the basis for researchers to assess performance decrements on different elements of the information processing system. Much of the work on memory has been performed using N_2O as the narcotic agent. Experiments on the effects of nitrous oxide on STM have produced equivocal results, with Steinberg (1954), Biersner (1972), Fowler (1973) and Adam (1979) showing negligible effects, while Garfield *et al.* (1975), Berry (1965, 1968) and Cook *et al.* (1978) demonstrated a significant decrement. More recent evidence has pointed to perceptual deficits, unrelated to the narcotic properties of N_2O, as a possible reason for the observed STM deficit, at least upon dichotic listening tasks (Fowler *et al.* 1980). Furthermore, Fowler *et al.* (1980) suggested that the decrement found in some N_2O STM studies may have been due to confusing the measurement of STM with that of LTM. One reason for this confusion may be the result of STM memory tasks defined on the basis of elapsed time between presentation of the stimulus and recall (Fowler *et al.* 1980). An alternative approach to the problem of distinguishing between STM and LTM is

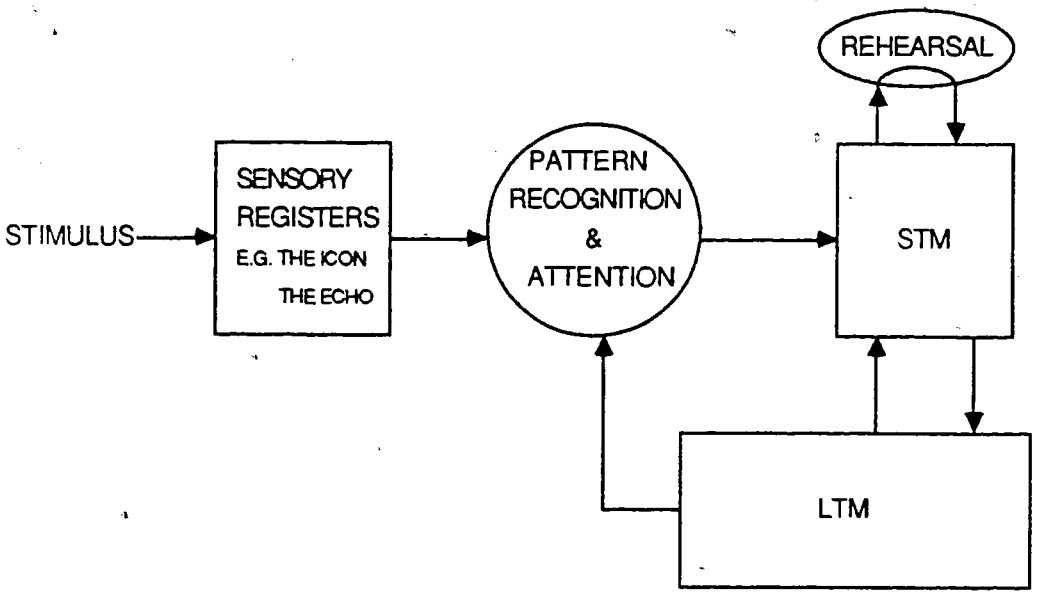


Figure 2.1 A simplistic model of the human information processing system. (Adapted from Klatzky, 1975 and Fowler *et al.* 1985).

to catagorize STM and LTM according to the initial and final responses on a serial position curve. These curves when plotted typically show an initial negative slope (the primacy effect), a flat central portion, and a final positive slope for the last few items on the serial list (the recency effect).

According to the duplex theory of human memory, the primacy effect is a result of recall from LTM, while the recency effect demonstrates recall from STM (Klatzky, 1975). Analyzing narcotic memory deficits on STM and LTM in this manner, Fowler *et al.* (1980) concluded that both N_2O and hyperbaric air impairs LTM but not STM. Furthermore, using cued recall, the LTM deficit was found to be caused by impaired learning rather than a disruption of memory organization. Similar conclusions by Steinberg and Summerfield (1957), Fowler (1973) and Fowler and Ackles (1975), have suggested the view that input to, rather than retrieval from, LTM is the source of the narcotic deficit.

2.0.3 The Role of Carbon Dioxide as a Causative Factor in Compressed Air Narcosis

One school of thought initiated by the work of Bean (1947, 1950) suggested that CO_2 should be considered as an alternative or at least a contributory cause of compressed air intoxication. Bean suggested that hyperbaria increases the gas density and viscosity to a point when respiratory flow is inhibited, leading to retention of CO_2 and a deficiency of O_2. Carbon dioxide retention was also thought to be enhanced by the fast rate of compression producing an inflow of CO_2. More recent support for this hypothesis has been provided by Seusing and Drube (1960), Buhlman (1963), and Vail (1971) based on similar arguments.

However, the hypothesis that carbon dioxide is the predominant cause of compressed air narcosis failed to be supported by experiments conducted by Rashbass (1955) and Cabarrou (1959,1964). In the former experiment the initial rise in P_ACO_2 following compression to 8.6 ATA was manipulated by hyperventilation. The degree of narcosis impairment was assessed using a two–figure by one–figure multiplication task. Although 5 minutes of hyperventilation reduced the initial mean P_ACO_2 from 5.2 to 3.9% the score on the arithmetic test remained unchanged. In the latter studies by Cabarrou, compression (at very slow rates) or hyperventilation (before or after compression), ensured that P_ACO_2 levels at pressure remained normal. It was implied from these experiments that raised levels of CO_2 were not singly responsible for narcosis at depth.

Although there is ample evidence that respiratory embarrassment does occur at increased pressures (Miles, 1957; Wood *et al.* 1962; Lanphier 1963; Wood 1963) and that inadequate ventilation leads to CO_2 retention, the CO_2 apparently only potentiates the narcotic action of the inert gas (Bennett, 1982).

Further evidence in conflict with the CO_2 hypothesis, has been obtained by Bennett (1965) from in vivo measurements of the brain CO_2 and O_2 tensions in chloralosed cats. It was found that during compression the cortical partial pressure of CO_2 fell, differing from the increased P_ACO_2 measurements noted by Cabarrou (1959, 1964) and Bean (1950). The assumption made by the latter authors, that the tissue partial pressure of CO_2 was equally high, may therefore not necessarily be correct. Furthermore, investigations using a variety of inert gas mixtures with depression of auditory evoked potentials as a measure of narcosis (Bennett, 1964) showed no correlation between CO_2 retention and the degree of narcosis. From these experiments Bennett (1966) concluded that the postulated cause of the initial narcosis was unlikely to be due to an increased cortical PCO_2. More recent evidence pointing towards nitrogen as the principle cause of compressed air narcosis rather than CO_2 was provided by the work of Weiss and Torley (1975).

In this investigation chickens were used as the animal model, and the amplitude of the visually evoked response (VER) as the measure of narcosis. On exposure to normoxic, normocapnic air at 6 ATA the birds showed a depression of the VER amplitude. The effect of pressure *per se* was ruled out as the cause of this depression as there was no change in the amplitude of the VER when Helium was substituted for N_2 as the inert diluteni. Weiss and Torley (1975) also investigated the effects of changes in lung PCO_2 on the VER and found that for lung PCO_2 tensions between 0 and 70mmHg there was no effect on VER at surface or at 6 ATA. It was also, noted that the birds became increasingly harder to handle at the higher concentrations of CO_2 and seemed to be suffering from CNS hyperexcitability. They concluded that the depression of VER appeared to be a reflection of narcosis induced by high N_2 pressure and not solely hypoxia, hypercapnia or pressure *per se*.

The validity of the cortical evoked response as a measure of inert gas narcosis has however recieved a critical blow by Fowler and Ackles (1977). For a non behavioural measure to be accepted as a valid indicator of narcosis they argue that all the following criteria must be met:

1. For any given gas the measure must show a change as a function of the partial pressure of the gas;
2. The measure must reflect the order of narcotic potency which has been established for some of the inert gases...
3. For any given gas, there must be a correlation between the nonbehavioural measure and behavioural indicators of narcosis.
(Fowler & Ackles, 1977, p.82)

Although most studies have shown the amplitude of the evoked response to decrease with increasing ambient pressure, this decrease has not been found to correlate with any behavioural measure of narcosis,

nor be reflected by the narcotic potency of the breathing mixture (Fowler & Ackles, 1977). These observations indicate that the evoked response satisfies the first criterion but not the second or third criteria. The conclusions of Bennett (1964, 1966) and Weiss and Torley (1975) should therefore be carefully considered on the basis that the evoked response does not measure inert gas narcosis according to the criteria set out by Fowler and Ackles (1977).

Despite lack of support for CO_2 as the major cause of compressed air narcosis, the possibility of an interacting effect of hypercapnia and nitrogen narcosis should not be taken lightly. An early study conducted by Case and Haldane (1941) in which subjects were exposed to 4-6% CO_2 at 10 ATA graphically emphasised the subjective sensations experienced under these conditions. Initially, individual symptoms of confusion, distress, impending syncope, euphoria and elation were experienced by their subjects at 8.5 or 10 ATA. These symptoms became more severe when CO_2 was added to the air at 10 ATA. As the inspired CO_2 was increased from 4% to 9% the divers generally lost consciousness within 1-5 minutes breathing the highest level of CO_2. It was also demonstrated that individuals lose consciousness at very different partial pressures of CO_2 indicating a wide range of human susceptibility. Although breathing was found to increase noticeably with CO_2 partial pressures above 3% (surface equivalent), the subjective stress to this level of CO_2 was reported to be much less at increased ambient pressure than at surface. Case and Haldane (1941) tentatively attributed this to the narcotic effect of nitrogen. Breathing 4% CO_2 at pressures between 8.6 and 10 ATA resulted in a significant decrement in manual dexterity and an increase of 46% in the mean percentage of mistakes on an arithmetic task. It was also recorded that one subject, when attempting the arithmetic test under these conditions, simply stared at the paper for 7 minutes, managing to write down only two digits, one of which was wrong. From their observations, Case and Haldane (1941), suggest that the partial pressure of CO_2 at 10 ATA should be kept below 3% to avoid severe impairment of performance.

Whether the increased P_aCO_2 is due to inhaled CO_2 or to metabolically produced CO_2 the physiological effect is the same (Lambertsen, 1971). Lanphier (1963) reported that loss of consciousness can be precipitated at lower pressures by exercise-induced hypercapnia. Lanphier noted this effect while testing a new bicycle ergometer and breathing system, at 7.8 ATA in a dry chamber. It should be noted however, that the breathing system supplied only about half of the subjects' respiratory needs (Lanphier and Comparesi, 1982). In this case dyspnoea was very prominent and narcosis proceeded rapidly to coma. It was suggested by Lanphier and Camporesi (1982) that if this experience occurred in open water the probability of survival would be very poor.

Subsequent tests were conducted by Lanphier and Camporesi (1982) providing free access to air or replacement of the nitrogen content of the breathing gas while exercising on the bicycle ergometer at depth. In both these cases the divers remained conscious, indicating that the synergistic narcotic effects of N_2 were necessary to produce loss of consciousness (Lanphier and Camporesi, 1982).

This view was shared by Morrison *et al.* (1978) who reported a study of two divers who had been rescued after losing consciousness at depth. When the conditions were simulated in the laboratory, both divers exhibited marked CO_2 retention during exercise at 4 ATA, ($P_{ET}CO_2$= 65 and 57 mmHg respectively) and at 8 ATA one diver stopped work in less than 3 minutes because of severe dizziness and impending loss of consciousness. A tentative explanation for this phenomenon was provided in terms of reduced sensitivity to CO_2, perhaps caused by the interaction of hypercapnia and N_2 narcosis (Morrison *et al.* 1978).

It has been suggested that, in addition to a reduced chemosensitivity to CO_2, experienced divers develop a conditioned response following repeated hyperbaric exposures (Lally *et al.* 1974). During exercise, divers have been found to exhibit a markedly slower and deeper breathing pattern compared to sedentary non-divers (Broussolle *et al.* 1972; Morrison *et al.* 1978, 1981) and athletes (runners) (Lally *et al.* 1972). Hypoventilation, however, leads to a reduced elimination of CO_2 and a subsequent CO_2 retention (Broussolle *et al.* 1972; Morrison *et al.* 1981). Although this "adaptation" may be efficient in terms of air conservation, the resulting hypercapnia may enhance O_2 toxicity and nitrogen narcosis (Lanphier, 1982), and be a potential hazard to the diver.

Clearly, further studies are required to clarify the role of hypercapnia and nitrogen narcosis in such cases. It has been suggested by Morrison *et al.* (1978) that a narcosis test would be a valuable addition to future investigations of hypercapnia in the hyperbaric environment, and for screening potential "CO_2 retainers".

One of the first attempts to evaluate quantitatively the combined effects of raised N_2 pressure and varying degrees of hypercapnia on perfomance was carried out by Hesser *et al.* (1971). These authors compared the effects of a variety of CO_2 partial pressures in air at 6 ATA with the same partial pressures in O_2 at 1.3 ATA. Consequently the P_IO_2 was approximately 1.2 ATA in both conditions whereas the inspired N_2 differed by 4.7 ATA. Performance responses to the different CO_2 partial pressures were determined by the Moede perceptual-motor ability test and the Stroop stress sensitivity test. The results indicated that the CO_2 component was negligible at P_ACO_2 tensions below 40 mmHg and that high

alveolar N_2 and CO_2 pressures were simply additive in their effects on causing a deterioration in performance. In addition, when related to the inspired gas tension, changes in performance induced by simultaneously raising the N_2 and CO_2 pressure were greater than the arithmetic sum of the changes induced by either gas alone (Hesser *et al.* 1971).

From Hesser *et al.*'s results, it was concluded that the synergistic relationship between CO_2 and N_2 narcosis was an artifact arising from the measurement of inspired CO_2 rather than P_ACO_2. However a study by Adolfson *et al.* (1973) demonstrated that, in contrast to cognitive and dexterity tests , body sway showed synergy between P_ACO_2 and air at 8 ATA. An effect that was generally enhanced when the eyes were closed.

Further experimentation by Hesser *et al.* in 1978 attempted to determine the roles of N_2, O_2 and CO_2 in compressed air narcosis. In this study increased P_ACO_2 was found to be additive with hyperbaric air at 8 ATA for mental arithmetic, but while CO_2 degraded manual dexterity, hyperbaric air did not. In addition, when the O_2 partial pressure was varied independently at 1.7 ATA, it was found to parallel the pattern of effects found for hyperbaric air. The authors concluded from this that the mechanism of CO_2 narcosis differs fundamentally from that of N_2 and O_2 narcosis.

Although it is well recognised that high PCO_2 levels enhance the severity of N_2 narcosis (Case & Haldane 1941; Hesser 1963; Lanphier 1963; Marshall 1951), the underlying relationship between CO_2 intoxication and N_2 narcosis in terms of potentiation, and/or additive or synergistic effects still requires further research.

2.0.4 Symptomatology of Raised CO_2 and N_2 Concentrations

At surface pressures, raised CO_2 concentrations have marked effects on both the heart and circulation as well as the nervous system. In general CO_2 inhalation produces respiratory stimulation, cerebral dilation, headache and a decrease in brain excitability. If the CO_2 concentration is raised above 10% at sea level, there is also confusion leading to unconsciousness (Greenbaum & Hoff, 1966; Woodbury *et al.* 1958). More specifically, acute exposure to CO_2 in human subjects for 15 minutes (7.5% proving minimum for generalized symptoms) results in various combinations of dyspnea and headaches, vertigo, sweating and numbness, over activity of limbs, increased motor activity and restlessness, visual and colour distortions, loss of balance and mental disorientation (Greenbaum & Hoff, 1966). In addition, as the PCO_2 is increased, cardiac output tends to increase, as does systolic and diastolic blood pressure, heart rate and

respiratory minute volume. Plasma concentrations of epinephrines, norepinephrine and 17–OH corticosteroids have also been shown to increase during hypercarbia (Sechzer *et al.* 1960).

The symptoms of CO_2 intoxication can occur, however, without the warning signs of a compensatory increase in ventilation. Barlow *et al.* (1944) and Donald (1946) have shown that rebreathing 50 litres of O_2 at 1.66 ATA, without absorbing CO_2, produces CO_2 intoxication with no warning signs of distress. A considerable variation of intoxication in the different subjects, which was related to their rate of ventilation, was also observed. Among the signs and symptoms which occured were dizziness, tingling of the limbs and visual disturbances. Haziness, euphoria or sleepiness were found to be common in sensitive subjects, without any change in ventilation rate. Flushing of the face, dilation of the pupils and sweating were present and muscular trembling was reported in some men, mainly in the arm, shoulder and neck muscles. Muscular tremor was widely diversified among subjects, in some the tremor was hardly detectable, while in others were coarse and violent and spread to all parts of the body. As the CO_2 intoxication became severe the subjects were seen to work at a feverishly increased rate, ignoring instructions, until they eventually reached a stuporous condition and consciousness was lost.

When the subjects regained consciousness during the recovery period Barlow *et al.* (1944) reported that most subjects experienced an "off effect" of CO_2 intoxication, which included symptoms of frontal headache, nausea or vomiting and a general fatigue malaise.

The signs and symptoms of CO_2 intoxication in the presence of high O_2 tensions indicate the relative importance of oxygen partial pressures in the contribution to CO_2 retention and the resultant danger from hypercapnia at depth.

When the effects of CO_2 intoxication are compared to N_2 narcosis the symptoms are distinctly different. Unlike CO_2 intoxication, exposure to high nitrogen pressures does not significantly stimulate ventilation nor elevate heart rate. In addition, although a certain degree of fatigue may be present after a dive, no symptoms of frontal headache, nausea or vomiting are induced by raised N_2 pressures. However, the symptomatology of N_2 narcosis, like CO_2 intoxication, is also dependent upon the PO_2 level of the particular breathing mixture used.

It was demonstrated by Criscuoli and Albano (1971) that exposure to high N_2 pressures, with normal PO_2, caused a progressive impairment in the subjects of the vigilance state, ideation, fixation memory, practical activity, and affective self-control. Logical thought and attenuated associative processes were

clouded, as well as the capacity for and the consistency for critical synthesis. The use of sensory functions for practical purposes was also impaired. Furthermore, it was reported that the subjects exhibited a reactive dysphoria, which tended to break down under external stimulation (Criscuoli & Albano, 1971).

When the above symptoms were compared to those experienced during exposure to compressed air at 12 ATA a quantitative change in the conscious state was found. This state was distinguished by a euphoria which was accompanied by irritability and impulsiveness. In addition, Criscuoli and Albano (1971) noted frequent perceptual dysfunction in their subjects, particularly in the senses of touch, sight and hearing. Distal motor hyperkinesis and subliminal tremor was found to impair working ability, and execution of physical tasks remained defective throughout the testing period. Also noted in the study was the fact that habitual motor acts were often performed with perseveration (Criscuoli & Albano, 1971).

2.0.5 Proposed Mechanisms of Compressed Air Narcosis and Carbon Dioxide Intoxication

Despite volumes of research the cause of compressed air narcosis is considered by some not to be fully proved. This has led to conflicting views, resulting in a number of hypotheses as to the mechanisms of the narcosis.

In the past, many attempts have been made to connect a number of factors with the mechanisms of narcosis. These factors have included histotoxic hypoxia, depression of metabolism, cell membrane stabilization causing a block in ion permeability, inhibition of the sodium extrusion pump, increased production of inhibitors such as gamma-aminobutyric acid and interference with ATP production (Butler 1950; Pittinger & Keasling 1959; Featherstone & Muehlbaecher 1963; Latner 1965; Quastel 1963; Bennett 1966). However due to the inert nature of the rare gases and nitrogen, i.e., their inability to form covalent or hydrogen bonds in biochemical processes within living tissue, physical rather than chemical properties should be considered as more likely mechanisms by which narcosis is produced (Bennett 1966).

With regard to the biochemical theories of narcosis very little satisfactory evidence has been obtained of biochemical changes at pressures which may nonetheless produce inert gas narcosis (Levy & Featherstone, 1954; Carpenter, 1955, 1956; Leon & Cook, 1960; Thomas *et al.* 1963; Schatte & Bennett, 1973). This has led to many authors favouring the physical theories, based on the polarisability and volume of the inert gas molecule, as being a more likely mechanism of inert gas narcosis (Featherstone & Muehlbaecher, 1963).

However, McIlwain (1962) has also proposed a theory linking the biochemical and physical theories which suggests that inert gases (such as volatile anaesthetics in general) interact with the cell membrane, blocking ion exchange, and resulting in a decrease in cellular O_2 consumption. The ionic block mechanism was also favoured by Mullins (1954) who postulated that the inert gas molecules acted by occluding the pores of the membrane. Similarily, Sears (1962) suggested that interaction of the inert gases within the membranes of the synaptic vesicles may prevent the release of their chemical transmitter contents.

The basis of the inert gas theory, which suggested the lipid phase of the cell membrane as the site of action of the inert gases, was originally conceived from the collective ideas of Meyer and Overton (Meyer, 1899; Overton, 1901). The suggestion that there is a parallel between the affinity of an aliphatic anaesthetic for lipid and its narcotic potency led Behnke *et al.* (1935) to propose that it is the increased nitrogen partial pressure that is responsible for compressed air narcosis.

Despite some anomalies, in connection with anaesthetic agents in general, excellent correlations betweeen lipid solubility and narcotic potency have been shown (Bennett, 1982). Other factors including the partial molal-free energy, derived from the fugacity of the gas (Ferguson, 1939; Brink & Posternak, 1948; Ferguson & Hawkins, 1949; Marshall, 1951; Carpenter, 1954) and the van der Waal's physical chemical constants (Wulf & Featherstone 1957) have also shown good correlations with narcotic potency.

All the above correlations are based on an interaction with the lipid phase of the nervous system. However, although the lipid phase is generally accepted as the site of action of anaesthetics (Meyer & Hopff, 1923; Butler, 1950), other authors (Miller, 1961; Pauling, 1961) have suggested the aqueous phase as an alternative site.

The aqueous phase theories are based on the formation of hydrates. In 1961 Pauling proposed that during narcosis microcrystals of hydrates of the clathrate type are formed which are stabilized by proteins. Such clathrates are considered to cause narcosis by increasing the impedance of nerve tissue, trapping electrically charged ions associated with impulse conduction and decreasing metabolism (Bennett, 1982).

An alternative but related theory proposed by Miller in the same year implied that the inert gases may increase the area of highly ordered water, or icebergs, surrounding a dissolved molecule. In this manner the conductance of the brain tissue would be lowered, the lipid membranes stiffened and the membranes occluded.

More recent investigations (Dawe *et al.* 1964; Miller *et al.* 1965,1967; and Eger *et al.* 1969) have provided little evidence for support of the aqueous medium as the critical locus of anaesthetic action. Indeed other experiments have suggested another binding mechanism in which inert gases are bound to specific sites within protein molecules (Featherstone *et al.* 1961; Featherstone & Meuhlbaecher, 1963; Schoenborn *et al.* 1965; Schoenborn, 1965; Eyring *et al.* 1973; Katz & Simon, 1977; Franks & Lieb, 1978). However the connection of this latter theory with the mechanisms of anaesthesia still remains to be elucidated (Bennett, 1982).

At the electrophysical level pioneering work by Marshall and Fenn (1950) on frogs and by Carpenter (1953, 1954, 1955) on mice, have led to the inference that the site of action for inert gas narcosis is at central synapses. In addition, later work showing a correlation between changes in the mental state of divers and depression of the polysynaptic ascending reticular formation of the brain stem, as shown by an abolition of the alpha blocking response (Bennett & Glass, 1961) or change in the fusion frequency of flicker (Bennett & Cross, 1960), have also indirectly supported involvement of the synapse. From these experiments it was inferred that a fundamental change occurs at polysynaptic sites in the brain when a critical tension of N_2 or inert gas is exceeded (Bennett, 1982).

This concept is related to the 'critical volume of occupation' proposed by Mullins (1954) which was further developed by Miller *et al.* (1973) to produce the so called 'critical volume hypothesis'. This hypothesis states that anaesthesia occurs when the volume of a hydrophobic region is caused to expand beyond a certain critical volume as a result of adsorption of an anaesthetic agent. The hypothesis also stated that all anaesthetics act at the same molecular site. However, work by Halsey *et al.* (1978) does not support this latter view and has led to their developing the multi-site expansion hypothesis which was summarised by Bennett (1982) as follows:

1. General anaesthesia can be produced by the expansion of more than one molecular site and these sites may have differing physical properties.
2. The physical properties of a molecular site may themselves be influenced by the presence of anaesthetics or pressure— e.g., the compressibility.
3. The molecular sites do not behave as if they were bulk solvents but have a finite size and a finite degree of occupancy.
4. Pressure itself need not necessarily act at the same site as the anaesthetic. Depending on the anaesthetic one of the sites may predominate in determining the interaction with pressure.
5. The molecular sites for anaesthesia are not perturbed by a decrease in temperature in a manner analogous to an increase in pressure.

Lateral expansion of membranes in the presence of anaesthesia has been shown by Clements and Wilson (1962). They noted that narcotic agents such as nitrous oxide have an affinity for lipid monolayers

and concluded that inert gases at partial pressures, sufficient to bring about a standard effect in a biological system, act on a lipoprotein-water interface to cause a standard decrease of 0.39 dyn/cm in the interfacial tension. Further support showing that the inert gases, as well as O_2 and CO_2, penetrate a lipid monolayer and cause changes in surface tension was provided by Bennett *et al.* (1967). It was found that, even at very low depths, CO_2 readily penetrates the lipid monolayer producing a relatively large change in film pressure. This may explain in part the relatively high narcotic potency of CO_2 found in the studies by Hesser *et al.* (1978). However this assumes that the mechanism of action of CO_2 intoxication is mediated through an effect on the lipid membrane. Unfortunately, at present, there is no conclusive evidence for the underlying mechanism of CO_2 narcosis.

Hesser *et al.* (1978) hypothesised that the high narcotic potency of CO_2 noted in their studies was possibly mediated by hydrogen ions. In support of this statement, they cite the observations of Eisele *et al.* (1967), who noted that in dogs the anaesthetic effect of CO_2 correlated better with the pH of the cerebral spinal fluid rather than P_aCO_2. However, Hesser *et al.* (1978) did not completely discount the possibility that CO_2 has narcotic effects that act in accordance with the lipid solubility theory. These authors have suggested, albeit with little experimental support, that the narcotic effects produced by CO_2, in accordance with the solubility theory, may well be masked by hydrogen ion induced narcosis occuring at far lower PCO_2 levels.

Earlier evidence does however implicate the role of hydrogen ions in CO_2 narcosis. Meyer & Waltz (1961) have suggested that CO_2 narcosis produces a slowing of the EEG which correlates with cortical pH rather than the PCO_2. This increased acidity is thought to cause a reversible suppression of the "sodium extrusion pump" which may lead to a synergistic narcotic effect.

A third mechanism by which CO_2 may produce its narcotic effects is via histotoxic hypoxia. A raised CO_2 tension will tend to reverse the chemical equilibrium between O_2 and CO_2. This would decrease the entry of O_2 into the cell. Normally the degree of hypoxia caused by this mechanism may not be enough to produce signs and symptoms of narcosis. However, Bennett (1966) has suggested that, combined with a histotoxic hypoxia as a result of the inert gas partial pressure, a synergistic action of CO_2 might well result.

Another explanation for the enhancement of narcosis by CO_2 may be due to its effects on cerebral blood flow. Carbon dioxide inhalation has been shown to increase cerebral blood flow (Novack *et al.* 1953; Sokoloff, 1960); consequently, during hyperbaric exposure, any increase in P_aCO_2 levels will induce

cerebral vasodilation producing a greater blood flow to the brain. This increase in blood flow may cause potentiation of the narcosis by allowing greater levels of N_2 to reach the brain tissues.

Patterson *et al.* (1955) have reported that the vasodilator response of normal cerebral vessels to rapid increases in P_aCO_2 appears to be a threshold type of phenomenon. It is of note that this phenomenon is also indicated in the observations of Hesser *et al.* (1978), in that the role of CO_2 as a causative factor in narcosis is negligible as long as the P_aCO_2 does not exceed 40mmHg. Above this level the role of the CO_2 component in compressed air narcosis was found to markedly increase with the rise in CO_2 pressure.

It is clear that the mechanisms of CO_2 toxicity may be mediated by a number of different causes, namley (1) increased concentrations of molecular CO_2, (2) increased hydrogen ion concentration, or (3) concurrent effects of No. 1 and No. 2. The overall picture of CO_2 toxicity is complicated by the fact that the toxic actions of hypercapnia vary according to the level of PCO_2 and cell type. Sudden, extreme elevation of CO_2 tension, such as that caused by inhalation of 30% CO_2, produces simultaneous manifestations of depression and stimulation in the CNS (Lambertsen, 1974). Convulsions caused by CNS hyperactivity at this level of inspired CO_2 are thought to be the paradoxical result of a depressant effect on the cerebral cortex, releasing subcortical centers from normally powerful inhibitory influences (Woodbury & Karler, 1960). Thus what may actually be a depressant or disruptive effect possibly of molecular CO_2 as well as hydrogen ions, is expressed in the form of increased activity (Lambertsen, 1974).

2.0.6 Concluding Remarks

From the review presented, it is clear that there are insufficient quantitative data concerning the role of CO_2 in compressed air narcosis. The evidence suggests CO_2 has additive, not synergistic, effects on cognitive function in combination with hyperbaric air, however this view is far from conclusive. Fowler *et al.* (1985) point out that there is some evidence, although sparse, which suggests that CO_2 does not potentiate narcosis. This observation is based on the fact that N_2 and CO_2 affect performance tests in different ways (Hesser *et al.* 1978), implying that CO_2 intoxication acts through a different mechanism to that of N_2 narcosis. In a review paper by Fowler *et al.* (1985) it was suggested that, to avoid confusion over the interpretation of how these two factors act together, a more concise definition of the terminology used should be adhered to. In response to this suggestion several of the terms used to denote the outcome

of responses when CO_2 and N_2 act together are defined in Section 1.2.

Decrements in performance have been demonstrated to occur above a P_ACO_2 of 53mbar (40mmHg) at depth (Hesser *et al.* 1978) and yet it is known that a P_ACO_2 of 67mbar and above is not uncommon in trained divers who hypoventilate (Morrison & Reimers, 1982). In addition, P_ACO_2 tensions as high as 93mbar (72mmHg) have been recorded in subjects exercising on an underwater ergometer at 6 ATA during the last few minutes of an incremental exercise test (Taylor, 1987).

From these studies it is clear that raised pressures of CO_2 in combination with elevated pressures of N_2 have marked effects on cognitive function which may lead to serious threats to diver safety. The relative importance of hypercapnia in producing these effects has been demonstrated by studies on the narcotic action of various gases. It has been shown that the ratio of narcotic or anaesthetic potency of CO_2 and N_2O approximates 5:1 (Severinghous, 1974) and that of N_2O and N_2 is estimated at 28:1 (Brauer and Way, 1970). From these figures it can be inferred that an increase of 4 mmHg in alveolar PCO_2 would have a narcotic affect roughly equivalent to the addition of 1 ATA of air (Morrison *et al.* 1978). This suggests the narcotic potency of CO_2 is at least 140 times that of nitrogen. However, Hesser *et al.* (1978) (who have produced the only quantitative data on CO_2 narcosis reported in the literature to date) have indicated that this figure is even greater. From the limited data reported in their studies they have proposed that CO_2 is several hundred times as narcotic as N_2 (Hesser *et al.* 1971, 1978). As these two studies are the only source of quantitative data on CO_2 narcosis in the hyperbaric environment the relative contribution of CO_2 in compressed air narcosis is at present only beginning to be revealed.

In view of the apparent importance of this factor to the diving community and the present lack of quantitative data available on this topic, the primary objective of this thesis is to expand current knowledge in this area and attempt to provide a working relationship for the role of CO_2 narcosis on cognitive and psychomotor performance in the hyperbaric environment. To accommodate this a number of pencil and paper tests and a test of fine manual dexterity were utilized to assess performance on a variety of underlying cognitive and psychomotor factors thought to be important for efficient performance in the hyperbaric environment. By ensuring a fully balanced experimental design, controling for practice effects, and employing a reasonable subject population size, many of the problems associated with previous hyperbaric research, was minimized.

2.1 HYPOTHESES

The hypotheses formulated for the present study are as follows:

1. When cognitive performance scores are compared at the two pressure levels (1 ATA and 6 ATA) under the same hypercapnic conditions statistically significant impairment in performance will be observed at 6 ATA.
2. When the $P_{ET}CO_2$ tension is increased from baseline to over 40mmHg at 1 ATA of pressure, statistically significant impairment of psychomotor and cognitive abilities will occur.
3. That psychomotor performance as measured via the Purdue pegboard test will be statistically significantly impaired by high $P_{ET}CO_2$ levels (> 40mmHg) but not statistically significantly affected by hyperbaric exposure.
4. That $P_{ET}CO_2$ tensions in excess of 40mmHg at 6 ATA will result in further deteriorations in psychomotor and cognitive abilities when compared to performance at 6 ATA breathing air containing normal hyperbaric levels of CO_2.
5. By dividing performance impairment into CO_2 and N_2 components it is hypothesised that; the test scores will show an additive effect of $P_{ET}CO_2$ on performance impairment when the effects of raising the $P_{ET}CO_2$ and P_IN_2 tensions simultaneously are analyzed.

Primary and Recency Memory Responses

6. Increasing the P_IN_2 to 6 times normal levels will result in a lower probability of recall in the primacy portion of the serial response curve on both the immediate and delayed recall responses to a paired association memory task.
7. Increasing the P_IN_2 to 6 times normal levels will have no effect on the probability of recall in the recency portion of the memory curve on both the immediate and delayed recall responses to a paired association task.
8. When the $P_{ET}CO_2$ is increased above 40mmHg, there will be a lower probability of recall in the primacy portion of the memory curve for immediate and delayed recall responses at both normobaric pressure and at 6 ATA of pressure.
9. Under the same conditions described in hypothesis 8 there will be no statistically significant effect of $P_{ET}CO_2$ on the probability of recall in the recency portion of the memory curve on either the immediate or delayed responses on the paired association task.

CHAPTER 3

METHODS

3.0.1 Subjects

Twelve healthy male volunteers, from the university population were paid to participate in this study. To obtain medical clearance for diving, in accordance with the Worker's Compensation Board of British Columbia, all subjects were required to complete a medical questionnaire and be examined by the departmental physician. Candidates with a current and/or history of any significant cardiorespiratory diseases, or other physical disorders which would contraindicate hyperbaric exposure, were excluded from the study. All subjects received information packages and signed informed consent releases.

Nine subjects had previously experienced the compressed air environment, in the hyperbaric chamber, before volunteering for the present study. The remaining three subjects were provided with an extensive orientation of the hyperbaric chamber facilities, culminating in a test dive to 2 ATA. Five of the subjects had previously been involved in studies using similar tests of cognitive and psychomotor function, however, all subjects were given sufficient practice on the tasks in the test battery to reach a learning plateau of performance before commencing the experimental trials.

3.0.2 Apparatus

All experiments were conducted in the Simon Fraser University Environmental Physiological Unit's (E.P.U.) hypo–hyperbaric (dry) chamber. This facility permits simulation of ocean depths to 300m to be conducted within the relative safety of a controlled laboratory setting, thereby reducing some of the potential risks associated with hyperbaric exposure in open water.

A semi–closed respiratory circuit was designed to elevate and maintain the subjects $P_{ET}CO_2$ at the desired levels for the duration of the experiment (Figure 3.1). The respiratory circuit consisted of: various lengths of 0.04m diameter respiratory tubing, an 8.4 litre mixing box containing several baffles to mix inspired and expired gases, a two–way valve, a mouthpiece and breathing valve, and a Conshelf 30 (U.S. Divers) open circuit demand regulator connected to a high pressure air supply. The subject inspired from the regulator through a length of respiratory tubing connected to the mixing box. Expired gases were either vented to the chamber via a two–way valve in the exhalation line, (for the non rebreathing

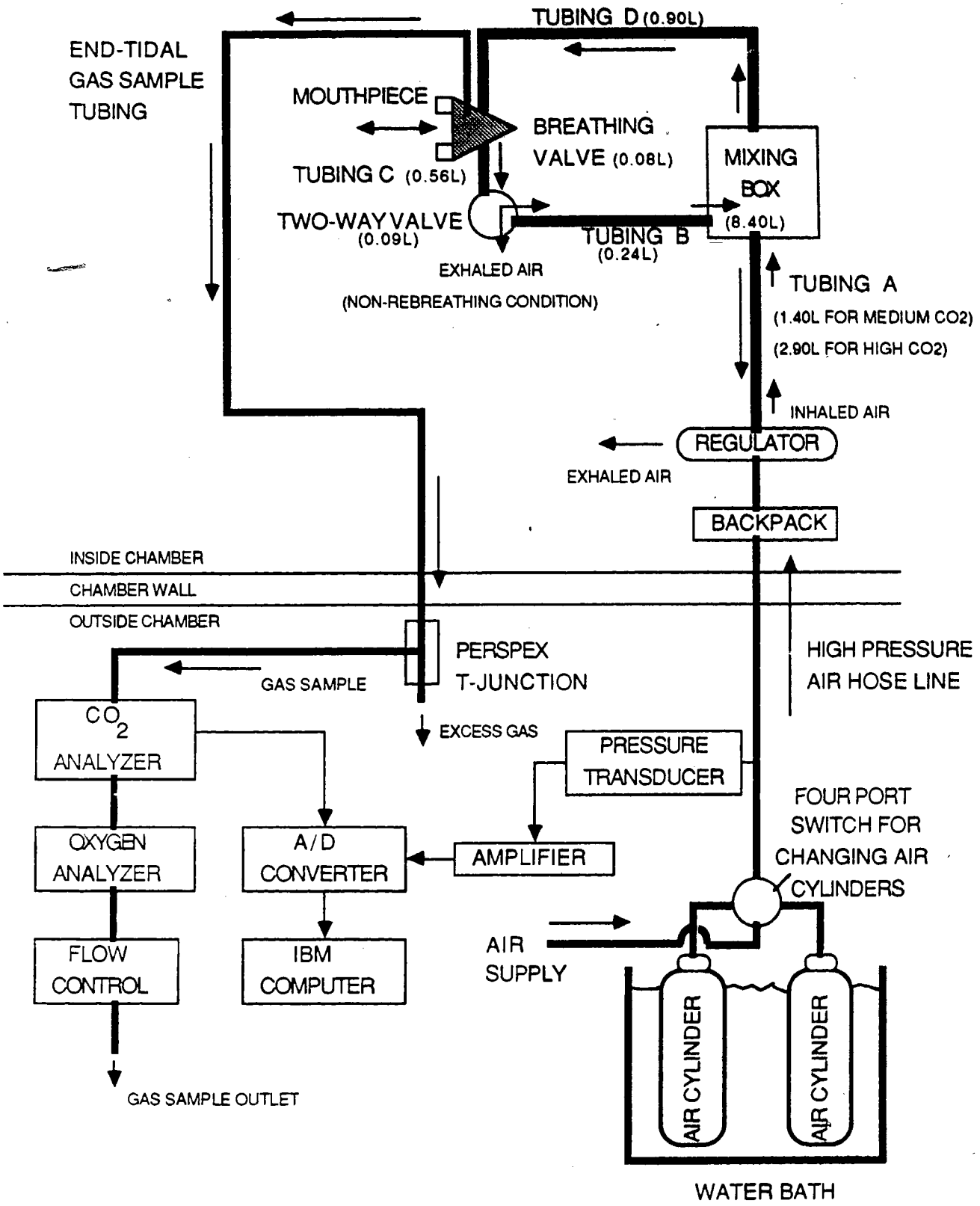


Figure 3.1. **Schematic diagram of the apparatus and instrumentation used during the chamber dives. (The figures in brackets represent volumes in litres).**

condition) or directed to the mixing chamber for rebreathing. The level of hypercapnia produced by this apparatus was induced by the introduction of appropriate lengths of 0.04m diameter tubing in the subject's breathing circuit (i.e. extending or shortening the length of tubing A shown in Figure 3.1). Pilot studies indicated that, for the majority of subjects, the volume of tubing A required to induce $P_{ET}CO_2$ values in the range of 45–50 and 55–60mmHg was 1.4 and 2.9 litres respectively.

Air supply to the subjects was provided from two calibrated gas cylinders, located external to the chamber. At the end of each minute, subjects were switched from one cylinder to the next, with the cylinder pressures being recorded at the start and end of each minute (Celesco Transducer Production Inc., Model PLD, ± 34.53MPa (±5000PSI). The cylinder, not in use, was refilled to a pressure of 13.8MPa (2000PSI) from the reserve cylinder bank. Between the air cylinders and the subject was a small surge bottle to prevent transient pressure drops, at the regulator, while changing cylinders. For surface trials 0.566m^3 capacity cylinders were used to enhance pressure measurement accuracy. At 6 ATA (50m), 1.416m^3 capacity cylinders were necessary to supply the air requirements under the increased ambient pressure.

Total system volumes, including cylinders, pressure hosing and the suerge bottle, were 3.68 and 7.85 litres for surface and 50m systems respectively. Ventilation was computed each minute from measurements of system volume and initial and final cylinder pressures (see Section 2.0.5)

End-tidal gas samples were drawn through the chamber wall and analyzed for CO_2 concentration (Ametek Applied Electrochemistry CD-3A CO_2 analyser). Sample lines of two internal diameters were used; 2.117mm for surface trials and 0.022mm for dive trials. A previous study using this arrangement (Taylor, 1987), indicated that these diameters were sufficient to maintain the fidelity of the CO_2 signal allowing minimal gas mixing to occur prior to analysis. Both systems were found to produce exponential CO_2 growth curves, with a rapid return to a stable baseline on inspiration (Taylor, 1987).

Both cylinder pressure and CO_2 signals were passed to an IBM(PC) micro-computer via an analogue to digital converter (Tecmar Labpac) for sampling and storage. A custom-designed program (Appendix 3) sampled the data on-line at a frequency of 33Hz and stored the values on 130mm MD-2D floppy disk for later analysis. Prior to sampling the pressure transducer values were amplified using a Daytronic LVDT model 9130 amplifier. No prior amplification was required for the analogue output of the CO_2 analyzer before sampling by the computer.

For administration and completion of the cognitive tests, materials included: pencils, paper and stopwatch. Psychomotor performance was assessed using the Purdue Pegboard (Purdue Research Foundation, 1948)

3.0.3 Procedure

Ethical and medical approval was sought and informed consents obtained specifying the purpose and nature of the experiment and possible adverse effects.

Once selected, each subject participated in a block practice session (5 practice trials) in the chamber at surface (1 ATA) on the test battery described below. It was expected that this practice session would familiarize subjects with the protocol and provide a plateau of learning performance on the individual tests. In addition, throughout the study each subject was required to complete a practice test battery immediately prior to commencing each experimental condition. This resulted in a total of eleven practice test batteries being completed by each subject.

In the absence of reliable objective physiological correlates of cortical functioning, paper-and-pencil psychological tests, were used to quatitatively assess various aspects of cognitive performance. Each test focused on a specific factor in cognitive or psychomotor performance, however there was some overlap in the factors being tested among the different tasks in the test battery. The individual tasks included in the test battery are described below.

1. *Paired Association test:* This test involved learning 12 pairs of word and number combinations. A different set of 12 words were used during each testing session. All words were randomly drawn from the pool developed by Modigliani and Seamon (1974) and consisted of common one and two-syllable concrete nouns. The number associated with each word was randomly assigned from a list of numbers between 1 and 100. To minimize proactive interference between successive memory tests, a number, once it had been used, was ommited from inclusion in subsequent word/number lists until all numbers between 1 and 100 had been used. In presenting the word/number combinations, a new word/number pair was revealed every 5 seconds and the previous word/number combination removed from the field of view. This resulted in the sequential presentation of all word/number combinations within a time frame of 1 minute. Immediate recall of the word/number associations was tested during the minute following presentation and delayed

recall tested 5 minutes following completion of the test battery (13 minutes after the initial presentation). Subjects were required to recall the word/number associations on a test paper containing only the rearranged words. A correct response was recorded when the correct number was matched with the corresponding word.

2. *Arithmetic test:* This test comprised 48 different arithmetic problems each consisting of adding or subtracting the result of one digit by one digit multiplication from a two digit number, such as 37+3X8= , 85–6X3=, etc. Subjects were instructed to multiply first and then add or subtract, and to emphasize both speed and accuracy. The time limit for this test was 2 minutes. Performance measures consisted of the number of problems attempted, as well as, the number of correctly and incorrectly solved problems.

3. *Copying test:* Each item consisted of a four line geometric configuration and a square matrix of dots. The task was to copy the figure onto the dots. It is believed that this task requires flexibility of closure in the act of superimposing the particular configuration on a strong visual field (Ekstrom *et al.* 1976). The subject was instructed to work as rapidly as possible without sacrificing accuracy. The score recorded was the number of correctly copied patterns or portions of patterns in a time period of 2 minutes. In addition, the number or portion of patterns attempted, as well as the errors, were recorded.

4. *Number Comparison test*: In this test subjects inspected pairs of multi-digit numbers and indicated whether the numbers in each pair were the same or different. The performance was scored on the number marked correctly, the number marked incorrectly and the total amount of numbers attempted in a time period of 45 seconds. This test primarily measures perceptual speed. It may be the centroid of several subfactors (including form discrimination) which can be separated but are more usefully treated as a single phenomenon for research purposes (Ekstrom *et al.*, 1976).

5. *Letter Cancellation test (modified Stroop test)*: This test consisted of the words *red* or *blue* typed on a page with ten letters afterwards. The colour name was underlined in either red or blue ink randomly. If the colour word was underlined in the same coloured ink then succeeding vowels were cancelled, otherwise consonants in the following ten letters were cancelled. Overall performance on the test was calculated according to the number attempted, the number correct and the number of errors within a time period of 1 minute. This test has been regarded as a measure of stress sensitivity in decision making (Stroop, 1935).

6. ***Purdue Pegboard test***: For this test subjects used both hands at the same time. They assembled a series of pins, collars and washers such that the order for one assembly consisted of a pin, a washer,

a collar and a washer. The score recorded was the number of individual parts assembled in 1 minute. This test primarily measures fine manual dexterity (Purdue Research Foundation, 1947).

Although catagorization of complexity of the above tasks, according to the neural organization or phylogenetic level required for efficient performance, is at best speculative, an estimate of the level of comparative difficulty of the individual tasks may be made on *a priori* grounds. On this basis the Purdue pegboard, the Number comparison test and the Copying test seem to be least difficult, whereas the Stroop, Arithmetic, Immediate and Delayed recall tests appear the most difficult. In the former catagory psychomotor and visual perception processes are the predominant factors tested. According to the literature these factors are least susceptible to narcosis and therefore would be expected to show only minor decrements in performance under stress. The latter catagory, however, contains tasks in which higher cognitive processes predominate, including immediate and long term memory. Consequently it was expected that these tests would demonstrate most impairment under narcosis.

After the practice trials, the subjects were split randomly into two groups of six. The first group completed control trials at 1 ATA, breathing normal air. The second group were compressed to 6 ATA where they breathed hyperbaric air. In each group, every subject experienced three levels of P_ICO_2 which was induced and regulated via the rebreathing circuit. After group 1 completed the 1 ATA trials, they underwent the 6 ATA condition, and vice versa for group 2.

The three CO_2 concentrations breathed at each pressure level were regulated so that $P_{ET}CO_2$ values fell within the ranges, 30–35 mmHg, 45–50 mmHg and 55–60 mmHg. Using the above $P_{ET}CO_2$ ranges, the upper time limit for breathing hypercapnic air was no more than 20 minutes during any one dive. Immediately before each 50m dive, the gas analyzers were calibrated using room air (20.93% O_2, 0.03% CO_2) and a primary standard gas containing 0.98% CO_2 and 20.00% O_2 with the balance N_2. Calibration of the gas analyzers prior to the surface trials was completed using room air and a primary standard gas containing 4.02% CO_2 and 16.72% O_2 with the balance N_2. These gas concentrations were chosen so that the gas analyzers were calibrated approximately within the mid-range of the expected gas analyzer readings for each pressure level. Linearity of the CO_2 gas analyzer was checked prior to the study using a series of 10 primary standard gasses ranging in CO_2 concentration from 0–10%. Linear regression analysis, comparing the known primary gas concentration of CO_2 with the CO_2 gas analyser meter readings, revealed reliable accuracy for the CO_2 gas analyzer throughout the measurement ranges employed in the

current study ($r^2 = 1$, residual mean square = 0.002% CO_2).

Once the required depth was reached, the subject would rebreathe from the respiratory circuit until the level of $P_{ET}CO_2$ was met. Upon reaching the desired $P_{ET}CO_2$ range (normally between 3 and 5 minutes) the first test of cognitive function was administered. The order in which the tests were presented was the same for each subject at each trial and followed the same order in which they are described in the above text.

Throughout each experimental trial an E.P.U. technician continuously monitored $P_{ET}CO_2$ levels and switched the air supply cylinder every minute. If at any time during the experiment the subject's $P_{ET}CO_2$ rose above the desired range, it was lowered by opening a two-way valve on the exhalation side of the rebreathing apparatus. This allowed exhaled concentrations of CO_2 to be vented to the chamber atmosphere and a subsequent lower P_ICO_2 in the respiratory circuit. Normally, the two-way valve would be left open for only one expiration in order to bring the $P_{ET}CO_2$ within the desired range.

During the surface trials 100% O_2 was supplied to the mixing box at a flow rate of 0.5 litres/minute. This was done to prevent the P_IO_2 dropping below normal levels during rebreathing. Inspired PO_2 was therefore maintained between 0.21 and 0.30 ATA during experimental trials at surface. At 6 ATA the increase in P_IO_2 caused by the ambient pressure increase was considered sufficient to prevent hypoxic stress during rebreathing (P_IO_2 at 6 ATA = 1.26 ATA.)

To minimize unwanted sequence effects between trials, subjects were randomly assigned to one of six treatment orders within each dive group. Successive trials on the same subject were conducted no earlier than 24 hours after the previous dive. Decompression for all dives was according to the Canadian Forces Air Diving Tables and Procedures (D.C.I.E.M., February 1986 revision). Most dives were completed within a bottom time of 20 minutes, resulting in a total decompression time of one hour.

3.0.4 Experimental Safety

Prior to commencing trials, all procedures were reviewed and approved by the E.P.U. Ethics Committee and by the Simon Fraser University Ethics Committee.

All subjects were examined, by a physician trained in hyperbaric medicine, and informed of the risks of compressed air diving. Those subjects who had no prior dive experience were required to complete a

test dive to 10 metres to assess suitability for hyperbaric exposure. Any subject who could not complete this test dive, or felt uncomfortable in the chamber, was not included in the study. Throughout all hyperbaric exposures a cardiovascular *crash cart* was available in the laboratory, and a physician present to oversee the dive.

The diving facility was controlled by two E.P.U. technicians. Two way communication was available with the diving tender through a Telex 2400 headset (Helle Engineering). In addition, all 50 metre dives were monitored on a closed circuit television by the attending physician and E.P.U. technicians.

Atmospheric control of the chamber environment was facilitated through an external Environmental Control System, which removed expired CO_2 from the air, and controlled air temperature and humidity. In case of emergencies air and O_2 demand breathing masks were also available. Due to the increased risk of fire hazard in the hyperbaric environment, all divers were required to dress in fire retardent clothing, and the O_2 breathing system exhaust gases were dumped external to the chamber.

The diving tender acompanying each subject was an experienced diver, trained in Cardiopulmonary Rescusitation (CPR), and familiar with chamber operation procedures. The diving tender was responsible for the subjects safety within the chamber as well as administration of the experiment at depth. When required, the rate of compression could be controlled from within the chamber by the diving tender.

Decompression procedures followed those specified by the Canadian Forces (Canadian Forces Air Diving Tables and Procedures, D.C.I.E.M., Feb. 1986 revision).

All subjects as well as the dive tender were closely monitored for latent decompression sickness for one hour following the 50 metre dives. The attending physician and E.P.U. technicians were available during this monitoring period. Following the one hour *bends watch* all divers received medical alert bracelets, which were worn for 24 hours after the dive. To avoid the use of repetitive dive tables, diving tenders and subjects were required to wait 24 hours before commencing succeeding dives.

3.0.5 Calculations

Data acquisition and analysis were performed by the computer programs listed in Appendix 3. Calibration programs were obtained from Morrison and Wood (1986).

End- tidal CO_2 tension

Carbon dioxide concentrations were calculated from the data collected during the first 30 seconds of each recording minute. These data were smoothed using a weighted five point, moving average before identifying peak (end-tidal) and trough (inspired) concentrations. The mean $P_{ET}CO_2$ for each minute were then calculated from the average of these peaks according to the equation below:

$$P_{ET}CO_2 = F_{ET}CO_2 [P_A/(P_A - P_{H_2O})].(P_A + PD - 47.1) \quad \textbf{Equation 1}$$

where:

$F_{ET}CO_2$ = mean end-tidal fraction of CO_2 concentration.

P_{H_2O} = water vapour pressure for ambient air (mmHg).

P_A = barometric pressure (mmHg).

PD = ambient pressure at dive depth (mmHg).

Ventilation

Minute ventilations ($\dot{V}_I$) were computed as dry volumes at standard pressure and ambient temperature ($\dot{V}_I SP$) from changes in air cylinder pressure. Since the cylinder volume was known, $\dot{V}_I SP$ was proportional to the change in cylinder pressure. The pressure data were subjected to a five point floating average before calculating $\dot{V}_I$. Both air cylinders were placed in a water bath, at room temperature, to minimise temperature changes during discharge and refilling; however, pressure data still required correction to account for isothermal expansion. Morrison and Wood (1986) performed a series of tests in which the effect of temperature on cylinder pressure changes, over one minute, was studied. Their results provided linear correction coefficients for both cylinder sizes. These coefficients were used within the present calculations.

$$\dot{V}_I BTPS = \dot{V}_I SP [760/(PD + P_A - 47.1)].[310/(273 + T)] \quad \textbf{Equation 2}$$

where:

T = temperature of cylinder bath.

PD = ambient pressure at dive depth (mmHg).

P_A = barometric pressure (mmHg).

3.0.6 Analysis

A comparison of cognitive and psychomotor performance differences between the three levels of $P_{ET}CO_2$ at 1 ATA and 6 ATA and between the two levels of P_IN_2 over the full range of hypercapnic conditions was conducted using two–way anovas with repeated measures. Following significant F statistics *post hoc* multiple comparisons using Tukey's HSD were used to isolate sources of significant diference. Where there was an insignificant interaction between N_2 and CO_2 the pooled data was used during subsequent *post hoc* analysis. This permited performance differences to be compared (1) at different P_IN_2 tensions over the full range of hypercapnic conditions [Hypothesis 1], (2) at $P_{ET}CO_2$ tensions above 40 mmHg under conditions of low P_IN_2 [Hypotheses 2], and (3) at $P_{ET}CO_2$ tensions >40 mmHg in combination with raised partial pressures of N_2 [Hypotheses 3,4].

Due to the possibility that delayed and immediate recall maybe governed by an array of psychological and/or strategic processes, producing different patterns of results; the subsequent analysis of immediate and delayed recall was performed separately. For both immediate and delayed recall the primacy and recency effects were analyzed by *a priori* division of the word/number presentation order into early, middle and late categories.

The first four word/number associations were considered to represent the primacy effect, while the last two word/number pairs represented the recency effect. These word/number associations were selected as depicting the most robust primacy and receny responses on the serial position curve, according to the literature (Modigliani, personal communication). Two–way ANOVA's with repeated measures across all factors were then performed to search for significant main effects of N_2 and CO_2 on the primacy and recency portion of the recall curve separately for both immediate and delayed recall tests. *A priori* significance was set at the 0.05 level for all statistical tests.

Using the above statistical comparisons, performance impairment was analyzed into N_2 and CO_2 components to assess the effects of increasing the partial pressure of both N_2 and CO_2 simultaneously, and of increasing one or the other gas singly.

CHAPTER 4

RESULTS

4.1 CHARACTERISTICS OF SUBJECTS

Physical characteristics of the subjects are given in Table 4.1. The mean age and weight of the subject population was 25.6 ± 0.6 years and 76.4 ± 0.7kg respectively. Five subjects were certified sport divers with the minimum qualification under the sanction of the National Association of Underwater Instructors (NAUI). All subjects, apart from subjects 6, 10 and 12, had prior exposure to the compressed air environment in the hyperbaric chamber, at pressures in excess of 4 ATA, before volunteering to participate in the present study.

All compressions and decompressions proceeded without incident; however, a number of mild cases of "skin bends" were reported by several subjects and the diving tender after arrival at surface. The symptoms experienced were skin irritations, including blotchiness and itchiness, which disappeared within 60 minutes without treatment.

Common symptoms of mild CO_2 toxicity, including frontal headache and slight dizziness were also reported following rebreathing experiments under the medium and high hypercapnic stresses. Subjectively, these symptoms were reported to be the most severe following the highest hypercapnic level.

4.2 PHYSIOLOGICAL DATA

Group mean values and standard errors for respiratory responses to the three levels of hypercapnia at 1 and 6 ATA are shown in Table 4.2. All but the mean values for the non-rebreathing (low) end-tidal CO_2 tensions fell within the preset $P_{ET}CO_2$ ranges specified in chapter 2.

End-tidal CO_2 values in the non-rebreathing condition were very similar at surface and 6 ATA and were only 1 mmHg short of the $P_{ET}CO_2$ range specified for the normocapnic level. Again, there was little difference (approximately 1 mmHg) in the mean $P_{ET}CO_2$ values at surface and pressure for the medium level of hypercapnia. The mean value for end-tidal CO_2 under the high level of hypercapnia at 6 ATA, however, was approximately 3.5 mmHg greater than for the corresponding level of hypercapnia at surface.

Table 4.1: **Physical characteristics of subjects**

SUBJECT	Age (years)	Mass (kg)	Diver
1	35	84.0	N
2	22	79.5	N
3	28	73.6	Y
4	27	82.0	Y
5	31	86.6	N
6	22	75.0	N
7	24	75.0	Y
8	27	79.5	Y
9	24	73.5	Y
10	24	76.4	N
11	22	68.2	N
12	22	63.6	N
MEAN	25.6	76.4	
SEM	1.2	1.9	

Y = Yes, N = No

Inspired CO_2 tensions, on the other hand, demonstrated markedly different results at surface compared to depth (see Figure 4.1). In the non-rebreathing conditions there was little difference (approximatly 1 mmHg) in inspired CO_2 at surface and depth. Under the rebreathing conditions however, a greater level of P_ICO_2 was required to raise the $P_{ET}CO_2$ to the required ranges at 1 ATA than during the 6 ATA trials. Interestingly, Figure 4.1 indicates a greater positive slope for the P_ICO_2 curve at 1 ATA than for the curve at 6 ATA. Statistical analysis revealed the interaction between $P_{ET}CO_2$ and P_ICO_2 was significant ($F= 32.97$; $df=2, 22$; $p<0.05$), indicating a synergistic relationship between these two variables.

As the levels of hypercapnia increased, inspired minute ventilation rose uniformly at surface and depth; however, this increase was much greater at 1 ATA than at 6 ATA ($F= 16.51$; $df= 2, 22$; $p<0.05$) (Figure 4.2). At 6 ATA an overall significant hypoventilation was observed compared to the normal ventilatory responses to CO_2 at surface pressures ($F= 9.84$; $df= 1, 11$; $p<0.05$). Although respiratory rates

increased with increases in $P_{ET}CO_2$, they were 18% lower at 6 ATA throughout the entire range of hypercapnic conditions (see Figure 4.3A). Correspondingly, for a given ventilation, the mean tidal volume was greater at depth compared to surface. (see Figure 4.3B).

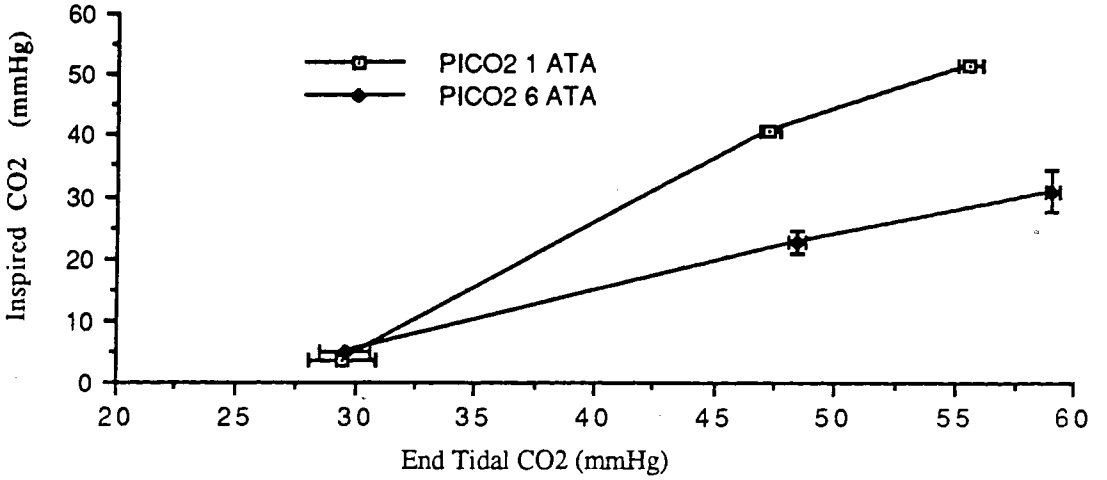


Figure 4.1: Inspired CO_2 vs. end-tidal CO_2 at 1 ATA and 6 ATA during CO_2 rebreathing of various hypercapnic levels in a (dry) hyperbaric pressure chamber. Values are means (n=12); bars represent SEM.

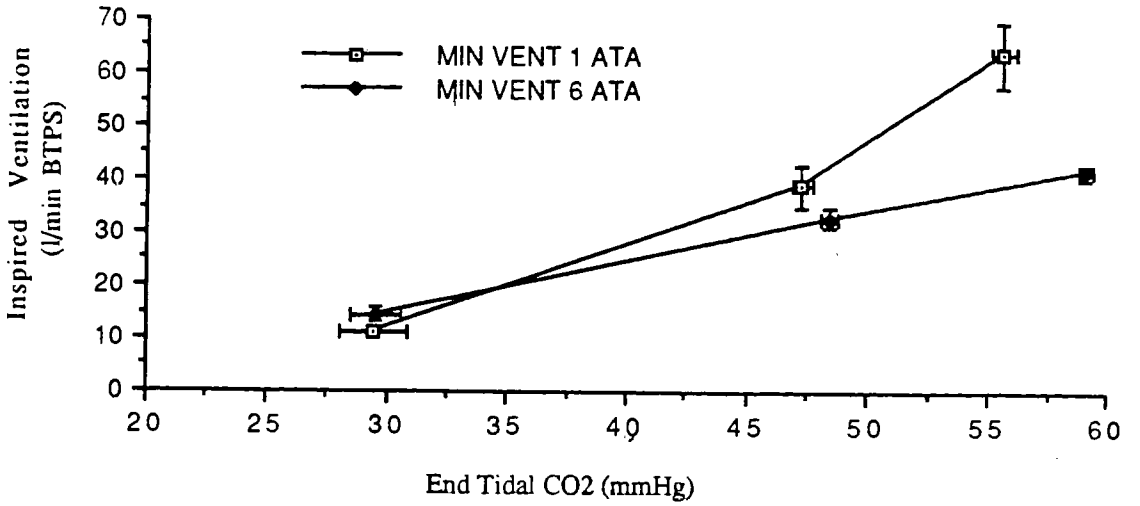


Figure 4.2: Ventilatory responses (inspired minute ventilation) to increasing $P_{ET}CO_2$ tensions at 1 and 6 ATA during CO_2 rebreathing in a (dry) hyperbaric pressure chamber. Values are means (n=12); bars represent SEM.

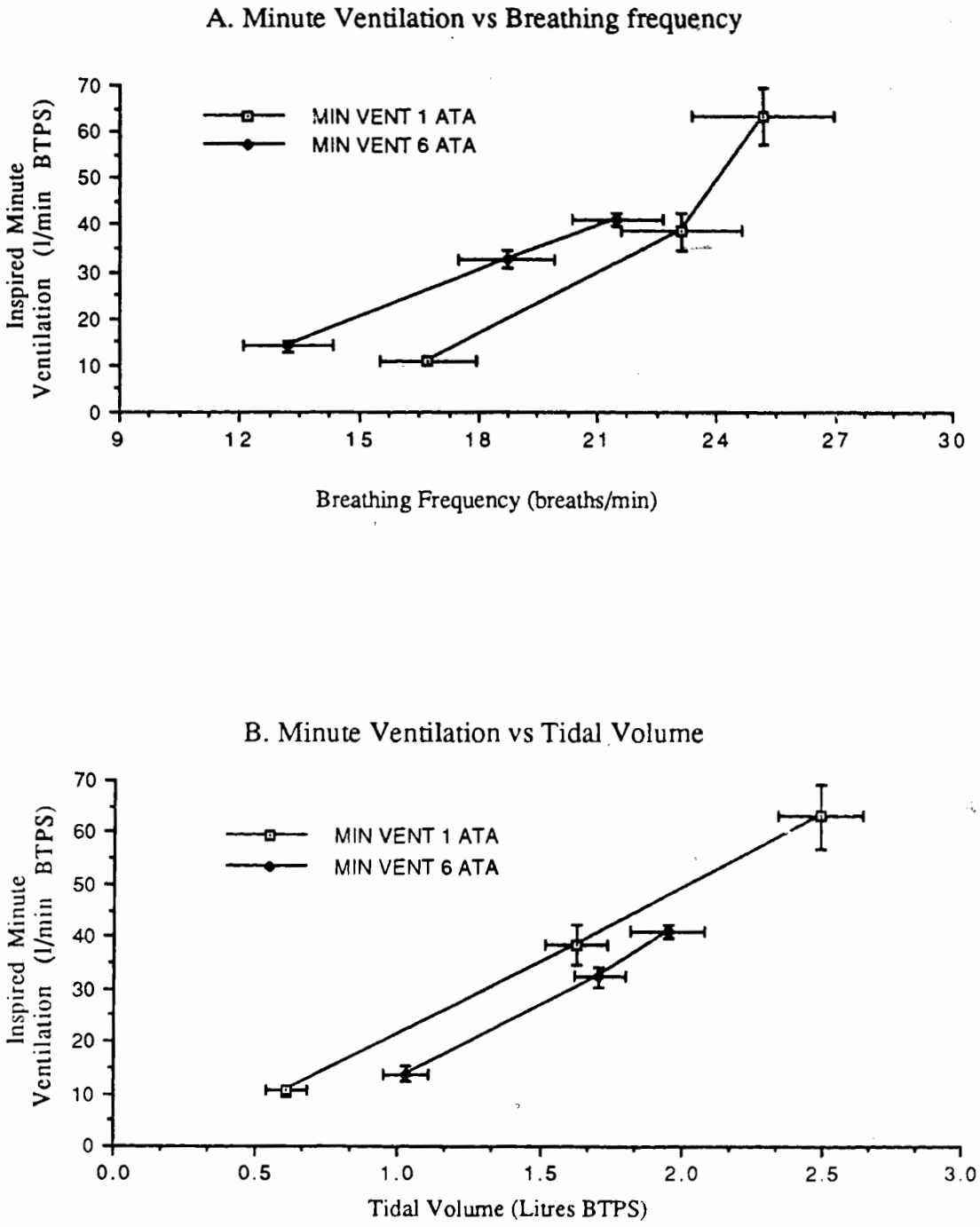


Figure 4.3: Inspired minute ventilation vs breathing frequency (A) and tidal volume (B) at 1 ATA and 6 ATA during CO_2 rebreathing in a (dry) hyperbaric pressure chamber. Values are means (n=12); bars represent SEM.

Table 4.2: **Group mean values and standard errors for respiratory responses to three levels of hypercapnia (induced by CO_2 rebreathing) at 1 ATA and 6 ATA in a hyperbaric (dry) pressure chamber. (n=12).**

	1 ATA			6 ATA		
	Level of Hypercapnia					
	LOW	MEDIUM	HIGH	LOW	MEDIUM	HIGH
$P_{ET}CO_2$ (mmHg)						
MEAN	29.00	46.83	55.13	29.09	47.92	58.62
SEM	1.42	0.42	0.54	1.05	0.33	0.31
P_ICO_2 (mmHg)						
MEAN	2.00	38.61	49.67	3.03	20.06	28.97
SEM	0.42	0.73	0.97	0.22	1.75	3.40
Respiratory rate (breaths/min.)						
MEAN	16.54	22.93	24.96	13.03	18.53	21.33
SEM	1.21	1.51	1.80	1.13	1.21	1.13
$\dot{V}_I$ (litres/min BTPS)						
MEAN	8.90	36.71	61.37	12.16	30.56	39.27
SEM	1.09	3.89	6.22	1.29	1.84	1.29
V_T (litres, BTPS)						
MEAN	0.58	1.60	2.46	1.00	1.68	1.93
SEM	0.07	0.11	0.15	0.08	0.09	0.13

4.3 PSYCHOLOGICAL DATA

4.3.1 Test- Retest Reliabilities of the Performance Tasks in the Test Battery

Table 4.3 summarizes the results of the test-retest reliability for each test on the test battery. The reliability coefficients were computed from a comparison of the scores (number correct) between the penultimate and final pre-test practice trials. Scores for immediate and delayed recall presented in Table 4.3 were computed from the total number of word/number associations correctly recalled, for each subject, on each of the final two pre-test practice trials, irrespective of word/number position.

Most tests demonstrated acceptable test-retest correlation coefficients indicating their reasonable reliability. Apart from the copying test, the estimated test-retest accuracy of the psychological tests fell between 70 and 80%.

During the initial block practice session, attempted and correct performance scores showed a large improvement, while error scores were high and demonstrated a wide variability among subjects. Prior to the experimental trials, subjects were given feedback on their performance scores following the block practice session. This served to reduce the error score and error variance quite considerably for successive practice trials on the individual tests. Figures showing progression of the performance scores on individual tests in the test battery, throughout the block practice sessions and the six pre-trial practice sessions, are presented in Appendix 5.

Although correct scores on most tests were on a plateau of performance by the final block practice trial, some learning was still evident across experimental trials. Learning/practice effects were estimated from linear regression curves fitted to the pre-test practice trial data (practice trials 6 to 11). Percent increases in performance across these trials were calculated from differences in the correct score between the first and last pre-test practice trial (scores being derived from the regression equations) (see Table 4.3).

Improvement due to learning ranged from 3% for the copying task to 30% for immediate recall between the first and last pre-test practice trial. These learning effects were, however, accounted for through a fully balanced, randomized presentation of the three hypercapnic levels and two pressure levels, across the subject population.

Table 4.3: **Test-retest reliability coefficients and learning effects for performance on the tasks included in the test battery (n=12). Performance measures are the mean number of correct responses/scores for each test.**

Performance Test	Practice Trial 10 (SCORE) MEAN±SEM	Practice Trial 11 (SCORE) MEAN±SEM	r	r^2	Percent learning increase
Copy	82.75 ±3.87	84.08 ±3.30	0.75*	0.57	3
Math	19.50 ±2.17	26.08 ±2.28	0.88*	0.77	18
Ncomp	14.92 ±1.03	13.83 ±0.63	0.87*	0.76	16
Stroop	143.08 ±9.03	142.83 ±5.46	0.85*	0.73	10
Purdue	47.91 ±1.75	48.83 ±1.50	0.83*	0.70	15
IR	7.42 ±0.81	7.46 ±0.88	0.84*	0.72	30
DR	5.92 ±1.04	5.67 ±0.94	0.89*	0.79	26

Abbreviations: IR = Immediate recall, DR = Delayed recall, Ncomp = Number comparison test.

* $p<0.05$

r = Pearson product moment correlation coefficient (critical value = 0.576, df=10, alpha=0.05).

r^2 = Coefficient of determination.

Learning effects were estimated from linear regression curves fitted to the pre-test practice trial data (practice trials 6 to 11). Percent increases in performance over these practice trials were calculated from the correct score difference, between first and last pre-test practice trial, derived from the regression equations.

4.3.2 Performance Test Scores

Mean performance test scores for each task on the test battery, under all experimental conditions, are shown in Table 4.4. The performance scores for each task were compared with their respective control values at 1 ATA under the non-rebreathing condition (column 1, Table 4.4). The resulting mean percentage change in performance scores for the different $P_{ET}CO_2$ tensions at each pressure level are illustrated in Table 4.5. This Table also shows the results of the Tukey's Post hoc analysis following significant main effects for $P_{ET}CO_2$ on the two-way ANOVA.

No interaction was found between P_IN_2 and $P_{ET}CO_2$ ($p>0.05$) on any of the performance scores which demonstrated a significant main effect for CO_2. Consequently, for the purpose of Post hoc analysis, the end-tidal CO_2 dependent measures were collapsed over the two levels of P_IN_2. Thus the significant differences shown in Table 4.5 for the 6 ATA data represent significant changes in scores for the combined CO_2 data at 1 and 6 ATA.

Tables 4.6A and B provide summary statistics for the main effect of P_IN_2 on performance scores at the two pressure levels. Raw analysis of variance results for $P_{ET}CO_2$ and interaction effects, along with Post hoc analysis martrices Tables are presented in Appendix 6. The outcome of this analysis, with reference to the above cited Tables, is presented for each performance test separately below.

4.3.3 Copying Test

Figures 4.4A, B and C denote the relationship between performance scores on the Copying test and the $P_{ET}CO_2$ tensions at each pressure level. A significant main effect of end-tidal CO_2 on the number/portion of patterns attempted ($F= 13.53$; $df= 2, 22$; $p<0.05$) and the total number correct ($F= 14.28$; $df= 2, 22$; $p<0.05$) was evident following the two-way analysis of variance. Post hoc analysis revealed statistically significant deteriorations on these performance scores between the non-rebreathing condition and the medium and high $P_{ET}CO_2$ tensions at both pressure levels ($p<0.05$). While the number of errors on the copying test was unaffected by CO_2 ($F= 1.57$; $df= 2, 22$; $p>0.05$), inspired N_2 on the other hand demonstrated a statistically significant influence on the error score ($F= 18.17$; $df= 1, 11$; $p<0.05$). At 6 ATA the number of errors increased on average 102 percent over performance on the copying test at 1 ATA. In contrast, inspired N_2 had no effect on the number of figures attempted ($F= 0.31$; $df= 1, 11$; $p>0.05$). An insignificant interaction between the P_IN_2 and $P_{ET}CO_2$ was found for all performance scores on the copying test (attempted $F= 0.06$; error $F= 0.29$; correct $F= 0.11$; $df= 2, 22$; $p>0.05$).

Table 4.4: **Effects of end-tidal PCO_2 on performance test scores while breathing air at 1 ATA and 6 ATA. Mean values ± S.E.M. (n=12).**

	1 ATA			6 ATA		
	Mean End-tidal CO_2 tensions (mmHg)					
	LOW 29.0	MEDIUM 46.8	HIGH 55.1	LOW 29.1	MEDIUM 47.9	HIGH 58.6
Copying Test						
Attempted	89.1 ±5.3	81.3 ±5.8	76.9 ±5.6	88.1 ±4.6	79.4 ±4.2	74.0 ±5.0
Errors	3.9 ±0.7	3.1 ±0.8	4.3 ±0.7	7.1 ±1.2	7.4 ±1.6	8.2 ±1.1
Correct	85.5 ±5.5	78.3 ±5.9	72.7 ±5.4	80.8 ±4.9	71.2 ±4.0	65.8 ±4.5
Math Test						
Attempted	23.0 ±2.2	21.0 ±2.1	18.8 ±2.0	19.8 ±1.9	18.8 ±2.1	14.4 ±1.9
Errors	2.7 ±0.5	2.0 ±0.7	2.8 ±0.7	2.7 ±0.6	3.7 ±0.6	3.3 ±0.4
Correct	20.8 ±2.3	19.0 ±1.9	16.0 ±2.0	17.6 ±2.2	15.1 ±2.2	11.1 ±2.0
NComp Test						
Attempted	14.3 ±0.5	13.8 ±0.7	12.7 ±0.7	13.9 ±0.7	13.4 ±0.8	12.8 ±0.9
Errors	0.3 ±0.1	0.7 ±0.3	0.3 ±0.2	1.0 ±0.4	0.8 ±0.3	1.6 ±0.4
Correct	13.9 ±0.5	13.1 ±0.7	12.3 ±0.6	12.9 ±0.6	12.5 ±0.9	11.3 ±0.8
Stroop Test						
Attempted	145.5 ±9.2	137.1 ±9.5	126.1 ±7.3	135.9 ±7.4	120.2 ±6.7	109.8 ±5.2
Errors	1.8 ±0.8	2.1 ±0.8	1.6 ±0.8	2.2 ±0.9	3.5 ±1.6	4.6 ±1.9
Correct	143.8 ±9.3	135.0 ±9.5	124.5 ±7.7	133.8 ±7.2	118.2 ±7.6	105.3 ±6.1
IR (No. of Corr. recalls)	6.9 ±0.8	5.4 ±0.7	5.3 ±0.9	4.7 ±0.9	4.1 ±0.8	2.5 ±0.5
DR(No. of Corr. recalls)	5.5 ±0.9	4.4 ±0.9	4.2 ±1.0	4.0 ±1.0	3.2 ±1.0	2.0 ±0.6
Purdue Pegboard (Prts assbld/min)	48.1 ±1.4	46.6 ±1.7	42.0 ±1.1	44.5 ±1.6	42.2 ±1.9	38.2 ±1.6

Abbreviations: IR = Immediate recall, DR = Delayed recall, Ncomp = Number comparison test.

Table 4.5: **Mean percentage change in performance scores as compared to control values at 1 ATA** (*cf.*, Table 4.4, 1st column).

	1 ATA		6 ATA		
	Mean End-tidal CO_2 tension (mmHg)				
	MEDIUM 46.8	HIGH 55.1	LOW 29.1	MEDIUM 47.9	HIGH 58.6
Copying Test					
Attempted	-8.7	-13.7	-1.1	-10.9	-16.9
Errors	-20.5	+10.3	+8.2	+89.7	+110.3
Correct	-8.4	-15.0	-5.5	-16.7	-23.0
Math Test					
Attempted	-8.7	-18.3	-13.9	-18.3	-37.4
Errors	-25.9	+3.7	0.0	+37.0	+22.2
Correct	-8.7	-23.1	-15.4	-27.4	-46.6
NComp Test					
Attempted	-3.5	-11.1	-2.8	-6.3	-10.5
Errors	+133.0	0.0	+233.0	+166.7	+433.3
Correct	-5.8	-11.5	-7.2	-10.1	-18.7
Stroop Test					
Attempted	-5.8	-13.3	-6.6	-17.4	-24.6
Errors	+16.7	-11.1	+22.2	+94.4	+155.6
Correct	-6.1	-13.4	-7.0	-17.8	-26.8
IR (No. of Corr recalls)	-21.7	-23.2	-31.1	-40.6	-63.8
DR(No. of Corr. recalls)	-20.0	-23.6	-27.3	-48.1	-63.6
Purdue Pegboard (Prts assbld/min)	-3.1	-12.7	-7.5	-12.3	-20.6

•
†
‡

Underscores represent those performance values significantly different ($p<0.05$) between: LOW and MEDIUM (•), MEDIUM and HIGH (†) and, LOW and HIGH (‡) hypercapnic conditions following Post hoc analysis. For calrity Post hoc analysis is shown only on the 6 ATA data. This analysis represents the CO_2 data collapsed across both pressure levels (see text for details).
Abbreviations: as for Table 4.4.

Table 4.6A: **Summary statistics for the mean percent decrement in performance scores between 1 and 6 ATA on the individual tasks in the test battery. (Data collapsed across the CO_2 levels).**

Performance Test	Percent Decrement	MSE	F	p	
Copying Test (Correct score)	8	273.46	2.55	0.1360	NS
Math Test (Correct score)	22	10.98	26.59	0.0005	*
NComp Test (Correct score)	7	4.68	3.04	0.1063	NS
Stroop Test (Correct score)	11	236.95	17.92	0.0017	*
IR (Total score)	36	1.37	58.40	0.0000	*
(Primacy)	26	0.98	4.08	0.0660	NS
(Recency)	35	0.35	14.44	0.0032	*
DR (Total score)	35	2.01	24.01	0.0007	*
(Primacy)	29	0.50	9.00	0.0117	*
(Recency)	31	0.26	2.66	0.1287	NS
Purdue Pegboard (Prts assbld/min)	9	17.87	15.67	0.0025	*

df = 1, 11
n = 12
* $p<0.05$
Abbreviations: IR = Immediate recall, DR = Delayed recall, Ncomp = Number comparison test.
NS = Not significant.
MSE = Mean square of the error term (within-group variance estimate).

Table 4.6B: **Summary statistics for the mean percent change in performance scores between 1 and 6 ATA on the cognitive tasks in the test battery. (Data collapsed across the CO_2 levels).**

Performance Test	Percent Change	MSE	F	p	
Copying Test					
(Attempted score)	−2	220.74	0.31	0.5900	NS
(Error score)	+102	14.35	18.17	0.0016	•
Math Test					
(Attempted score)	−16	6.63	29.16	0.0004	•
(Error score)	+40	4.79	3.16	0.1005	NS
NComp Test					
(Attempted score)	−1	6.29	0.08	0.7733	NS
(Error score)	+152	0.79	10.15	0.0086	•
Stroop Test					
(Attempted score)	−10	223.91	16.32	0.0022	•
(Error score)	+89	6.18	7.56	0.0181	•

df = 1, 11
n = 12
• $p<0.05$
Abbreviations: IR = Immediate recall, DR = Delayed recall, Ncomp = Number comparison test.
NS = Not significant.
MSE = Mean square of the error term (within-group variance estimate).

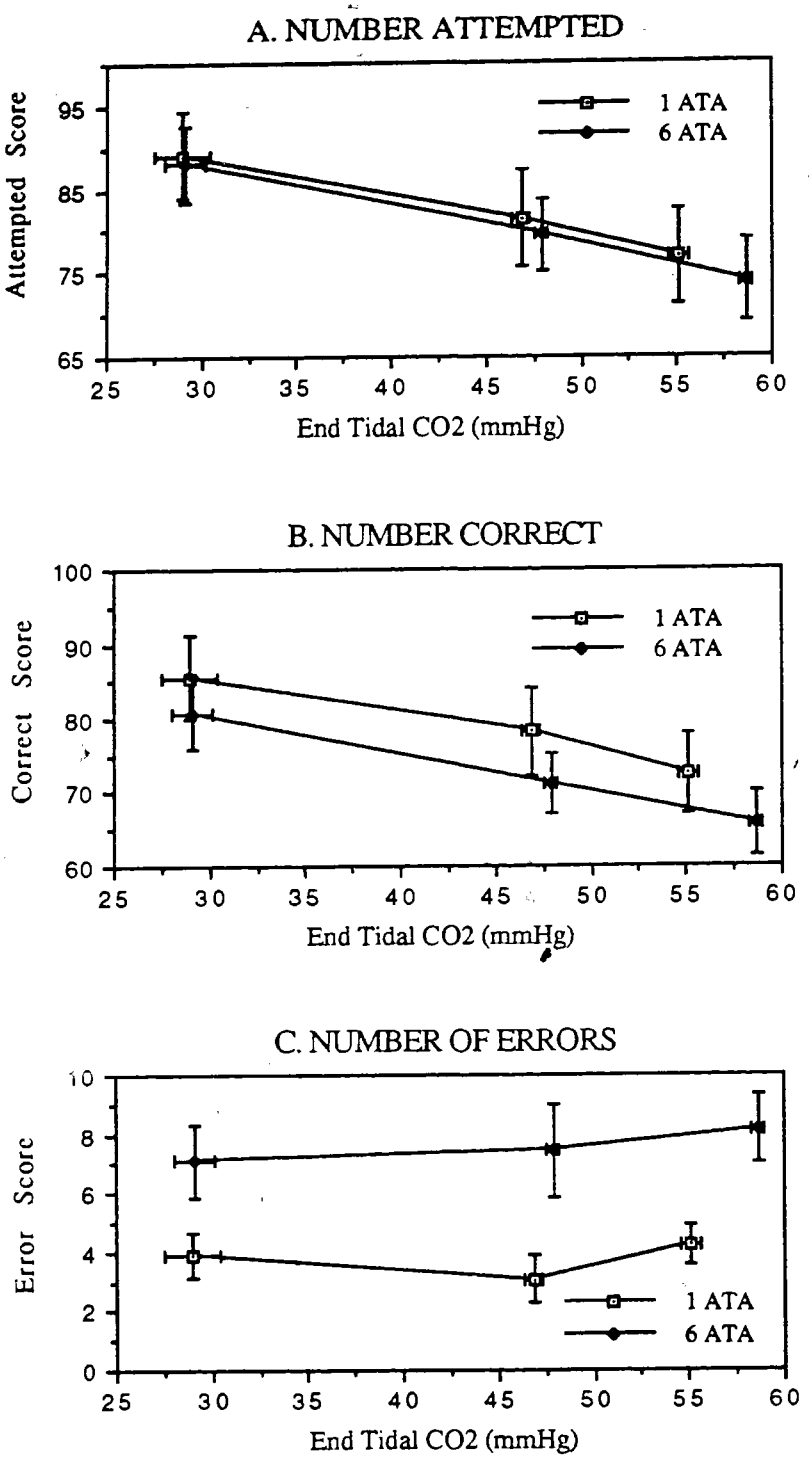


Figure 4.4. Separate and combined effects of changes in P_IN_2 and $P_{ET}CO_2$ on performance scores in the Copying test. A: Number/portion of patterns attempted. B: Number/portion of patterns correct. C: Number of errors. Bars represent SEM (n=12).

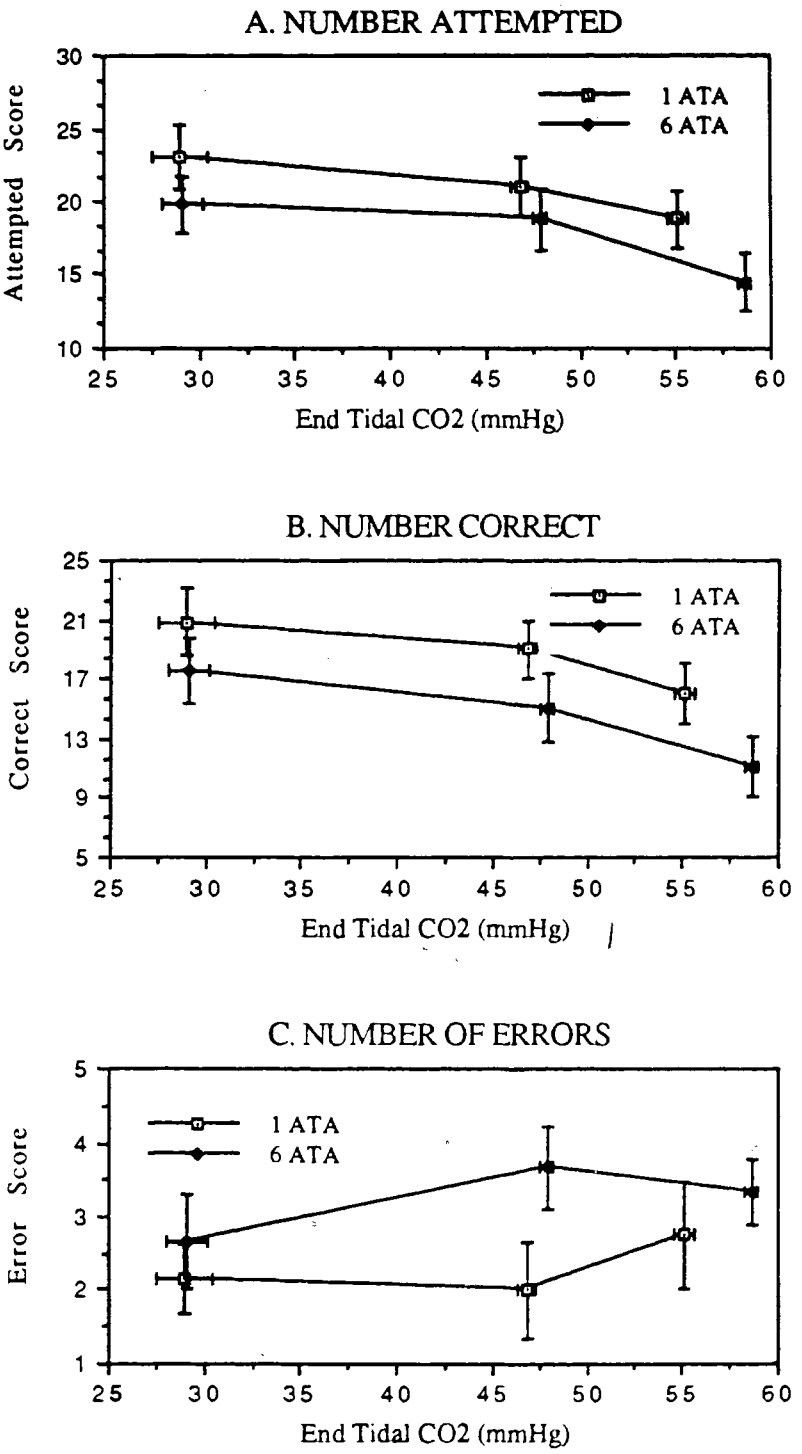


Figure 4.5. Separate and combined effects of changes in P_IN_2 and P_ICO_2 on performance scores in the Math test. A: Number of problems attempted. B: Number of problems correct. C: Number of incorrectly solved problems. Bars represent SEM (n=12).

4.3.4 Math Test

The overall relationship between end-tidal CO_2 and the individual performance scores on the Math test at each pressure level are shown in Figures 4.5 A, B and C. At 6 ATA the number of problems attempted dropped significantly by an average of 16 percent compared to the mean number of problems attempted at 1 ATA ($F= 29.16$; $df= 1, 11$; $p<0.05$). This contributed to a statistically significant 22 percent reduction in the number of problems correctly solved at 6 ATA ($F= 26.59$; $df= 1, 11$; $p<0.05$). Both the inspired N_2 and end-tidal CO_2 had no effect on the number of errors generated on the Math test ($F(P_IN_2)= 0.78$; $df= 1, 11$; $p>0.05$; $F(P_{ET}CO_2)= 3.15$; $df= 2, 22$; $p>0.05$;).

A significant main effect of $P_{ET}CO_2$ was found for both the number of problems attempted ($F= 26.07$; $df= 2, 22$; $p<0.05$) and the number of problems correct ($F= 20.31$; $df= 2, 22$; $p<0.05$). Post hoc analysis indicated the statistically significant decrements on the attempted and correct score occurred between the no-rebreathing condition and the high $P_{ET}CO_2$ tensions as well as between the medium and high $P_{ET}CO_2$ tensions at both pressure levels ($p<0.05$). Again, all performance tests indicated the absence of an interaction between inspired N_2 and end-tidal CO_2 (attempted $F= 0.39$; error $F= 1.28$; correct $F= 0.21$; $df= 2, 22$; $p>0.05$).

4.3.5 Number Comparison Test

The relationship between the performance scores on the Number Comparison test and the $P_{ET}CO_2$ tension for each pressure level are revealed in Figures 4.6 A, B and C. A significant main effect of end-tidal CO_2 on the number of comparisons attempted ($F= 4.39$; $df= 2, 22$; $p<0.05$) as well as the number correct ($F= 4.99$; $df 2, 22$; $p<0.05$) were found following the two-way analysis of variance. Post hoc analysis revealed a statistically significant decrement for the attempted and correct scores only under the high $P_{ET}CO_2$ tensions at each pressure level when compared to the non-reabreathing conditions ($p<0.05$). When the P_IN_2 was raised from 0.79 ATA to 4.74 ATA there was little effect on the number of correct comparisons made ($F= 3.04$; $df= 1, 11$; $p>0.05$) and number attempted ($F= 0.08$; $df= 1, 11$; $p>0.05$); however, the number of incorrect comparisons at 6 ATA increased significantly, on average 152 percent, compared to error scores at 1 ATA ($F= 10.15$; $df= 1, 11$; $p<0.05$). The error score on the number comparison test was the only performance score on the whole test battery that indicated a statistically significant interaction between inspired N_2 and end-tidal CO_2 ($F= 3.99$; $df= 2, 22$; $p<0.05$).

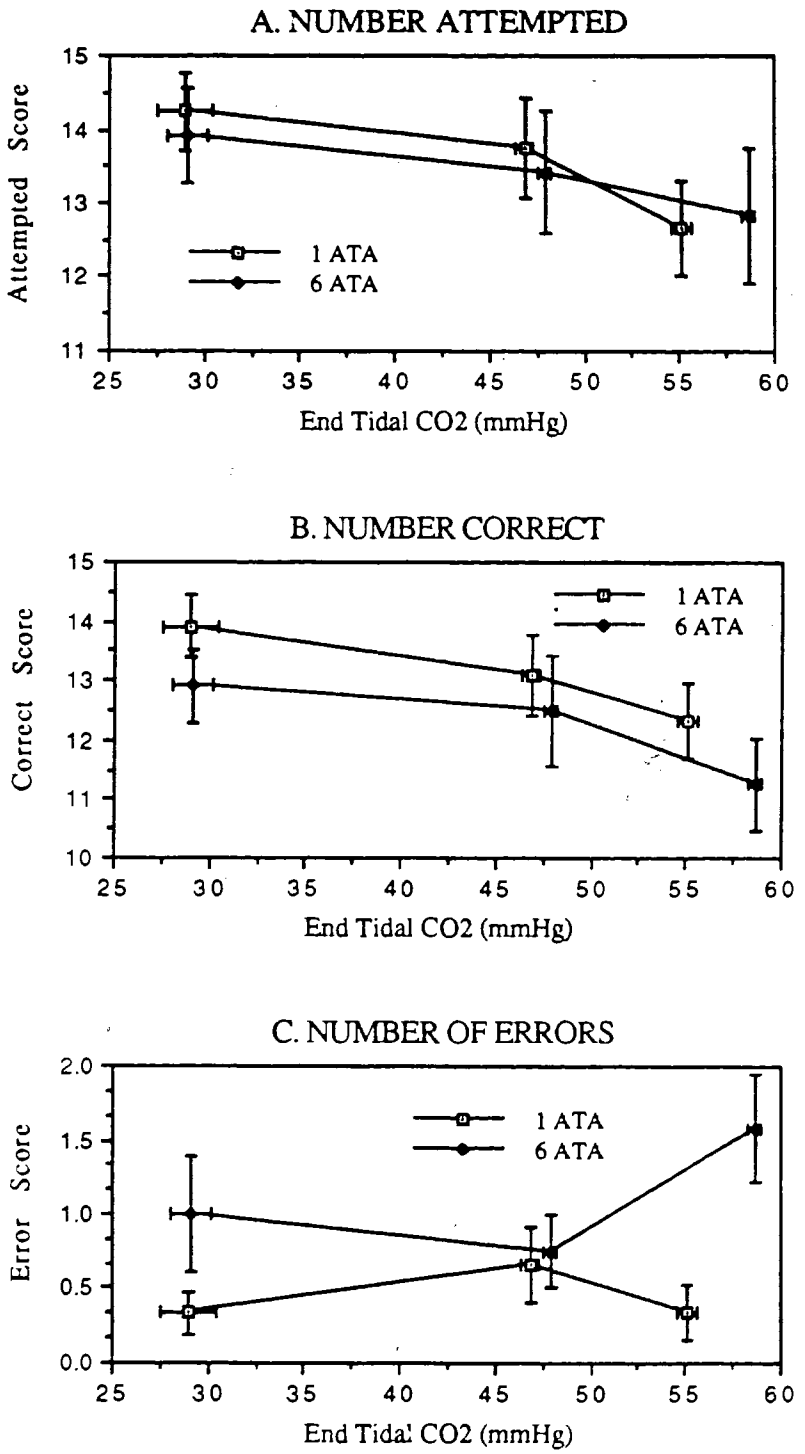


Figure 4.6. Separate and combined effects of changes in P_IN_2 and $P_{ET}CO_2$ on performance scores in the Number comparison test. A: Number attempted. B: Number correct. C: Number of errors. Bars represent SEM (n=12).

4.3.6 Stroop Test

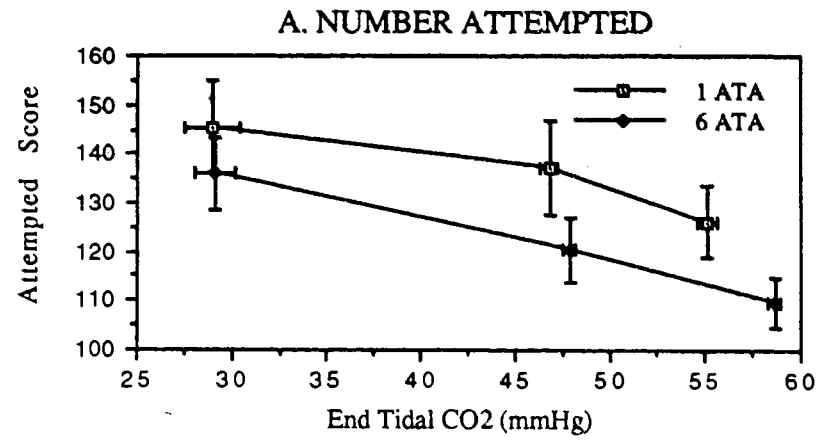
Figures 4.7 A, B and C indicate the relationship between end-tidal CO_2 and the performance scores on the Stroop test. A significant main effect of CO_2 was revealed for the attempted score ($F= 14.14$; $df= 2, 22$; $p<0.05$) as well as for the number of letters correctly crossed out ($F= 12.65$; $df= 2, 22$; $p<0.05$). Post hoc analysis indicated that for both these performance scores statistically significant decrements ($p<0.05$) occured when the $P_{ET}CO_2$ was raised from resting tensions to approximatly 47 mmHg at both pressure levels.

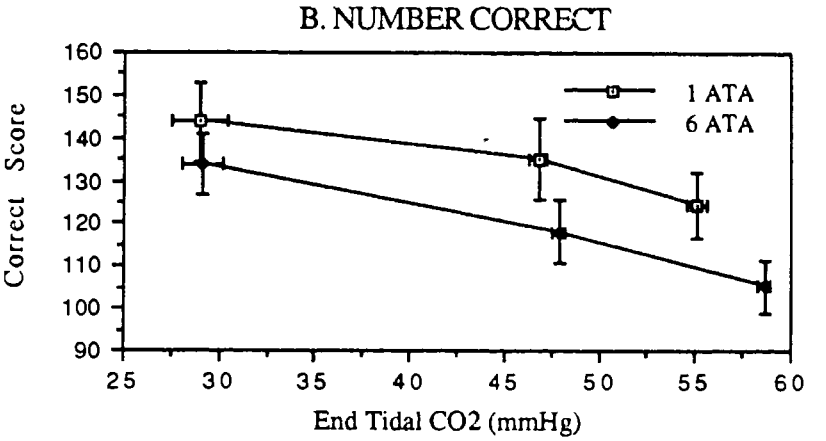
On average, the number of letters crossed out decreased by 10 percent ($F= 16.32$; $df= 1, 11$; $p<0.05$), the number correct by 11 percent ($F= 17.92$; $df= 1, 11$; $p<0.05$) and the number of errors increased by 90 percent ($F= 7.56$; $df= 1, 11$; $p<0.05$) when the ambient pressure was raised from 1 ATA to 6 ATA. Neither attempted, correct nor error scores exhibited a statistically significant interaction between inspired N_2 and end-tidal CO_2 (attempted $F= 0.82$; error $F= 0.60$; correct $F= 0.87$; $df= 2, 22$; $p>0.05$).

4.3.7 Purdue Pegboard

Analysis of variance displayed significant main effects for both $P_{ET}CO_2$ ($F= 17.14$; $df= 2, 22$; $p<0.05$) and P_IN_2 ($F= 15.67$; $df= 1, 11$; $p<0.05$) on the number/parts assembled on the Purdue pegboard task. On average the number of parts assembled in 1 minute decreased by 9 percent when the ambient pressure was raised from 1 ATA to 6 ATA. When the $P_{ET}CO_2$ was raised at a constant ambient pressure there was a gradual decline in the number of assemblies completed at each pressure level with increasing $P_{ET}CO_2$ tensions. Post hoc analysis revealed the main statistically significant effects on performance ($p<0.05$) occured when the $P_{ET}CO_2$ tension was raised to 55 mmHg at each pressure level. In addition, scores on this task were statistically significantly lower at the high level of $P_{ET}CO_2$ when compared to performance under the medium level of hypercapnia ($p<0.05$).

The relationship between the number of parts assembled and the $P_{ET}CO_2$ at each pressure level is presented in Figure 4.8. The two sets of scores obtained at 1 ATA and 6 ATA respectively, show a progressive and almost parallel decrease as the $P_{ET}CO_2$ tension rises. This was evident from the analysis of variance which indicated a statistically insignificant F ratio for the interaction effect on the number of assemblies constructed in one minute ($F= 0.08$; $df= 2, 22$; $p>0.05$).





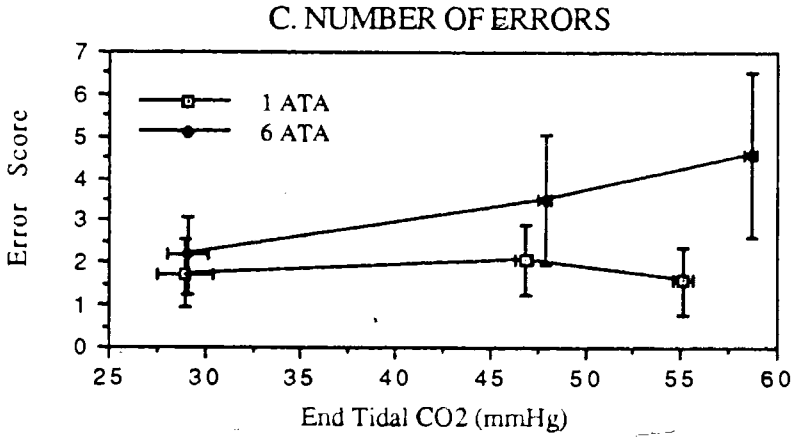


Figure 4.7. Separate and combined effects of changes in P_IN_2 and $P_{ET}CO_2$ on performance scores in the Stroop test. A: Number of letters attempted. B: Number of letters correctly crossed out. C: Number of letters incorrect crossed out. Bars represent SEM (n=12).

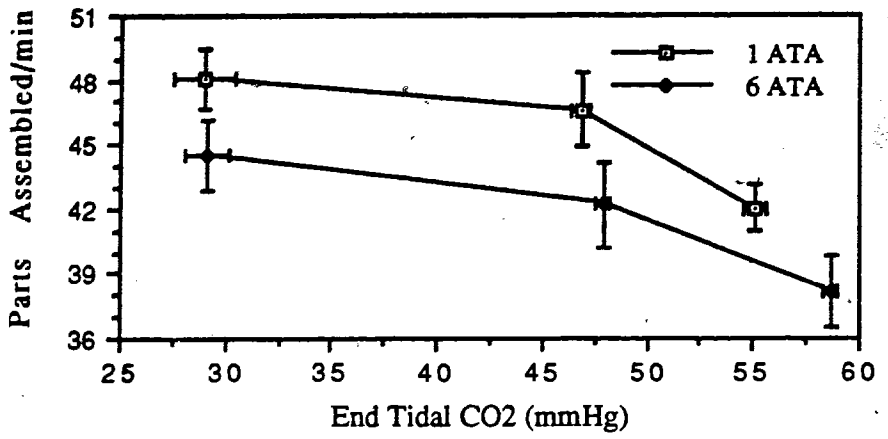


Figure 4.8. **Separate and combined effects of changes in P_IN_2 and $P_{ET}CO_2$ on performance on the Purdue pegboard. Bars represent SEM (n=12).**

4.3.8 Immediate Recall

When the ambient pressure was raised from 1 to 6 ATA the total number of word/number pairs correct following immediate recall fell on average 36 percent (approximatly 2 word/number pairs) ($F= 58.40$; $df= 1, 11$; $p<0.05$) (see the vertical distance between curves in Figure 4.9A). After subjecting the primary and recency responses on the serial word/number curve to a separate analysis of variance, a significant main effect of P_IN_2 was found on the recency portion of the curve ($F= 14.44$; $df= 1, 11$; $p<0.05$), but not on the primacy ($F= 4.08$; $df= 1, 11$; $p>0.05$). These results indicate that recall of the first few word/number pairs was unaffected at the higher ambient pressure whereas recall of the last two word/number pairs was significantly hindered at 6 ATA.

Although increasing $P_{ET}CO_2$ tensions produced a significant decrease in the total number of word/number pairs remembered correctly during immediate recall ($F= 4.87$; $df= 2, 22$; $p<0.05$), this decrement could not be be attributed solely to an effect on the primacy ($F= 0.73$; $df= 2, 22$; $p>0.05$) or recency ($F= 1.55$, $df= 2, 22$; $p>0.05$) portion of the curve. This is illustrated in Figures 4.10A and B, where the probability of recall for a particular word/number position is plotted for the three levels of $P_{ET}CO_2$ at

surface and depth respectively. Analysis of variance also indicated an insignificant interaction between P_IN_2 and $P_{ET}CO_2$ tensions on the total number of word/number pairs recalled correctly (F= 0.69; df= 2, 22; p>0.05).

4.3.9 Delayed Recall

Delayed recall of the word/number pairs was administered 15 minutes after initial presentation of the word/number list. At this point during the experiment the subject was removed from the rebreathing apparatus and had been breathing atmospheric air containing normal tensions of inspired CO_2 for at least 5 minutes. During the 6 ATA conditions delayed recall was administered at the first decompression stop (approximately 12 msw following a 20 minute dive schedule at 51 msw). Consequently, delayed recall following experimental trials was administered under lower P_IN_2 tensions at non-narcotic pressures.

Although there was a statistically insignificant effect of end-tidal CO_2 on the total number of word/number pairs recalled during delayed recall (F= 3.36; df= 2, 22; p>0.05), performance was significantly reduced following the 6 ATA dives (F= 24.00; df= 1, 11; p<0.05) (see Figure 4.9B). This decrement occured predominantly in the primacy portion of the word/number series curve (F= 9.00; df= 1, 11; p<0.05) with little effect on the recency portion of the curve (F= 2.66; df= 1, 11; p>0.05). (see figures 4.11A and 4.11B) Again there was no interaction between P_IN_2 and $P_{ET}CO_2$ tensions (F= 0.38; df= 2, 22; p>0.05).

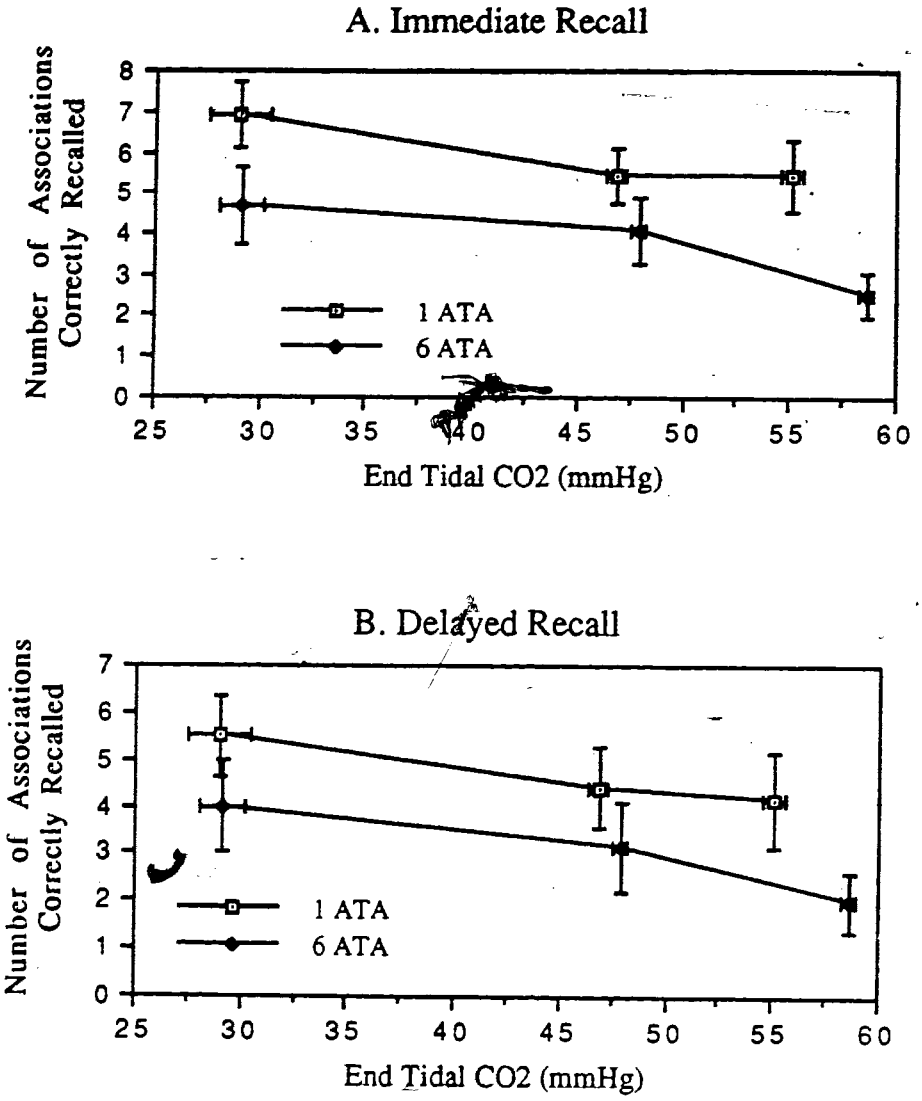
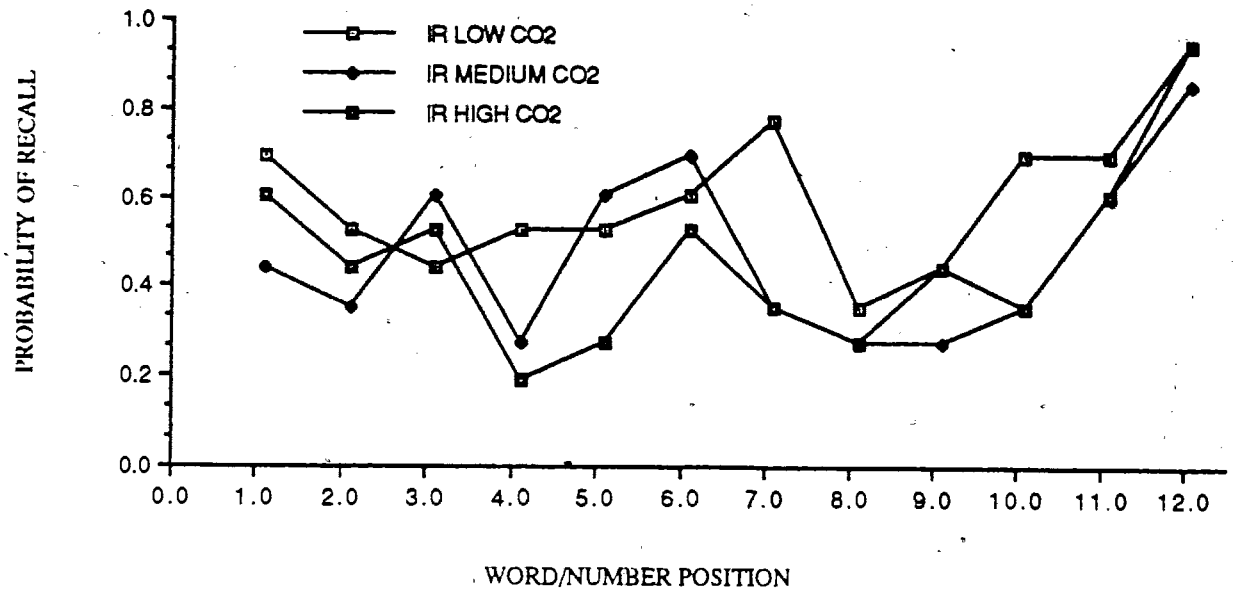


Figure 4.9. **Separate and combined effects of changes in P_IN_2 and $P_{ET}CO_2$ on immediate recall (A) and delayed recall (B) Bars represent SEM (n=12).**

A. IMMEDIATE RECALL SURFACE



B. IMMEDIATE RECALL 6 ATA

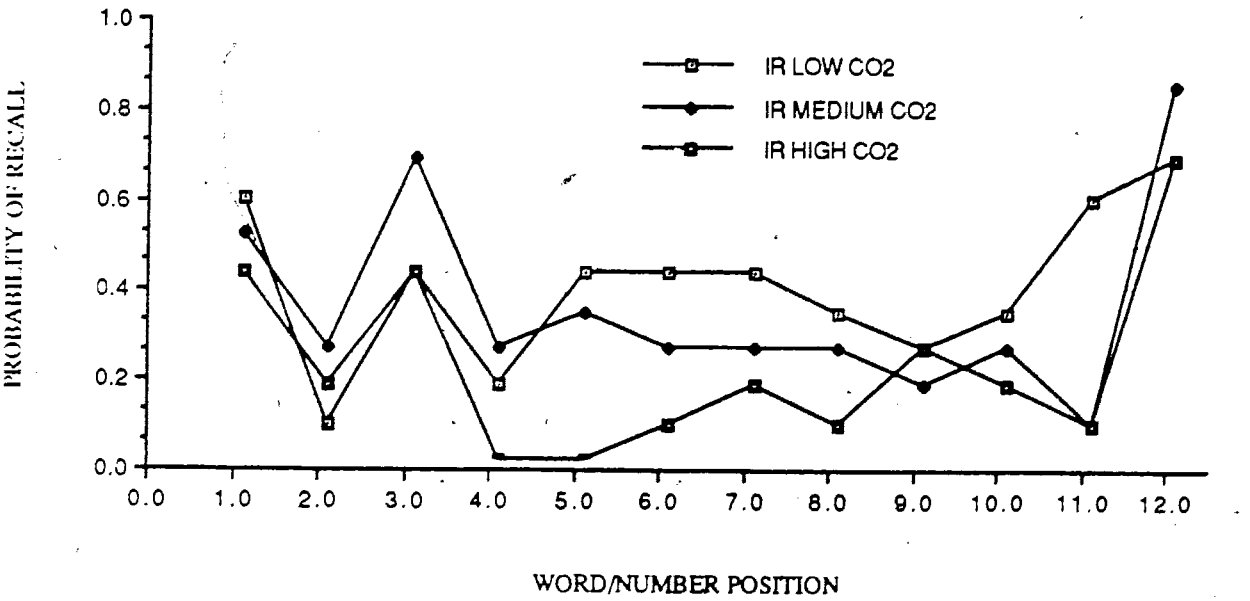
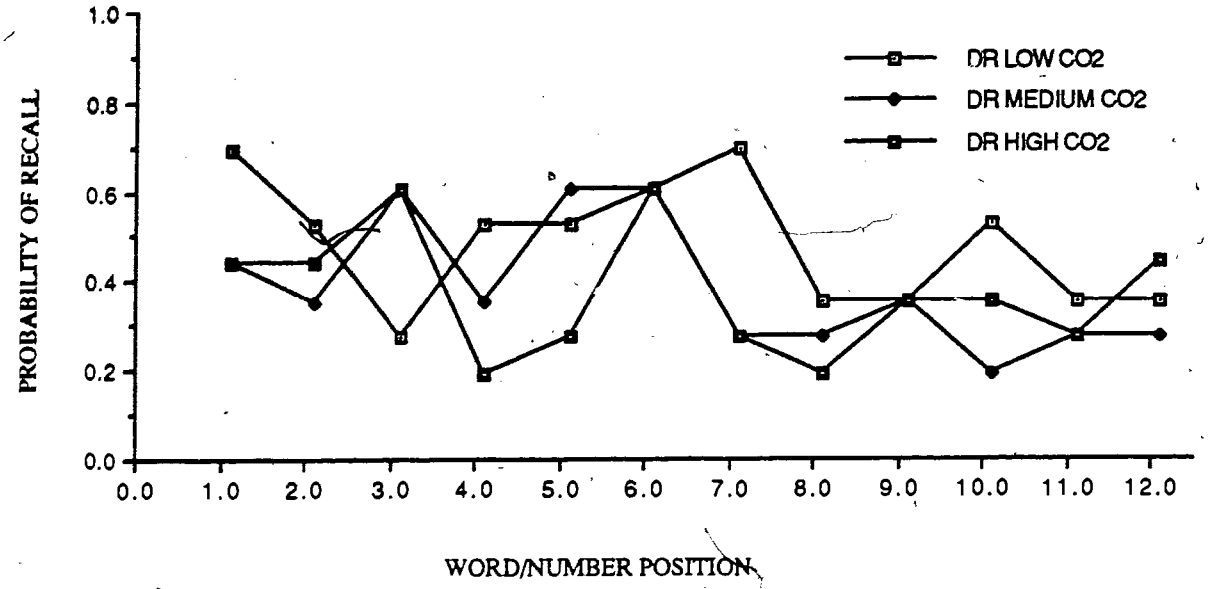


Figure 4.10. **Probability of immediate recall as a function of serial position on the paired association memory task while exposed to low, medium and high PCO_2 tensions at 1 ATA (A), and 6 ATA (B) of pressure. Error bars are ommited for the sake of clarity. (n=12).**

A. DELAYED RECALL SURFACE



B. DELAYED RECALL 6 ATA

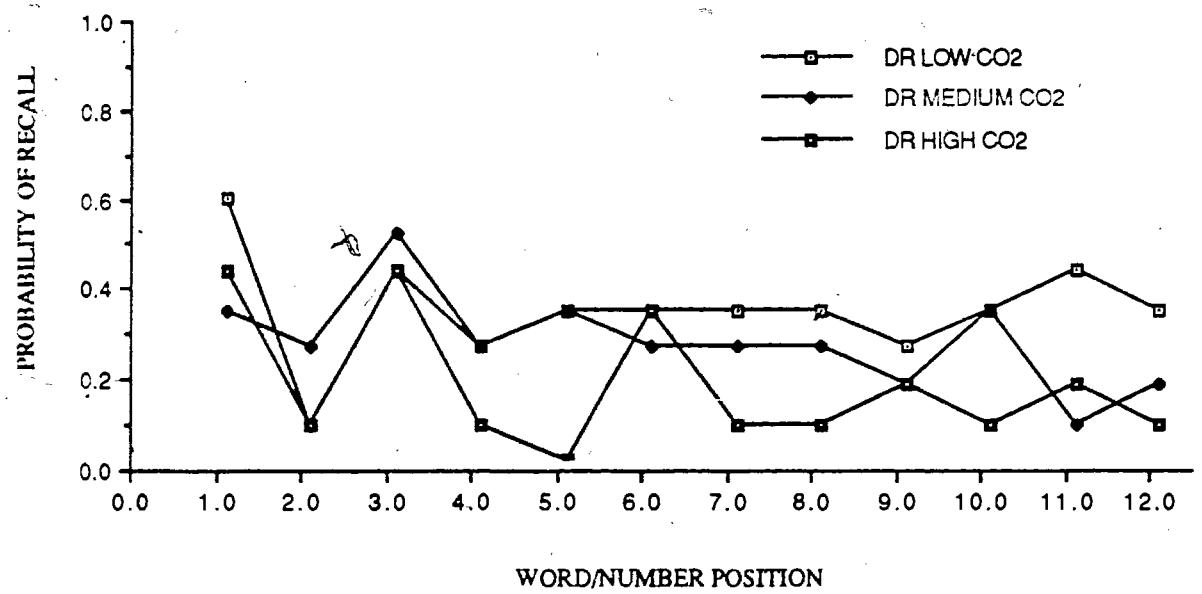


Figure 4.11. **Probability of delayed recall as a function of serial position on the paired association memory task, following exposure to low, medium and high PCO_2 tensions at 1 ATA (A), and 6 ATA (B) of pressure. Error bars are ommited for the sake of clarity. (n=12).**

CHAPTER 5
DISCUSSION

Many interfering variables, other than raised partial pressures of inspired gases, may influence man's performance and efficiency in the hyperbaric environment. Factors such as anxiety, adaptation, practice effects and learning can significantly contaminate data and produce misleading results. (for a review see Bachrach, 1975). To reduce or eliminate the influence of these interfering variables special measures were taken in the design and protocol of the experiments. Nine of the twelve test subjects had previously participated in similar hyperbaric air experiments and were familiar with the hyperbaric chamber, as well as, the compresssed air environment. The three remaining novice subjects were provided with extensive information on chamber operations and procedures, and completed a training dive to 2 ATA of pressure prior to experimental trials. Before starting the experimental sessions, subjects performed extensive practice trials on each performance test in the test battery. This reduced intra-subject variability on the individual tests and ensured stable scores were obtained prior to the experimental sessions. Therefore previous test naivete was not a significant factor in the experiment. Any further learning or practice effects, as well as, possible adaptation across experimental trials, were counterbalanced by the experimental design. The experimental design also permitted assessment of the effects on performance when the partial pressure of N_2 and CO_2 were raised individually as well as simultaneously. By analyzing the data in this way it was possible to divide performance impairment into N_2 and CO_2 components.

5.0.10 Nitrogen Effect

Raising the ambient pressure from 1 to 6 ATA produced an approximate 4.0 ATA increase in the inspired nitrogen pressure. The effect of this P_IN_2 increase on performance is demonstrated by the vertical distance between each pair of curves for performance scores on the individual tasks presented in the Results. Both the Copying test and the Number comparison test demonstrated little alteration in the attempted or correct scores under narcosis. This may indicate the level of narcosis at 6 ATA was insufficient to cause significant slowing of perceptual speed or flexibility of closure. On the other hand, the error score on both these tests showed a significant increase at 6 ATA. One explanation for this could be that subjects were experiencing visual or perceptual disturbances as a result of some kind of structural or functional deficit induced by the narcosis, but this explanation is unlikely as non of the subjects reported any kind of visual disturbance at depth. Unfortunately, formal quantification of subjective experiences at depth were not measured and therefore conclusions on subjective effects of narcosis are limitted.

In studies on compressed air narcosis at pressures in excess of 10 ATA, some deterioration in visual discrimination and changes in the visual figure background contrast have been reported (Adolfson & Muren, 1985). The presure of N_2 at these depths, however, is excessive and it is only when the human limits of compressed air tolerance are reached, that reports of psychosensorial changes begin to appear.

A more likely explanation for the increase in error scores may be provided on the basis of the speed-accuracy nature of these tests. The euphoria and exuberance, as a consequence of the narcosis, may have resulted in a disinclination of the subjects to maintain a given speed-accuracy setting. It may be that the subjects preferred to divide the impact of narcosis between speed and accuracy. The significant error scores may be viewed as strategic changes rather than structural or functional deficits of the visual processing system. A contributing factor in the significant error score on the Copying task could also be, in part, due to a motor component in the test. Fine manual dexterity was found to be significantly effected at 6 ATA, and therefore, impaired fine muscular coordination may have led to some inaccuracies while drawing the four line geometrical figures.

As perceptual speed has been related to flexibilty of closure (Pawlik, 1966; Ekstrom, 1973), it was expected that similar results would be produced by the Number comparison and the Copying task. Both require the subject to make a visual match but disembedding is required in flexibility of closure, while mere location is required in the perceptual speed test (Ekstrom *et al.* 1976). The results, despite the differences between tests and the aforementioned explanation of the error scores, agree with other studies which have investigated visual functions, uncomplicated by motor or cognitive processes. Biersner (1972), using 30% N_2O to simulate the anaesthetic properties of air at 7.4 ATA found no changes in visual acuity, accommodation or visual memory. In addition, Schellart (1976), reported only small and inconsequential losses of visual acuity for subjects breathing air at 7 ATA in a diving tank. In conclusion, it is unlikely that narcosis at this depth effects perceptual speed and flexibility of closure at a structural level. However, the nature of the particular tests, used to assess these factors, is susceptible to motivational and strategic changes induced by the narcotic condition.

The Stroop test, conversely, demonstrated a significant decrease in the number of letters crossed-out and a significant increase in the error score. These findings imply a reduced ability to make simple decisions quickly and correctly. This suggests slowing of neural processes involved in reasoning and immediate memory and an unwillingness on behalf of the subject to maintain an accurate setting. A comparison of this test with those in the literature is complicated by procedural differences and diverse

variations of the Stroop test employed by various investigators. It is interesting to note, that Hesser *et al.* (1971), reported significant ($p<0.05$) 16% decrement on the timing score for their version of the Stroop colour-word test. Although errors were monitored on this task no consistent changes occurred with the increase in N_2 pressure.

Contrary to the above test discrepencies, the exact same arithmetic task employed by Hesser *et al.* (1978) was used in the present study. A signficant 10% drop in both the number of problems attempted and the number of correctly solved problems was noted by Hesser *et al.* when the inspired PN_2 was raised by 6.3 ATA at a constant inspired PO_2, which was somewhat smaller than the decrement observed on the same scores in the present study. This is also contrary to a 1.5 ATA greater difference in the P_IN_2 pressure between control and depth conditions in Hesser *et al.*'s study. The unexpected disparity maybe partly explained by the approximate 1 ATA difference in inspired PO_2 between surface and 6 ATA in the present study.

It has been shown that an increase in PO_2 of this magnitude can result in a significant decrement in arithmetic skill (Hesser *et al.* 1978). It is therefore likely that the raised inspired PO_2 at 6 ATA would have contributed to some portion of the significant decrement noted in this test and in many of the other tests of cognitive function. Other factors which may cause variations between tests may be due to subject susceptibilty to narcosis and experimental procedural differences. An explanation, for decrements seen on this seemingly simple cognitive task, is made difficult by the complex number of underlying processes important in producing successful responses.

Simple arithmetic manipulation is thought to be part of an "automatic" process factor, incorporating both number facility and perceptual speed which is not a major component in mathematical reasoning or higher mathematical skills (Ekstrom *et al.* 1976). According to Carroll (1974), basic arithmetic operations involve both "retrieving appropriate number associations and algorithms from long-term memory and performing serial operations on the stimulus materials using these associations and algorithms" in working memory. Using this concept Fowler and Granger (1981) have proposed an explanation of why simple arithmetic tests are degraded by narcosis. The decrement in the number of problems attempted is thought to be a result of a slowing of the operation involved in each stage of processing of the interim computations by the narcosis. In the present arithmetic task there were two distinct arithmetic operations. Firstly, the subject had to determine the product of a one by one digit multiplication, and secondly, to add or subtract this result from a two digit number. Slowing of the two processes by narcosis, could therefore lead to the

lower attempted score noted at 6 ATA.

Increased errors on arithmetic tests at depth have been attributed to forgetting of the interim calculations stored in STM, as a result of the increased time (slowing due to narcosis) required to carry out operations utilizing LTM (Fowler & Granger, 1981). There may also be, insufficient slowing to maintain accuracy constant due to a strategic change in the speed accuracy setting employed by the subject. Although there was a 40% increase in the mean number of errors on the arithmetic test at 6 ATA, this performance impairment was not significant.

The results of the Stroop and the math test, clearly indicate that raised nitrogen partial pressures impaired cognitive ability. However, a significant decrement in pyschomotor performance was unexpected at 6 ATA. Previous studies have shown little effect of narcosis on manual dexterity using the Vagland screw plate test even up to 8 ATA of pressure (Adolfson, 1965; Hesser *et al.* 1978). These findings have led to the conclusion that nitrogen narcosis degrades psychomotor skill to a lesser extent than cognitive function (Bachrach, 1975). Unfortunately, one of the problems with comparing different psychomotor tasks, presumed to measure the same variable, is that they may in fact be measuring subtle but important differences in dexterity. Factor analysis of human performance has shown that a fine coordination test such as the Purdue pegboard, is really a finger dexterity exercise while manual dexterity is a factor reserved for arm and hand; a coordination more gross than the finger dexterity of the pegboard (Bachrach, 1975). As a result, it maybe argued that the Purdue pegboard would be more sensitive to narcosis decrement than the Vagland screw plate test.

Bennett and Towse (1971) have also indicated the Purdue pegboard to be more sensitive to psychomotor impairment by narcosis than either the touch or ball bearing test. Although these authors note a decrement in performance on the Purdue pegboard at pressures between 6.5 and 7.5 ATA they neglect to say whether the impairment was significant. The worst scenario they report is a mean decrease of 7.1% in finger dexterity at 7.5 ATA compared to surface control values. A similar percent decrement (7.9%), in psychomotor performance on the Purdue pegboard, was reported by Kiessling and Maag (1962). However, these authors' results were significant at a much lower pressure of 4 ATA. In comparison, the 9% decrement on the Purdue pegboard at 6 ATA noted in the present study agrees favourably with the results of Kiessling and Maag, and therefore lends support to the observation of significant decrements on fine manual dexterity at comparatively low N_2 pressures. Immediate and delayed recall, conversely, demonstrated large decrements at 6 ATA. Compared to performance on the Purdue pegboard, memory

impairment under narcosis was four times greater than the decrement shown for fine manual dexterity.

While the immediate recall (IR) test may be considered a STM task on the basis of elapsed time (Fowler *et al.* 1980), the serial position curve analyses revealed that a single overall performance measure concealed the fact that not all parts of the task were affected in the same manner by compressed air narcosis. It was found that, when breathing hyperbaric air at 6 ATA, immediate recall was significantly affected in the recency portion of the curve but not in the primacy portion. Although the percent decrement in the total number of words recalled on this test is similar to the observations reported by Fowler *et al.* on a free recall task while under the influence of 35% N_2O; a comparison of the primacy and recency responses with those in the latter study are contradictory.

Fowler *et al.* (1980) concluded from their results that a significant decrement in the primacy response was indicative of an impairment in (long term memory) LTM, while the insignificant change in the recency response suggested (short term memory) STM was unaffected by the narcosis. This conclusion was based on the commonly held view that the primacy portions of the curve represent LTM while the recency portion of the curve represents STM (Atkinson and Shiffrin, 1971; Glanzer, 1972).

The present results, however portray an opposing view, in that hyperbaric air appears to affect the recency, and not the primacy portion of the serial response curve. Based on these observations it would appear that narcosis impairs STM but not LTM but the reason for this discrepancy is unclear. One explanation could be due to the different approaches to analysis of the primacy and recency responses on the serial position curves employed by Fowler *et al.* (1980) and the present study. Fowler *et al.* (1980) used a total of 15 words in their serial word list, and determined primacy and recency responses from *post hoc* inspection of the serial response curve. The recency response was determined from the final part of the serial response curve (serial positions 9–15), showing a positive slope, while the primacy response was defined as the initial part of the serial response curve that demonstrated a negative slope. Although statistical analysis was provided for the recency response, no statistics or mention of the actual serial positions considered to represent the primacy effect were reported. In comparison, the present study determined primacy and recency responses on *a priori* grounds, based on criterion according to the literature (see Section 3.0.6).

An alternative explanation could be that hyperbaric air and N_2O may produce a different pattern of effects on immediate recall responses. However, most of the evidence points towards a commonality of effects of these two agents for memory recall (Steinberg, 1957; Fowler, 1973; Fowler and Ackles, 1975;

Biersner *et al.*, 1977; Fowler *et al.* 1980). Nevertheless, there has been some disagreement in the literature between the effects of hyperbaric air and N_2O on STM. A brief discussion on this point was made in Section 2.0.2.

The possibility of a strategic change as a result of the narcosis cannot be ruled out. At pressure, when the immediate recall response sheet was presented, subjects may have chosen to begin recalling the word/number associations presented at the start of the list before those associations at the end of the list. As the more recently presented associations would have had less time to be rehearsed in STM, it could be hypothesised that during recall there would be a high probability that they would be forgotten or lost from STM unless rehearsed or reported immediately. Forgetting the associations would therefore be most pronounced if the subject chose to attempt recall of the earlier associations before the latter associations.

At surface an alternate and more common strategy may have been used for recall. Klatzky (1975) noted on similar memory tasks that subjects usually tended to report words from the end of the list, first. Consequently, those items stored in STM were immediately retrieved resulting in the classic recency effect. Thus, if the majority of subjects had employed the first strategy at depth and the latter strategy at surface, this may explain the unexpected results obtained for the primacy and recency responses on the immediate recall test. Unfortunately, conclusive evidence for this arguement is not available from the current data, however this speculation provides an avenue for further research into learning/recall strategies under narcosis.

Although the insignificant change for the primacy effect during immediate recall, indicated narcosis had little effect on LTM, a subsequent test of delayed recall suggested quite the contrary. Based on elapsed-time and the fact the subjects completed distractor tasks between presentation of the associations and delayed recall, responses on the DR test could be considered as representative of LTM function.

All DR tests, following surface trials and depth trials, were conducted under non-narcotic conditions and at normocapnic levels. Consequently, the decrement found for DR following dive trials was unlikely to result from impaired retrieval from the long-term store. The fact that response sheets contained word cues to aid in retrieval of associations (Fowler *et al.* 1980) lends strength to the argument. This implicates input to LTM as the source of the learning deficit under narcosis.

This view, however, is far from conclusive as it is based on indirect inference rather than direct evidence. Indeed other studies have pointed toward a completely opposite view (Ghoneim *et al.* 1981;

Adam 1973; 1976). Thirty percent N_2O has been found to impair DR on a multiple trial free recall test irrespective of whether subjects learned the material under the influence of N_2O or not (Ghoneim *et al.* 1981). Similar observations have also been found using subanaesthetic concentrations of fluroxene and halathane (Adam 1976). Additionally, Adam (1973) reported that forced choice recognition of material, learned while breathing N_2O, improved after the event. These findings have led to the hypothesis that the effects of general anaesthetics on memory could be explained by a disruption of the processing required to both encode information into LTM and to retrieve it (Adam 1979). It is not clear in these experiments, whether narcosis impairs retrieval by directly reducing the accessibility of memory traces or by reducing the subjects' motivation to attempt recall of difficult or poorly learned items (Ghoneim *et al.* 1981).

Support for an alternative hypothesis, maintaining that there is a failure to input information into LTM due to some structural deficit, has also been strong (Fowler 1973; Fowler *et al.* 1980; Fowler & Granger 1981; Steinberg 1954). These authors conform to the view that input to, rather than retrieval from, LTM is the source of the learning deficit under narcosis. This proposal was based on the observations that a learning deficit induced by hyperbaric air or N_2O was not ameliorated by a return to the normal state (Steinberg 1954; Fowler 1973), word cueing (Fowler *et al.* 1980) or forced choice recognition (Fowler & Granger 1981). Although the present data implicate a failure/reduced capacity to input information into LTM at depth, conclusions regarding retrieval from LTM as a possible source of the memory deficit can not be made, as no measure of delayed recall was made under narcotic conditions.

An explanation of the narcotic deficits on LTM has been provided by Fowler *et al.* (1987). Using 35% N_2O they noted that narcosis altered the overt rehearsal strategy on a word list task. It was found there was a decrease in the overall rate of rehearsal, as well as, a decrease in the portion of the words that were rehearsed from the earlier part of the list. As rehearsal is regarded as an encoding process which serves to reinforce items into memory (Klatzky 1975; Geiselman *et al.* 1982), a slowing of this process would result in impaired input into LTM. Consequently, DR responses following exposure to narcosis would show an impairment that would tend to be greater in the primacy portion, of the serial response curve, than the recency portion. As no attempt was made to monitor rehearsal strategies of the subjects in the current study, comments on the above explanation are merely speculative. The DR responses do, however, reveal similar results for primacy and recency effects as those found by Fowler *et al.* (1980) for IR on a word list task.

5.0.11 Carbon Dioxide Effect

Due to the shortage of objective research on the effects of high PCO_2 levels on cognitive performance at increased ambient pressure, there is only a limited number of studies with which a comparison of the present data can be made.

At surface pressures, a recent study by Sayers *et al.* (1987) found that $P_{ET}CO_2$ pressures above 51 Torr significantly slowed performance on a reasoning task (AB logic problems), but had little effect on accuracy of reasoning. These authors also noted a significant rise in irritability and discomfort when subjects were exposed to inspired CO_2 concentrations >6%. Alertness, as well as, registration and recall of long-term memory were not affected by high concentrations of CO_2 (6.5%) (Sayers *et al.* 1987).

Although no attempt was made to quantify the subjective reports of subjects in the present study, it was interesting to note that most subjects became more irritable, and found it harder to concentrate on the tasks when they were exposed to high levels of CO_2. Objectively though, the pattern of results on the cognitive tests were similar to those reported for the reasoning test in Sayers *et al.*'s study. Noticeably on all the tests involving a speed and accuracy component in the performance scores, the decrement caused by hypercapnia was consistantly due to a slowing of performance rather than an effect on the accuracy of responding.

Further evidence of slowed processing induced by high levels of CO_2 has been provided by Hesser *et al.* (1971, 1978). These authors noted a significant 37% decrease in the number of arithmetic problems attempted when the end-tidal CO_2 was raised from 35 to 58 mmHg at 8 ATA (Hesser *et al.* 1978). Using this same arithmetic test, the present results showed a 27% drop in the number of arithmetic problems attempted when the $P_{ET}CO_2$ tension was raised from 29 to 59 mmHg at 6 ATA. The discrepancy between these results was likely due to subject variability and the extent to which the arithmetic test was practiced, as well as differences in experimental protocol.

In the earlier study by Hesser *et al.* (1971), CO_2 was again found to slow performance. In this study, the time score on a Stroop colour word test, both in O_2 experiments at 1.3 ATA and in air experiments at 6 ATA were significantly slower while breathing high levels of CO_2. The format of this test was quite different to the modified Stroop test used in the present study; however, the results demonstrated the same general trend on performance at high $P_{ET}CO_2$ tensions. Visual perception and flexibility of closure were also significantly affected by the high levels of CO_2 at surface pressure and 6

ATA. Unfortunately, the present data did not allow any definite conclusions as to the immediate cause of the observed effects of CO_2 on these factors, or indeed on any of the cognitive and psychomotor tasks employed in the study. It is interesting to note, however, that subjective observations of poor concentration during hypercapnic exposure implied attentional processes may have been affected. Several subjects reported the hyperventilation associated with high levels of CO_2 caused some discomfort and was particularly distractive while completing the test battery. Concentration may also have been affected by the hyperactivity associated with high levels of CO_2. The physiological discomforts of hypercapnia do not, however, explain the statistical significant decrements found on some of the performance tests under the medium level of hypercapnia. Under these conditions mean $P_{ET}CO_2$ tensions were only slightly above normal (47 mmHg) and ventilatory responses although elevated did not particularly stress the subjects.

Physiologicaly it is believed that the symptoms of CNS hyperactivity are the paradoxical result of a depressant effect of CO_2 on the cerebral cortex, releasing subcortical centers from normally powerful inhibitory influences (Woodbury & Karter 1960). This hyperactivity is manifested in extreme cases of CO_2 toxicity by convulsions. It is therefore not unreasonable to expect a certain degree of muscular tremor at lower concentrations of CO_2. Given that this is true, the decrement in fine muscular coordination at end-tidal PCO_2 tensions greater than 55 mmHg could be a result of increased muscular tremor induced by the hyperactive effect of CO_2.

It is intrigueing to speculate that the physiological hyperactivity induced by high levels of hypercapnia may be related to the psychological phenomenon of arousal, and thus to attentional processes. Arousal refers to the level of physiological excitation and its influence on behaviour (Thayer 1978; Vanderwolf & Robinson 1981). "The contention that physical arousal influences attentional processes has been accepted as virtually axiomatic by most learning theorists" (cf. Tomporowski & Ellis 1986). One theory proposed by Easterbrook (1959) maintains that any variation in physical arousal will produce a change in attentional processes. According to the words of Tomporowski & Ellis (1986, p339).

> ...an increase in arousal will result in the shift or "narrowing" of attention to those components of a task that are central to correct performance; attention to those components that play a limited role in correct performance will be reduced. As the level of physical arousal increases, the selection of task-relevant stimuli may be restricted because of continued narrowing of attention and, as a result, performance will deteriorate.

If CO_2 hyperexcitability can be considered as a form of physical arousal the above model would predict the same pattern of performance on tests of information processing (such as in the cognitive tasks used in the present study), as the inverted-U theory does for motor perfromance (Tomporowski & Ellis

1986). Evidence for a similar pattern of results between cognitive and psychomotor performance under high levels of PCO_2 was provided by the present results and by the experiments of Hesser *et al.* (1971; 1978). The model also predicts that compensatory strategies would attempt to maintain accuracy of processing at the expense of speed of processing when performance is affected by increased physical arousal levels. Interestingly, data on the cognitive tasks show this exact affect when $P_{ET}CO_2$ tensions were elevated beyond normocapnic levels.

It was further noted that some cognitive tests were more affected by CO_2 than others. A more recent extension of Easterbrook's model hypothesises that each individual has a fixed amount of attentional capacity that can be allotted to process incoming information (Kahneman 1973). Furthermore, some forms of information processing such as memory or spacial location occur automatically, whereas other processes such as imagery, rehearsal, and mnemonic techniques demand an effortful allocation of attention (Tomporowski & Ellis 1986). According to the present results, this observation is born out by the larger decrements noted for IR(33%) and DR(35%) compared to the Copying task (16%, (correct score)) and number comparison task (12% (correct score)), when the $P_{ET}CO_2$ was raised from 29 to 57 mmHg. Although the performance decrement for DR was nonsignificant, the calculated p value for the main effect of CO_2 was 0.052.

In comparing the relative percent change in performance from test to test, consideration of possible order effects should be taken into account. As the test order remained the same throughout the experiment, due to practical reasons, it is likely that the latter tests may have been at a more stable level of CSF PCO_2. In comparison, the earlier tests may reflect conditions in which CSF PCO_2 was still increasing. Consequently, considerable care should be taken when comparing one task to another.

Primacy and recency scores on the memory task were unaffected by high levels of $P_{ET}CO_2$. This suggested the impaired performance for IR was not confined to the word/number associations at the end or start of the list, but indicated all word/number pairs were equally affected by CO_2 narcosis. Whether the decrement for IR was due to an impaired input or a reduced capacity to retrieve from memory, or due to both, is uncertain.

Returning to the arousal theory as a possible explanation for the performance decrements; it may be argued that if CO_2 induces an over arousal and hyperbaric N_2 produces a CNS depression or decrease in arousal (Fowler *et al.* 1985; 1986), then why doesn't one effect ameliorate the other? This anomoly could be explained if CO_2 narcosis and N_2 narcosis produce their effects through unrelated mechanisms that

have no direct influence or interaction on each other. Indirect evidence is provided in the present study by the departure of the cognitive data from the predicted narcotic potency of CO_2 according to the lipid solubility theory of narcosis (see Section 2.0.5). According to this theory, the lipid solubility of CO_2 is 13 times greater than for N_2 (Dittmer & Grebe 1958) and therefore should be approximatly 13 times as potent a narcotic. The cognitive performance data, however, demonstrated CO_2 to be more than ten times as narcotic as predicted by the lipid solubility theory. This argument assumes the lipid solubility theory is a valid interpretation of the mechanism of compressed air narcosis. Unfortunately as was mentioned in Section 2.0.5, the underlying mechanism for inert gas narcosis is not fully understood. In addition, the lipid-like anaesthetic activity of CO_2 has not been tested because it seems to produce acidotic anaesthesia at much lower PCO_2 levels (Severinghaus 1974). It is therefore possible that CO_2 narcosis effects are related to hydrogen ions or to the extracellular pH (Eisele *et al.* 1967) which produce their effects at far lower PCO_2 levels than does molecular CO_2 in accordance with the lipid solubility theory.

Further evidence suggesting CO_2 has no direct influence or interaction with the N_2 effect was also provided by the present results. It was observed that most performance curves at 1 and 6 ATA were approximately parallel to each other over the measured range of $P_{ET}CO_2$ tensions. This suggested changes in CO_2 tension had little effect on the degree of narcosis produced by the high N_2 pressure itself. These observations further support the conclusions of Hesser *et al.* (1971) "that variations in alveolar CO_2 tension have no significant influence on the magnitude of the nitrogen component in compressed air narcosis." Thus the effect of high CO_2 pressures are purely additive in their effects on impaired cognitive and psychomotor performance at depth.

With regard to a global threshold of $P_{ET}CO_2$, above which cognitive performance shows significant decrements, the current data shows no clear or consistant onset. Hesser *et al.* (1978) reported "the role of CO_2 as a causative factor (in impairment of mental function) is negligible as long as the alveolar (arterial) PCO_2 does not exceed 40 mmHg." Later work by Sayers *et al.* (1987) observed a clear but higher threshold of 51 Torr for the effect of $P_{ET}CO_2$ on a reasoning task.

Evidence against a threshold for CO_2 narcosis has been provided by the work of McAleary *et al.* (1961). These authors found the anaesthetic potency of N_2O was altered by PCO_2, such that less N_2O was required to induce loss of consciousness at high PCO_2 tensions. The relationship between anaesthetic potency of N_2O (the minimum alveolar concentration producing a nonresponsive anaesthetic state) and end-tidal PCO_2 tension, was found to be linear over the measured range (20 to 60 mmHg) of $P_{ET}CO_2$.

McAleary *et al.* (1961) concluded from their results that CO_2 (or hydrogen ions) has five times the anaesthetic potency of N_2O. The present data provides further evidence against a common threshold phenomenon, and suggests that a threshold, if present at all, is dependent on the sensitivity of the particular task or cognitive factor to hypercapnic stress. This view is shared by Schaefer (1974) who contends that different areas of the brain have different sensitivities to CO_2. Thus the current data would indicate that the area of the brain responsible for visual perception and flexibility of closure is less affected by CO_2 than the area of the brain responsible for immediate memory.

5.0.12 Ventilatory Responses to CO_2 Elevation at 1 and 6 ATA

All but the baseline measures for the mean $P_{ET}CO_2$ tensions at 1 and 6 ATA fell within the desired $P_{ET}CO_2$ ranges specified prior to experimentation. The reason for the slight hypocapnia during the non rebreathing conditions at surface and depth is uncertain. One possible explanation may be due to differences in length between the end-tidal sampling tube and the length of tubing used during calibration of the CO_2 analyzer. As the end-tidal gas sample tubing was considerably longer than the calibration tubing, a larger pressure drop in the end-tidal gas sample line compared to that in the calibration line may have resulted in an artificially low reading for end-tidal CO_2 tensions. This hypothesis was subsequently tested by calibrating the CO_2 gas analyzer with a 1.03m length of gas sample tubing and comparing the calibrated meter reading to that obtained with the same calibration gas drawn through a 2.90m length of tubing of equivalent diameter. The resultant meter readings were found to be equivalent, indicating very little effect of gas sample tubing length on the integrity of the CO_2 gas analyzer readings.

A second reason for the low baseline $P_{ET}CO_2$ tensions could be due to the breathing resistance of the rebreathing circuit. The elevated effort required to breathe through the rebreathing circuit may have made the subjects more aware of their respiration. Consequently, subjects may have changed their breathing pattern sightly in response to the increased breathing resistance and the unfamiliarity with breathing through a mouth piece with the nasal pasage occluded by a noseclip. Evidence that breathing through a mouthpiece with the nostrils occlude changes the breathing pattern of humans has been demonstrated recently by Hirsch & Bishop (1982), Askanazi *et al.* (1980) and Sackner *et al.* (1980). Tidal volume has been shown to increase on a mouthpiece where as breathing frequency may decrease, however, changes in breathing frequency are reported to be variable. These changes in breathing pattern may have led to a greater off-loading of CO_2 compared to normal respiration and resulted in the observed hypocapnia during the control conditions. Unfortunately, this is only speculative as no comparisons

between the ventilatory response on the rebreathing circuit and normal unrestricted breathing, was made in the current study.

Ventilatory responses to hypercapnia at depth were similar to those observed in previous studies (Wood and Bryan, 1970, 1971; Doel *et al.* 1974; Linnarsson and Hesser, 1978; Hickey *et al.* 1987). Characteristically, a reduced breathing frequency and hypoventilation was noted in the hypercapnic responses to CO_2 rebreathing at 6 ATA compared to the normal ventilatory responses to elevated PCO_2 tensions at 1 ATA.

The reduced ventilatory response to hypercapnia under hyperbaric states, has been attributed to the increased gas density and breathing resistance rather than by any depressant action of high PN_2 on the respiratory center (Frankenhauser *et al.* 1963; Fagraeus and Hesser, 1970; Wood and Bryan, 1970, 1971; Linnarsson and Hesser, 1978). Wood and Bryan (1970, 1971) noted that for a given PCO_2, inspiratory work and tidal volume did not change with increased pressure. This was interpreted as an indication that CO_2 sensitivity of the respiratory centre was unaltered at depth. Consequently, because the increase in air flow resistance produces a decrease in airflow rates and respiratory frequency at depth, the same inspiratory work produces less ventilation at depth (Schaefer, 1975).

Of greater pertinence to diver safety, than ventilatory limitations *per se*, is the accumulation of CO_2 in the blood. The marked effect of CO_2 narcosis on cognitive and psychomotor performance at depth has already been discussed (See Section 5.0.11). It is clear, that any condition reducing $\dot{V}_I$, increasing the level of P_ICO_2, or elevating CO_2 production, will predispose divers to CO_2 accumulation.

While the effects of inhaled CO_2 have been reported to produce the same physiological responses as metabolically produced CO_2 (Lambertson, 1971), research looking at a commonality of effects on cognitive and pyschomotor performance between these two hypercapnic states has received little attention. This may be due to methodological problems associated with collecting cognitive data in the exercising hypercapnic state. However, a recent review on the effects of exercise on cognitive processes has been presented by Tomporowski and Ellis (1986). Providing researchers employ a theory-based parametric approach to this issue (c.f. Tomporowski and Ellis (1986)), a comparison of cognitive performance during exercise induced hypercapnia at depth with CO_2 rebreathing in the hyperbaric environment, will provide useful information relevant to the working diver.

One issue central to these experiments, is that in studies concerning psychophysiological effects of CO_2 the measured variable should be examined in relation to the CO_2 changes occuring at the site of action of CO_2 (Hesser *et al.* 1971). As CO_2 narcosis is thought to be due to an effect on the brain, performance changes should therefore be related to the cerebral rather than the inspired CO_2 tension. Since there are no acceptable methods for measuring cerebral PCO_2 *in vivo* in humans, PCO_2 tensions in the brain were assessed indirectly from changes observed in $P_{ET}CO_2$. This assumes the differences existing between arterial and end-tidal PCO_2 and between arterial and cerebral PCO_2, remain approximately constant when the inspired CO_2 tension is increased. A detailed investigation on the validity of $P_{ET}CO_2$ as a measure of P_aCO_2 has been presented by Jones *et al.* (1979). Briefly, the $P_{ET}CO_2$-P_aCO_2 differences noted in Jones *et al.*'s study were found to correlate with changes in V_T (r=0.791) and to a lesser extent with breathing frequency (r=-0.475). In the current study, all experiments were completed at a controlled steady state of $P_{ET}CO_2$ and therefore it was assumed that the $P_{ET}CO_2$ was in equilibrium with the P_aCO_2.

When P_aCO_2 values were calculated according to the regression equation derived by Jones *et al.* (1979), there was no change in the pattern of results observed. The mean P_aCO_2 values during the non-rebreathing condition at surface and depth were calculated to be 31.6 mmHg for both conditions. During the medium level of hypercapnia, the mean P_aCO_2 values were 47.6 and 48.6 mmHg for surface and depth conditions respectively, while under the high level of CO_2, P_aCO_2 were the same as their respective $P_{ET}CO_2$ values. The small differences in absolute values between the estimated P_aCO_2 and observed $P_{ET}CO_2$ (<2.6 mmHg) had no effect on the relationship or pattern of results for cognitive and psychomotor performance. In contrast, when test scores were compared to inspired PCO_2, performance impairments demonstrated a greater decrease with equivalent increases in P_IN_2 tension at depth compared to surface. Conclusions drawn from this observation would indicate the narcotic action of CO_2 is enhanced by high N_2 pressure. The reason for this becomes clear when the inspired CO_2 tension is related to end-tidal CO_2 tension (see Figure 4.1). At equal inspired CO_2 tensions, the end-tidal PCO_2 was found to rise to greater levels in the experiments at 6 ATA than in the experiments at surface. This explains the synergistic relationship between PCO_2 and cognitive performance at depth reported by early investigators (Case & Haldane, 1941; Marshall, 1950; Bennett, 1966).

The importance of CO_2 on performance in the diving environment, was revealed when the relative narcotic potency of CO_2 and N_2 was compared. Based on the combined average percent decrement for correct responses on the tasks in the test battery, it was calculated that CO_2 was approximately 135 times

more narcotic than N_2. From this figure it can be deduced that an increase of 4.4 mmHg in $P_{ET}CO_2$ would have a narcotic effect equivalent to the addition of 1 ATA of air. Consequently, relatively small increases in $P_{ET}CO_2$ can have substantial effects on the narcotic state of the diver. It should be noted, however, that the narcotic effects of CO_2 are slightly different to those induced by N_2 narcosis, and therefore, the above statement of narcotic equivalence, should not be taken to represent synonymous physiological/psychological causes and effects.

The present findings affirm the importance of CO_2 as a major threat to diver safety in the hyperbaric environment. Carbon dioxide is an important factor in the ergonomic design considerations of both open circuit breathing apparatus (dead space, helmet ventilation) and closed circuit rebreathing apparatus (CO_2 absorption) as well as in underwater habitats and environments. It has been suggested by Morrison *et al.* (1978), that narcosis tests, such as those employed in the current study, would be a valuable tool for identification of divers who may be particularly susceptable to the combined narcotic effects of nitrogen and hypercapnia at depth. One population group which has been targeted as being particularly at risk from CO_2 narcosis are those divers labelled as "CO_2 retainers". Further research, however, is required to assess the relative susceptability of CO_2 retainers to the ill effects of increased CO_2 pressure at depth. There is some evidence to suggest that CO_2 retainers experience fewer symptoms of CO_2 narcosis, in terms of headaches and mental or spatial disorientation during inhailation of 7.5% CO_2 (Schaefer, 1958). Unfortunately, this evidence is based on subjective observation and as yet no direct quantitative comparisons have been made on the narcotic susceptability of CO_2 retainers with the performance responses of divers exhibiting normal ventilatory responses to increasing PCO_2. Clearly, further research is required in this area before the present results can be applied to the working diver.

Other factors including cold, diving experience and apprehension in the hyperbaric environment may also interact with CO_2 narcosis and produce additional performance changes under hypercapnic stress. It is therefore essential that if real world problems are to be solved, the present research needs to be extended and compared to physiological and psychological responses of divers working in open water conditions. However, within the limited range of environmental conditions in the present study, it is suggested that inspired PCO_2 tensions be kept below 23 mmHg, or 3% CO_2 at surface equivalent pressures, in order to avoid serious cognitive and psychomotor deficits at depth. Furthermore, if a working diver begins to experience increasing severity of narcosis symptoms at depth, as a possible result of over exertion, he should immediately reduce the level of physical until the symptoms subside. In this way the possibility of diving accidents due to hypercapnia may be minimized.

CHAPTER 6

SUMMARY AND CONCLUSIONS

High $P_{ET}CO_2$ levels (>47mmHg) were found to have significant effects on cognitive and psychomotor performance at surface pressures. In addition, cognitive and psychomotor performance further deteriorated at depth when $P_{ET}CO_2$ levels were increased above 47 mmHg; however, no clear threshold for the onset of CO_2 narcosis was found from the performance scores. When the CO_2 performance data were collapsed over the two N_2 pressure levels it was found that a mean increase of 27 mmHg in $P_{ET}CO_2$ tension produced similar but slightly larger performance decrements than an increase of 3000 mmHg in P_IN_2. Based on these figures, the narcotic potency of CO_2 was calculated to be 135 times the narcotic potency of N_2. This figure agrees favourably with the calculations of narcotic potency of CO_2 reported by Morrison *et al.* (1978) and Hesser *et al.* (1978) (see Section 2.0.6). Hesser *et al.* (1978) did however, report that their data indicated a narcotic potency of CO_2 several hundred times greater than that of N_2; unfortunately, this observation was based on only two performance tests, an arithmetic task and the Vagland screwplate test. Thus conclusions of the narcotic potency of CO_2, with respect to other measures of cognitive and psychomotor performance, were extremely limited in Hessers *et al.*'s study. The present results provide a larger data base to assess the relative narcotic potency of CO_2 according to performance impairment on a variety of different cognitive and psychomotor tasks.

In general, when comparing performance decrements, across the various tests, it was found that the comparitive sensitivity of a particular test was the same for N_2 narcosis as it was for CO_2 narcosis. When the performance tests were arranged from least to most affected by N_2 narcosis (as determined from correct scores), the order remained the same for performance decrements induced by CO_2 narcosis. The pattern of results on these tests was however, quite different for N_2 and CO_2. While virtually all performance tests demonstrated significant decrements for the two main effects (P_IN_2 and $P_{ET}CO_2$), high levels of hypercapnia induced these decrements through a slowing of performance rather than a disruption of the accuracy of processing. Nitrogen narcosis produced significant impairment through both decreases in accuracy, as well as, a retarding of processing.

Although the performance data indicated changes in functional and strategic variables in response to the particular stressors, the definite strong pattern of effects observed for CO_2 suggests "that the mechanism of CO_2 narcosis differs fundamentally from that of N_2 narcosis" (c.f. Hesser *et al.* (1978)). This conclusion was further supported by the departure of the cognitive data from the narcotic potency of

CO_2 predicted by the lipid solubility theory. Furthermore, the lack of a significant interaction between P_IN_2 and $P_{ET}CO_2$ suggested the effects of high PCO_2 tensions were purely additive in their effects on impaired cognitive and psychomotor performance at depth. These conclusions do not exclude the possibility that CO_2 may have narcotic effects that act in accordance with the lipid solubility theory; however, it is likely that these effects are overwhelmed by the actions of acidotic anaesthesia occuring at much lower PCO_2 tensions.

It is interesting to note that those tests which were most sensitive to N_2 narcosis were also those tasks which were believed to test higher cognitive processes. Thus there is some evidence that indicates neural structures, supporting memory and decision-making, show greater functional impairment than do those supporting visual perception and fine manual dexterity when exposed to high N_2 pressures. This conclusion, however, should be considered with a certain degree of scepticism as order effects and test design artifacts may have contributed to some of the observed differences between individual tasks in the test battery. Additionally, comparisons of percent changes in performance scores, between the cognitive tasks and the psychomotor task, may not necessarily reflect those factors that limit the real functional ability of a diver working on a specific task underwater.

The current data were also viewed in terms of an information processing approach to performance and appear to agree with the slowed processing model of narcosis. Evidence from the present study could therefore be used to establish a link between the behavioural proposition that narcosis represents a slowing of information processing and the long standing physiological conception of narcosis as a CNS depressant.

The link between performance and arousal was further developed to explain the decrements seen with CO_2 narcosis. The paradoxical hyperactivity found on exposure to high PCO_2 levels were speculated to reduce cognitive performance through over arousal. This speculation was based on the pattern of results for the CO_2 performance data which compared favourably with that of Tomporowski and Ellis's (1986) description of the physical arousal hypothesis on information processing.

APPENDIX 1:

SUBJECT INFORMATION PACKAGE, INFORMED CONSENT AND MEDICAL QUESTIONNAIRE

SUBJECT INFORMATION PACKAGE

THE EFFECTS OF HYPERCAPNIA ON COGNITIVE AND PSYCHOMOTOR PERFORMANCE IN THE HYPERBARIC ENVIRONMENT

ITEM 1 PROJECT OBJECTIVES:

The primary objective of this study is to investigate the effects of various levels of carbon dioxide partial pressures on psychomotor and cognitive performance under hyperbaric conditions.

ITEM 2 TEST PROCEDURES:

Volunteers who participate in this study must first meet minimal standards of good health as certified by them in the subject Medical Questionnaire and after examination by the diving physician (Don Hedges, MD). In addition, a test dive to 30 feet of sea water (fsw) (10m) will be required to assess suitability for hyperbaric exposure. During the first few weeks of experimentation a practice session in the chamber at surface (1 ATA) will be undertaken on the test battery listed below so that all subjects will be on a plateau of learning/performance on the various simple tests. Each subject will be paid for their participation in this study.

All subjects will undergo three trials at 1 ATA (surface) and three hyperbaric dives (dry) to 165 fsw (6 atmospheres of pressure) using air as the breathing medium. During each dive, subjects will breathe one of three different carbon dioxide concentrations which will be induced and regulated by a rebreathing circuit previously used in other experiments within the department. The end tidal carbon dioxide tensions induced by this system will fall in the ranges 30–35 mmHg, 45–50 mmHg and 55–60 mmHg. An upper limit of 65 mmHg for end tidal carbon dioxide will be adhered to during all experimentation. In addition, no subject will be exposed to breathing hypercapnic air for more than 20 minutes during any one dive. Throughout the experimental trials physiological measurements of minute ventilation, respiratory frequency, partial pressures of the inspired gases (oxygen and carbon dioxide), and end tidal carbon dioxide will be recorded. Provision of gases will be via a SCUBA regulator connected to the rebreathing circuit. The oxygen and carbon dioxide content of the breathing mixture will be monitored throughout the experiment via gas analyzers located outside the chamber. At each pressure level the diving tender will

administer the test battery below:

1. *Arithmetic test:* For measuring number facility. This test comprises 48 different arithmetic problems each consisting of adding or subtracting the result of one-digit by one-digit multiplication from a two-digit number, such as 37+3X8=, 85-6X3= etc. The subjects will be instructed to multiply first and then add or subtract, and to emphasize both speed and accuracy: Time 2.00 min. The scores recorded will consist of the number of problems attempted as well as the number of correctly and incorrectly solved problems.
2. *Copying test:* Each item consists of a four line geometric configuration and a square matrix of dots. The task is to copy the figure onto the dots. It is believed that this requires flexibility of closure in the act of superimposing the particular configuration on a stong visual field. The subject should work as rapidly as possible without sacrificing accuracy: Time 2.00 min. The score recorded is the number of patterns correctly copied in this time period.
3. *Number Comparison test:* In this test subjects inspect pairs of multi-digit numbers and indicate whether the two numbers in each pair are the same or different. The score recorded is the number marked correctly minus the number marked incorrectly. The time given for this test is 45 seconds. This test primarily measures perceptual speed. It may be the centroid of several subfactors (including form discrimination) which can be separated, but are more usefully treated as a single concept for research purposes.
4. *Letter Cancellation test (modified Stroop test):* This test consists of the words *red* or *blue* typed with ten letters afterwards. The colour name is underlined in either red or blue ink randomly. If the colour word is underlined in the same colour then succeeding vowels are cancelled, otherwise consonants in the following ten letters are cancelled. The time for this test is 1.00 minute and overall performance is determined from the number of letters crossed out correctly, the number attempted and the number of errors.
5. *Purdue Pegboard test:* For this test subjects use both hands at the same time. They assemble a series of pins, collars and washers so that the order for one assembly consists of a pin, a washer, a collar and and a washer. The score recorded is the number of parts assembled in 1.00 minute. This test primarily measures fine manual dexterity.
6. *Paired Association test:* This test involves learning 12 pairs of word/number combinations. A different set of 12 words are used are used during each testing session. In presenting the word/number combinations a new word/number pair will be revealed every 5 seconds and the

previous word/number combination removed from the field of view. This will result in sequential presentation of all word/number combinations within 1 minute. Immediate recall of the word/number associations will be tested during the minute following presentation and delayed recall tested 5 minutes after completing the test battery. Subjects will be required to recall the word/number associations on a test paper containing only the rearranged words.

This test battery will be performed by all subjects in each session. After completing the test battery, decompression will commence according to the Canadian Forces Air Diving Tables and Procedures (D.C.I.E.M., FEB/86 revision). It is anticipated that each dive will last no longer than 80 minutes with a maximum bottom time of 25 minutes, however, each subject will be required to remain in the laboratory for a further 60 minutes as a safety precaution against the remote possibility of latent bends.

ITEM 3 RISKS AND DISCOMFORTS:

A(i) Hyperbaric Exposure

Exposure to hyperbaric air at 165 fsw induces a condition similar to that of alcohol intoxication. Its effects are immediate on reaching depth, and include subjective feelings of euphoria and hilarity. Unlike alcohol intoxication these symptoms disappear immediately on decompression. Subjects should be aware however of other more serious risks during hyperbaric chamber diving which are outlined on a separate form entitled "Risks During Exposure to Hyperbaric conditions". It is important that subjects READ THIS INFORMATION CAREFULLY.

A(ii) Safety Precautions

During each dive fully-trained technical staff, including two chamber operators, and a physician trained in diving medicine will be on hand. In addition a diving tender trained in CPR will accompany subjects during the chamber dives. Instruments and materials needed for resuscitation and for the treatment of decompression sickness and related conditions will be available in the laboratory at all times. Sessions in the chamber will be undertaken during hours when the Health Services is open and physicians there will be informed in advance of the nature of the sessions in the remote chance that their assistance would be required (e.g., should the physician on site need to enter the chamber to assist a subject).

(b) Carbon Dioxide Inhalation Under Hyperbaric Conditions

At the low concentrations of carbon dioxide (end tidal values < 40 mmHg) it is unlikely that any noticable physiological or psychological changes will occur. At the higher concentrations of carbon dioxide (end tidal values > 40 mmHg) in conjunction with raised partial pressures of nitrogen, subjects may experience an enhanced narcotic effect. In more severe cases, feelings of euphoria may progress into apathy, tunnel vision, and a distortion of the sense of time. For those rare subjects who may be labeled as carbon dioxide retainers the higher concentrations of carbon dioxide in combination with raised partial pressures of nitrogen may lead to loss of consciousness; however, this should not occur with the end tidal carbon dioxide ranges being produced by the present protocol. A frontal headache, nausia, hyperventilation and dyspnea my be experienced by subjects at the higher concentrations of carbon dioxide.

All the above symptoms are pass quickly and completely disapear within a few minutes of breathing air containing normal levels of carbon dioxide.

ITEM 4 INQUIRIES:

Questions concerning the procedures used are welcome. If you have any doubts please ask for further explanations.

ITEM 5 FREEDOM OF CONSENT:

Participation is on a voluntary basis. You are free to deny consent, if you so desire, at any time during or between trials, although discontinuation of a "dive" in progress still would require orderly decompression as per the D.C.I.E.M tables.

ITEM 6 CONFIDENTIALITY:

All questions, answers and results from this study will be treated with absolute confidentiality. Subjects will be identified in the resultant manuscript and/or publications by use of subject codes only.

Risks During Exposure to Hyperbaric Conditions

Risks during open-water or hypo/hyperbaric chamber diving include the following:

1. Otic barotrauma. Known as an "ear squeeze", this injury is caused by failure to equalize air pressure between the external environment and the middle ear, generally during pressurization. It involves ear discomfort, pain and sometimes ringing in the ears or bleeding in the ear drum; occasionally the drum is perforated. It generally resolves rapidly (days) but if severe may require medication and/or a specialist's attention. Its prevention requires gentle ear-clearing manoeuvres every second or two during changes in surrounding pressure. If such manoeuvres fail, reducing the pressure difference (e. g., by partial ascent during a dive) should be tried, with repetition of the clearing manoeuvres. If this fails, the exposure should be terminated in an orderly manner. Correction is easiest if undertaken at the first sign of inability to equalize. DO NOT WAIT UNTIL YOU FEEL PAIN! Equalization will be harder if you have a cold or "flu"; under these circumstances hypo/hyperbaric exposure must be postponed. Seasonal allergies may also require postponement of an exposure, unless advised otherwise by the physician responsible for medical aspects of the dive. DO NOT TAKE over-the-counter remedies unless so advised by the physician.

2. Decompression sickness. Generally known as "the bends", this condition develops when nitrogen bubbles form in body tissues during depressurization. Some nitrogen is dissolved in tissues even at the surface, but much more is "loaded" into the tissues during compressed air breathing, in direct proportion to the depth or pressure and to the time spent at hyperbaric pressure. On depressurization, the nitrogen is "supersaturated" in the tissues and if this depressurization occurs too rapidly to allow the offloading of the nitrogen from the blood into the alveoli of the lungs, the nitrogen in the blood and tissues can form bubbles large enough to do damage and to cause symptoms as described below.

    a. Limb bends. If the bubbles form in tissues in and around joints, the result will be a steadily increasing deep aching pain in the involved joint(s). This pain increases with time and can become excruciating if not treated. Predispositions include previous injury or surgery in a joint or, during exposure, a cramped posture limiting circulation in the joint area. Treatment is immediate repressurization in a hyperbaric chamber using a well-established protocol alternating air/oxygen breathing (U. S. Navy treatment tables are used at S. F. U.); this is virtually 100% effective in uncomplicated cases treated rapidly. It is thus mandatory for all divers, subjects and tenders exposed to hyperbaric conditions to maintain a one hour (or more) "bends watch", i. e., to stay under supervision for that time in the chamber laboratory; to report any and all symptoms which arise immediately to the chamber operator or physician responsible, to wear a diver's medical alert bracelet for the next 24 hours during which s/he must not be left alone; to report any and all symptoms during that or the subsequent period (to the operator, physician, or nearest treatment facility -- e. g., V. G. H.); and during that 24 hours to abstain from flying or diving except as approved by the operator and physician, since altitude depressurization (flying) increases

the rate of bubble formation and repetitive dives increases the onloading of nitrogen at a time when nitrogen from the first exposure may not have been fully off-loaded.

b. Nervous system bends. Bubble formation during decompression can occur in the circulation of the central nervous system, producing deleterious effects by direct mechanical obstruction of blood flow or indirectly by complex interactions with blood components. Commonly these effects occur at the spinal cord level, due to the sluggish blood flow in the extra-vertebral venous system. Spinal involvement can produce a variety of symptoms including numbness, weakness or paralysis of one or more limbs, loss of co-ordination, and changes in bowel or bladder control. Other manifestations of nervous system bends include dizziness with or without "ringing" in the ears and hearing loss (vestibular and/or auditory system involvement) as well as decreased alertness level of consciousness and ability to think clearly. THESE EFFECTS INDICATE AN EMERGENCY! They must be reported immediately since any delay in treatment reduces the likelihood of full recovery. Treatment is immediate recompression, as for limb bends, but with a different treatment table plus or minus the addition of certain drugs (e. g., steroids) and resuscitatory manoeuvres as needed. Although symptoms of nervous system involvement typically develop within the first few minutes after decompression, they may be subtle and/or arise later; the same bends watch and 24 hour surveillance is required as described for limb bends.

c. "The chokes". This condition develops when large numbers of bubbles come out of solution into the venous circulation and overwhelm the capacity of the lungs to filter them out (all venous blood passes from the right side of the heart into the pulmonary circulation, the vessels of which subdivide many times into smaller and smaller vessels where bubbles are trapped). Symptoms include a burning sort of chest pain, shortness of breath and a cough with or without haemoptysis (blood). Treatment is immediate recompression as described for limb and neurologic bends.

d. "Skin bends". Skin bends usually develops after short, deep dry chamber dives and involves bubble formation in the skin during depressurization. It is generally not serious although it may produce significant discomfort, including itchiness and tenderness with a reddening of the skin and/or a splotchy red rash. Although recompression is rarely required, skin bends may be associated with a higher probability of co-existent more serious forms of decompression sickness; hence, symptoms of skin bends must be reported immediately.

e. Dysbaric osteonecrosis. This is a delayed form of decompression sickness in which cysts form in bones, usually near large joints, and commonly in people who dive frequently over several years. It is believed that this condition rarely, if ever, develops unless the diver has missed a decompression stop (see below) during a previous dive. It is slowly progressive, so that continued diving may cause the cysts to enlarge, which is particularly problematic if the cysts come to involve the cartilage within a joint. Periodic long bone x-rays are sometimes used in monitoring for

cyst development among very frequent divers. There is no specific treatment for this condition. Evidence suggesting dysbaric osteonecrosis includes bone or joint pain and/or a fracture of a long bone; cystic bone breaks more easily than normal bone. Management includes orthopaedic consultation and sometimes surgery, as well as a discontinuation of diving.

f. Miscellaneous. Lymphatic congestion (bubbles) may develop on decompression, often manifested by facial swelling. Abdominal pain may sometimes arise and has been attributed to expanding gas in the intestines during decompression. Vague or unusual symptoms may also arise. All symptoms must be reported after decompression (including hypobaric exposure); decisions about treatment and follow-up and management should be left to the physician, not the individual exposed to pressure changes.

g. Prevention of DCS. Controlled depressurization is the key to the prevention of decompression sickness. Diving tables exist (e. g., U. S. Navy tables) which require a specific rate of pressure change (e. g., so many feet per minute ascent from a a dive) as well as "stops" for specified periods at specific depths (pressure levels) during ascent from a dive. Use of these tables is mandatory and is meant to allow a controlled off-loading of nitrogen from the tissues, into the blood and thence into the lungs, such that the risk of bends is reduced to less than 3 - 5%. These tables were derived empirically, that is, based on data from many dives in which various decompression profiles were used and the relative risk of decompression sickness was determined. A further decrease in risk can be obtained by over-estimating the amount of exposure to pressurization (depth and time), and by breathing 100% oxygen during one or more decompression stops (this increases the diffusion gradient for nitrogen both from the tissues to the blood and from the blood to the alveoli of the lungs). During chamber dives, the chamber operator and diving physician select the decompression table (ascent protocol) to be used.

3. Arterial gas embolism. This is a fairly rare, but sometimes lethal condition in which gas bubbles form in the arterial circulation; these bubbles can obstruct blood flow to the heart ("heart attack") or brain ("stroke"), producing emergency situations and/or death. The usual cause is a rapid decompression with the glottis closed (i. e., breath-holding on ascent), such that the air trapped within the lung space, increasing as it does in volume on depressurization, bursts through the lung membranes and enters the arterial circulation there. In this context it is important to remember that the volume of a unit amount of air doubles as pressure goes from twice normal to normal (atmospheric) pressure, i. e., as a diver ascends from 33 feet of seawater to the surface. Evidence of gas embolism is usually dramatic, with the commonest presentation being the unconscious diver on the surface, who has lost consciousness on ascent due to brain damage or a heart attack and who is therefore also at risk of drowning. In the conscious diver, evidence includes impaired alertness or thinking, deficits in movement, speech or sensation, or a symptom complex including chest pain (left or central), shortness of breath and nausea. Treatment includes resuscitation immediately (hence the need for persons trained in cardiopulmonary resuscitation to be on the scene during any hyperbaric exposure — including recreational dives), 100% oxygen and transport in a head-down position (reduces bubble

circulation to the brain) to the nearest hyperbaric treatment facility (hence the need for divers to know in advance where such facilities are and how transportation can be acquired). At S. F. U., we are able to initiate resuscitation and to undertake treatment by recompression within the hyperbaric chamber. The best form of treatment in this and all other conditions is prevention, by proper breathing techniques on ascent and by the exclusion from hyperbaric exposure of all individuals who are known to be predisposed to gas embolism. These people include anyone with a history of surgery in which the chest wall was opened (e. g., bypass grafts) as well as people with a known abnormal communication between the venous circulation by which venous gas bubbles can pass into the arteries (e. g., interventricular septal defect) or with known obstructive lung disease (chronic bronchitis, emphysema, asthma, etc.). People with asthma, for example, must not dive because they run a high risk of gas trapping in a pulmonary segment at depth, due to airway spasm and mucous secretions, with a consequent risk of "burst lung" and gas embolism on decompression. Although periodic chest x-rays with full inhalation and exhalation can help determine which individuals should be excluded from diving, some underlying conditions cannot be determined clinically, so that risks cannot be reduced to zero.

4. Hypoxia or anoxia. Periods of inadequate or zero oxygen supply to bodily tissues can lead to permanent injury or even death. Equipment failure or other accidents, both underwater and in the hyperbaric/hypobaric chamber, can potentially cause hypoxia or anoxia, for which the treatment is clearly the restoration of adequate oxygenation. Prevention requires proper maintenance and use of equipment, with special attention to equipment status before use (e. g., the check-list the chamber operators use) and to the provision of adequate back-up equipment as well as an emergency protocol which is rehearsed and understood beforehand. Individuals certified in cardiopulmonary resuscitation must be present during exposure.

5. Drowning. This special hazard of open-water diving occurs most often as a result of equipment failure or misuse, or as a result of unconsciousness due to one of the other risks listed above. Inadequate equipment preparation and maintenance, sudden weather changes, solo diving, panic, fatigue and hypothermia all contribute to the risk of drowning. Cardiopulmonary resuscitation and transport to a treatment centre are the immediate needs.

6. Oxygen toxicity. Oxygen is a direct toxin to tissues when present in concentrations significantly higher than normal (about 0.21 atmospheres of pressure). Nerve tissue and the lungs are particularly sensitive. Lung toxicity develops gradually, during exposure to hyperbaric oxygen for hours or days. Toxicity varies directly with the oxygen partial pressure and the duration of exposure, and manifests initially as a measurable decrease in vital capacity (reflecting in part a decreased elasticity of the lungs). This decrease is reversible once the hyperbaric oxygenation has ceased.

Oxygen toxicity to the nervous system develops much more rapidly (sometimes within minutes), producing in the extreme a grand-mal type of convulsion which may or may not be preceded by warning signs such as twitching of facial muscles, nausea, numbness and tingling sensations, dizziness, confusion or shortness of breath. The risk of convulsions varies directly with oxygen partial pressure, though there is great inter-individual variation in susceptibility and the risk is increased with exercise and with elevated temperature. Treatment includes basic resuscitation (airway, breathing, circulation and disability), discontinuation of hyperbaric

oxygenation (as soon as that can safely be done) and, occasionally, the administration of medications by a physician. The convulsions stop once hyperbaric oxygenation is discontinued and there are apparently no long-term effects of the convulsion, nor is there an increased risk of epileptic seizures outside the hyperbaric environment in these individuals. Those individuals who have a history of epilepsy, however, must not undertake hyperbaric exposure, both because of the strong likelihood of severe injury or death in the event of seizure onset "at depth" and because of a probable increased risk of oxygen convulsions in these individuals, rendering hyperbaric oxygenation treatment of diving-related accidents problematic.

It should be stressed that it is the partial pressure of oxygen rather than its percentage composition in the gas mixture that is critical in oxygen toxicity. Thus, breathing 40% oxygen in nitrogen at 6 atmospheres of pressure (partial pressure = 2.4 atmospheres) could cause convulsions in some individuals who would not succumb breathing 100% oxygen at 2 atmospheres of pressure (partial pressure = 2.0 atmospheres).

7. Hypothermia. Of special concern in open-water diving in B. C., hypothermia is defined as a lowering of the body core temperature. Mild hypothermia (core temperature in the range of 35 - 36 degrees Celcius) is generally well-tolerated; further decreases in core temperature are associated with impairment of cognitive function (planning, making judgments, responding to emergencies), decreased psychomotor ability, decreasing level of consciousness progressing to loss of consciousness and ultimately death due directly to hypothermia (ventricular fibrillation, or cardiac muscle contraction in an asynchronous manner which fails to pump blood, occurs at temperatures below 28 degrees Celcius) or do drowning related to loss of consciousness. Prevention in cold waters, other than limiting the time of exposure, requires the use of a dry suit or of a wet suit at least 3/8 of an inch thick. Treatment requires re-warming, which on the scene in open-water diving generally starts with body-to-body heat transfer (e. g., the victim and companion in skin contact in a sleeping bag), as well as basic C. P. R., with urgent transfer to a medical facility for further treatment and follow-up.

8. Fire hazards. Fire may occur whenever combustible materials are brought into contact with oxygen, especially hyperbaric oxygen and especially in the presence of sparks. Work in the hyperbaric chamber requires strict adherence to the regulations banning smoking and prohibiting the use of equipment which may produce sparks. Work in the dry chamber requires the wearing of flame-resistant suits and the elimination insofar as is possible of the accumulation of flammable materials inside the chamber. The inside and outside operators must be familiar with the fire-fighting apparatus, which must be kept in working order.

The above material describes the major risks and hazards associated with hypobaric exposure of short duration in a chamber, and with hyperbaric exposure both in the chamber and in open water. Before agreeing to undertake such exposure it is your responsibility to become informed of the risks and hazards well enough to be able to give truly informed consent to such exposure.

* *

I have read and understood this document. Name: _____

Signature: _____

Date: _____

INFORMED CONSENT

ENVIRONMENTAL PHYSIOLOGY UNIT
SCHOOL OF KINESIOLOGY
SIMON FRASER UNIVERSITY

The university and those conducting this project subscribe to the ethical conduct of research and to the protection at all times of the interests, comfort, and safety of subjects. This form and the information it contains are given to you for your own protection and full understanding of the procedures, risks and benefits involved.

Your signature on this form will signify that you have received the document described below regarding this project, that you have received adequate opportunity to consider the information in the document, and that you voluntarily agree to participate in the project.

Having been asked by D. Hedges M.D. of the School of Kinesiology of Simon Fraser University to participate in a research project experiment, I have read the procedures specified in the document entitled:

SUBJECT INFORMATION PACKAGE:
THE EFFECTS OF HYPERCAPNIA ON COGNITIVE AND PSYCHOMOTOR PERFORMANCE IN THE HYPERBARIC ENVIRONMENT

I understand the procedures to be used in this experiment and the personal risks to me in taking part.
I understand that I may withdraw my participation in this experiment at any time.
I also understand that I may register any complaint I might have about the experiment with the chief researcher named above or with Dr. J. Dickinson, Director of the School of Kinesiology, Simon Fraser University.
I may obtain a copy of the results of this study, upon its completion, by contacting Don Hedges M.D. or David Fothergill.

I agree to participate by performing the test battery (as described in the document referred to above) during three hyperbaric chamber dives to 165 fsw breathing normal air, and during three trials at surface while under hypercapnic conditions induced by rebreathing carbon dioxide to end tidal tensions of 35–40 mmHg, 45–50 mmHg and 55–60 mmHg. This testing will take place during the period 1/5/87 to 1/5/88 at the Environmental Physiology Unit (K8615).

SURNAME _____

GIVEN NAME _____

DATE OF BIRTH _____

ADDRESS _____

SIGNATURE _____

DATE _____

SIGNATURE OF WITNESS _____

MEDICAL QUESTIONNAIRE (CONFIDENTIAL)

SURNAME _____

GIVEN NAME _____

ADDRESS _____

PHONE _____

DATE OF BIRTH _____

WEIGHT _____

FAMILY PHYSICIAN _____

ADDRESS: _____

PHONE: _____

MEDICAL HISTORY

Please answer the following questions accurately since they are designed to identify subjects who should not participate within the proposed study. Please place a check-mark by any condition which applies to you. Responses will be viewed only by the principal investigator and departmental physician.

Have you suffered, or do you now suffer from any of the following?

1. asthma. [ ]
2. bronchitis. [ ]
3. tuberculosis, emphysema, fibrosis, pleurisy. [ ]
4. other respiratory abnormality or scarring. [ ]
5. pneumothorax or collapsed lung. [ ]
6. nasal obstruction. [ ]
7. frequent or severe nose bleeds. [ ]
8. frequent cold or sore throats. [ ]
9. chest pain and persistent cough. [ ]

10. coughing up blood (haemoptysis). [ ]

11. heart disease. [ ]

12. high or low blood pressure. [ ]

13. abnormal EKG. [ ]

14. claustrophobia. [ ]

15. alcohol or drug abuse. [ ]

16. allergies. [ ]

17. communicable diseases or contact with patients with same. [ ]

(exclude infectious diseases in the past of which you have been entirely cured).

18. diabetes. [ ]

19. dizziness, fainting spell or fits. [ ]

20. do you smoke or have you smoked in the past? [ ]

21. are you under medical care now or taking medication? [ ]

Please clarify affirmative answers.

I declare the above answers are, to the best of my knowledge, a true and accurate statement of my medical history.

SIGNED ---

DATE ---

WITNESS ---

Workers' Compensation Board of British Columbia Compressed Air Diving Form.

WORKERS' COMPENSATION BOARD
6295 Heather Street Vancouver B.C. V5Z 3L8 Telephone 266-0711 Telex 04-507785

COMPRESSED AIR – DIVING

Surname: _____ Employer: _____

First Names: _____ Male: ☐ Female: ☐

Address: _____ S.I.N. |_|_|_|_|_|_|_|_|_|

_____ Birthdate: |_Day_|_Month_|_Year_|

Position applied for: _____

Dept: _____ Family Physician: _____ M.D.

Location of Operation: _____ Address: _____

Date: |_Day_|_Month_|_Year_|

OCCUPATIONAL HISTORY:

Have you worked in compressed air before? _____

Other jobs: _____

_____

_____

FAMILY HISTORY:

Is there a family history of:	Yes	No	Clarify:
Lung disease	☐	☐	
Mental disorders	☐	☐	
Neurological disease	☐	☐	
Muscular disease	☐	☐	
Other	☐	☐	

MEDICAL HISTORY:

Have you suffered or do you now suffer from any of the following?

	Yes	No		Yes	No
1. The bends	☐	☐	29. Dislocated joints (shoulder, hip)	☐	☐
2. Other compressed air disease or injury	☐	☐	30. Rheumatism, arthritis, gout	☐	☐
3. Frequent colds or sore throat	☐	☐	31. Back disease or injury	☐	☐
4. Hay fever or sinus trouble	☐	☐	32. Hernia	☐	☐
5. Nasal obstruction	☐	☐	33. Severe or frequent headaches	☐	☐
6. Frequent or severe nose bleeds	☐	☐	34. Head injury or concussion	☐	☐
7. Otitis or ruptured ear drum	☐	☐	35. Dizziness, fainting spells or fits	☐	☐
8. Mastoiditis	☐	☐	36. Insomnia, nightmares or sleepwalking	☐	☐
9. Difficulty clearing ears when flying or diving	☐	☐	37. Nervous breakdown	☐	☐
10. Asthma	☐	☐	38. Marked anxiety or depression	☐	☐
11. Chest pain or persistent cough	☐	☐	39. Claustrophobia	☐	☐
12. Pneumothorax or collapsed lung	☐	☐	40. Fear of open spaces or heights	☐	☐
13. Haemoptysis (coughing up blood)	☐	☐	41. Neurological disease	☐	☐
14. Tuberculosis	☐	☐	42. Eye disease or injury	☐	☐
15. Anaemia	☐	☐	43. Sea or other motion sickness	☐	☐
16. Irregular or pounding heart beat	☐	☐	44. Alcohol or drug problem	☐	☐
17. High or low blood pressure	☐	☐	45. Dental bridgework or plates	☐	☐
18. Rheumatic fever	☐	☐	46. Thyroid or other glandular trouble	☐	☐
19. Heart trouble	☐	☐	47. Venereal disease	☐	☐
20. Indigestion or heart burn	☐	☐	48. Skin trouble	☐	☐
21. Peptic or duodenal ulcer	☐	☐	49. Allergies	☐	☐
22. Persistent stomach ache	☐	☐	50. Other serious disease or injury	☐	☐
23. Frequent diarrhea or blood or mucus in stool	☐	☐	51. Have you ever been hospitalized?	☐	☐
24. Jaundice or hepatitis	☐	☐	52. Have you had an abnormal E.K.G.?	☐	☐
25. Diabetes	☐	☐	53. Have you had an abnormal E.E.G.?	☐	☐
26. Kidney or bladder disease	☐	☐	54. Have you been refused or left employment for medical reasons?	☐	☐
27. Blood, sugar or albumin in urine	☐	☐	55. Are you under medical care now?	☐	☐
28. Broken bones	☐	☐			

Clarify affirmative answers by number: _____

_____

_____

_____

Females only: Day Month Year Regular Yes ☐ No ☐ Pregnancies:

L.M.P. |_|_|_| Dysmenorrhoea Yes ☐ No ☐

PERSONAL HISTORY:

Do you use tobacco? _____

alcohol? _____

drugs or medication? _____

Hobbies, Sports, etc. _____

Examination:

General:	Ht:	Wt:	Build:						Temp:
Vision:	R (Far): L (Far):	R (Cor.): L (Cor.):	Jaeger-R: Jaeger-L:		R-Jaeger (cor.) L-Jaeger (cor.)		Colour Fields		
Hearing:	Hz	250	500	1000	2000	3000	4000	6000	8000
	R								
	L								

Eyes:	External:	Light Reflexes	Accommodation:
	Pupils:		Fundi:
Ears:	External:	Canals:	Drums: Valsalva Manoeuvre. R L (observe drums)
Nose:	Shape:	Obstruc:	M.M.:
Mouth:	Teeth:	Dentures:	Gums & M.M.:
	Adequate:	Part: Complete:	Tongue:
Throat:	Tonsils:	Pharynx:	Soft Palate:
Glands:	Thyroid:	Lymph Glands: C: A: I	
C.V.S.:	Apex:	Size:	B. P.
	Sounds:	Murmurs:	After rest (15 min.)
	Pulse:	Exer. (if applicable)	P. after ex. (2 min.)
	Arteries:	(10-15 brisk step-ups onto chair): After 10 seconds — carotid sinus or eyeball pressure:	
Chest:	Symmetry:	Shape:	Resonance:
	Breasts — R: L:	Breath Sounds:	Adventitia:
Abdomen:	Musculature:	Obesity:	Scars:
	Organs:	Other Findings:	
Genitalia:		Hernial Orifices:	
Rectum:	Prostate:	Haemorrhoids:	Other:
Spine:	Shape:	Movements:	
Extremities:	Upper Limbs.	Hands:	
	Lower Limbs.	Feet:	Veins:
	Defects — Functional & Anatomical:		Gait:
Integument:	Scalp, Hair:	Skin, Nails:	Ident. marks & scars:
Reflexes:	Arms: R: SJ / TJ / BJ — L: SJ / TJ / BJ	Legs: R: KJ / AJ — L: KJ / AJ	Romberg:
	Abd. R: L:	Plantar R L	Tremor:
Impression re Psyche:	Co-operative _____ Unco-operative _____ Attitude _____ Appearance _____		

Blood:	HB:	Serology	Sed. Rate:
Urine:	Colour:	Acidity:	Spec. Grav.
	Protein:	Sugar	Micros

X-ray — Chest: full inspiration:		full expiration
— Shoulders:	R:	L:
— Hips:	R:	L:
— Knees:	R:	L:
E.C.G.:		
Compression Chamber Clearance:		
Oxygen Tolerance Test:		
Classification*:	A_____ B_____ C_1_____ C_2_____ D_____	
Next Examination:		

Dated this _____ day of _____ 19____ at _____

(Signature) _____
Physician

* A Fit:
B Permanent Physical Defect (Restricted).
C_1 Correctible Defect (Can continue at work and have treatment).
C_2 Correctible Defect (Not to continue at work until treated).
D Rejected:

Criteria of Rejection, apart from those obvious from examination, X-ray, chamber test, etc.
Absolute: Epilepsy, Pneumothorax, Lung Cyst, Chronic Pulmonary Disease, Vasovagal Attacks, Diabetes, Alcoholism, Arthritis.
Temporary (at discretion of physician): Sinusitis, Perforated Eardrum, Labyrinthitis, Skin rash, Dental disease, History of recurrent Nose and Throat, Pulmonary (tuberculosis), Gastro-intestinal or Genito-urinary disease, any impairment of joint function.

APPENDIX 2:

EXAMPLE TEST BATTERY FOR ASSESSMENT OF COGNITIVE PERFORMANCE

Contents

Word/number paired association list: ..... page 89

Immediate recall response sheet: ..... page 90

Arithmetic test: ..... page 91

Copying test: ..... page 92

Number comparison test: ..... page 93

Letter cancellation test: ..... page 94

Delayed recall response sheet: ..... page 95

FARM 9

GUEST 26

WALK 59

PARENT 78

DEVIL 93

CATTLE 67

MILK 18

FLOOR 42

KING 5

SMOKE 56

SCALE 80

GLASS 76

KING

CATTLE

SCALE

WALK

DEVIL

SMOKE

MILK

FLOOR

PARENT

GLASS

GUEST

FARM

NAME: _____ DATE: _____

CONDITION (circle one):

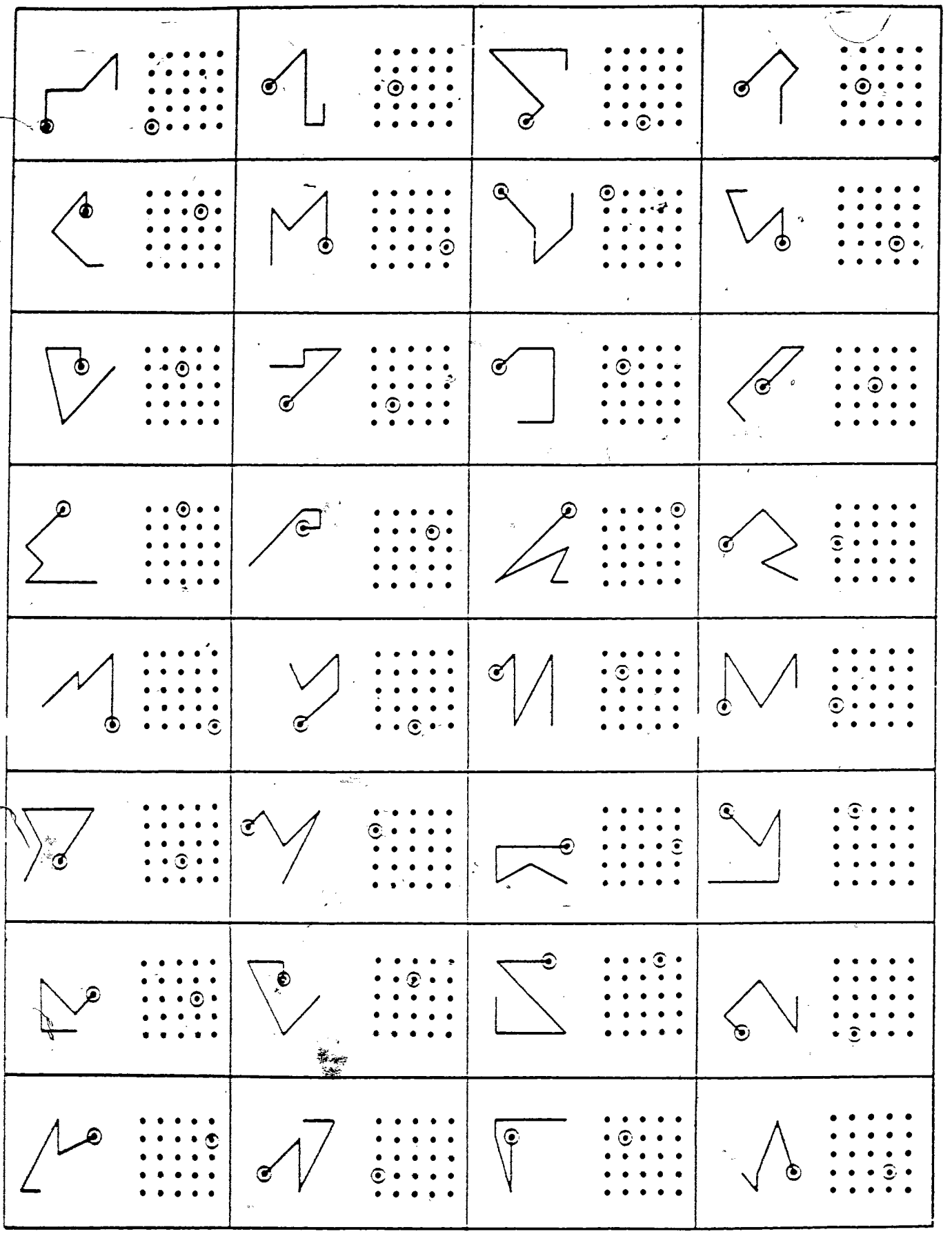
Practice Trial (Trial Number) 1 2 3 4 5 6 7 8 9

Chamber Dive 0 fsw (Trial Number) 1 2 3

Chamber Dive (165 fsw) 1 2 3

TEST 1-1

56-3x7=	51+2x8=	42-3x4=
95+6x7=	74-3x8=	83+9x7=
48-7x3=	27+9x8=	69-4x4=
82+9x8=	94-9x6=	78+6x8=
38-8x3=	75+7x9=	90-3x9=
52+4x9=	71-8x5=	83+4x6=
98-4x9=	34+7x5=	54-6x3=
63+2x8=	59-3x4=	26+8x9=
39-3x8=	95+7x9=	44-4x7=
88+9x8=	47-7x4=	38+4x3=
47-7x5=	28+3x8=	54-7x6=
59+9x6=	42-3x6=	78+8x8=
36-2x6=	78+8x6=	44-6x3=
56+6x8=	38-3x5=	58+5x4=
63-2x9=	47+7x4=	84-4x9=
62+5x6=	83-3x9=	37+6x7=



NAME: _____ DATE: _____

CONDITION (circle one):

Practice Trial (1 ATA) 1 2 3 4 5 6 7 8 9

Surface Test (Trial Number) 1 2 3

Chamber Dive (165 fsw) 1 2 3

TEST 3-1

639	___	639
4714306	___	4715306
65382	___	65372
710	___	710
43210573	___	43210573
6182653905221	___	6182653905221
43270105338	___	43276105338
27109816843	___	27109816853
519605	___	519605
923452170687	___	923452170687
370543141	___	310543141
2570665292	___	2570665292
32018591670	___	32018691670
5471075693	___	5471075683
621532992531	___	621582992531
24179830	___	24179830
70537051248	___	70537057248
7361408	___	7361708
39471307	___	39471507
508264987503	___	508264987503
4930582136	___	4930582136
136031794137	___	136031794137
705731795	___	705736195
38210435512	___	38210535512
414982	___	415982
60971	___	60971
16253948	___	16253948
42018591760	___	43018591760
647107569	___	647107569
721532992531	___	721582992531
341798301	___	341798701
80537051248	___	80537051248
5911306581491	___	5911306581491
83614081	___	83614081
49471307	___	47471307
6082649875	___	6082647875
5930582136	___	5730582136
236031794137	___	236031294137
805731195	___	805131195
48210435512	___	48210435612
405176841309	___	405176841309
80145349786	___	80145349796
53210573	___	53210573
718265390521	___	718265390521
5327010538	___	5327010538
37109816843	___	37189816843
619605	___	619505
123452170687	___	123452190687

NAME: _____ DATE: _____

CONDITION (circle one):

Practice Trial (1 ATA) 1 2 3 4 5 6 7 8 9

Surface Dive 1 ATA (Trial Number) 1 2 3

Chamber Dive (165 fsw) 1 2 3

TEST 4-A

red:	x q z e i z a u l d	blue:	k a n a n o u c m j	red:	a p j k o e y l a n
blue:	i i a y i c y y w f	red:	u y y v v e o i m m	red:	g y e u j m y y y g
red:	e o x c d o e q a x	red:	w e m i m k d i l s	blue:	p i u h o k h x f y
blue:	o e a k e c i e m a	red:	a b i r l k u h g i	red:	m u y s h g v s o y
blue:	i k n i o e o y a v	blue:	i d v d b u a y a i	red:	q d s a y e j u p w
red:	u k p e j y i i d y	blue:	f t w f e x e a z w	red:	a d v y u a o e y u
blue:	p o j w i y p v r g	blue:	t a x y u s e y u a	red:	z k y s b o i i x i
red:	u e i y e u y x l y	blue:	i q c j e o o i v e	red:	e o b z b z w a a c
blue:	s i o r m a y o e d	blue:	e i e u f a b l t l	red:	y u a u e h x i u h

MILK

FARM

GLASS

PARENT

FLOOR

GUEST

CATTLE

SCALE

KING

WALK

DEVIL

SMOKE

APPENDIX 3:
SOURCE CODE LISTING FOR COMPUTER AQUISITION AND ANALYSIS PROGRAMS

A. DIVE33D.C DATA COLLECTION PROGRAM WRITEN IN "C" LANGUAGE

```
#include <aquire.h>
#include <stdio.h>
#include <time.h>
#include <conio.h>

/*************************************************************/
/*      Data buffer info                                     */
/*************************************************************/


int buffer[1500][2];            /* data allocation area      */


struct adinfo {
int channels;
int sweeps;
int speed;
int gains[16];
int order[16];
};

main() {


/*************************************************************/
/*    loop control variables & file pointers                 */
/*************************************************************/

FILE *filepointer;  /* pointer to beginning of cal data file     */
int filehandle;     /* file handle number supplied by labpac     */
int result;         /* result of file operation */
char achar;         /* needed for yes no answers                 */
int handle;         /* filehandle                                */
int parlist;        /* while you are not satisfied with parameter list  */
int loops =0;       /* data aquisition loop control              */
int sweep;
/*************************************************************/
/*      aquire subroutines structures etc.                   */
/*************************************************************/

struct adinfo *par;  /* ad information declaration          */

int total_sweeps;   /* so the saving call know the total number of sweeps   */

/*************************************************************/
/*      Calibration information structure                    */
```

```
/*************************************************************/

struct calinfo {
float calphp,       /*  high pressure calibration coefficient     */
zoffhp,             /*  high pressure zero offset                 */
calco2,             /*  co2 calibration coefficient               */
co2int;             /*  co2 intercept                             */
}cal;

/*************************************************************/
/*      Dive parameter information                                 */
/*************************************************************/

struct diveparam {
float hpr,          /*                                            */
vp,                 /*                                            */
fco2r,              /*                                            */
ddepth;             /*                                            */

int botemp,         /*                                            */
botsiz,             /*                                            */
idepth,             /*                                            */
ipamb;                      /*                                    */

char filename[20],  /*                                            */
name[20],           /*                                            */
date[20];           /*                                            */
}param;

char tempbuf[20];
/*************************************************************/
/*      Begin main routine                                         */
/*************************************************************/

copyright();


printf("\n\n\n*****************************************");

printf("\nUnderwater Ergometer Experiment Program\n");
printf("\nConverted from Ian Wood's Fortran version");
printf("\n  Code by Rob Taylor & David Fothergill");
printf("\n        V 5.0 Feb 8 1988");
printf("\n*****************************************\n\n\n");

/*************************************************************/
/*      Begin calibration information retreaval          */
/*********************************************************************/ if ((filepointer =
fopen("calphp.dat","r" )) == NULL)  /* Go get file calibration values      */
 printf("\n\nCan't open file calphp.dat");   else
   printf("\n\nReading calphp.dat");
  fseek(filepointer,0L,SEEK_SET);
 fscanf(filepointer, "%f", &cal.calphp);
 fscanf(filepointer, "%f", &cal.zoffhp);
```

```
fclose(filepointer);    /* close calibration data file          */

if ((filepointer = fopen("calco2.dat","r" )) == NULL)  /* Go get file calibration values      */
     printf("\nCan't open file calco2.dat");
else
     printf("\nReading calco2.dat");

fseek(filepointer,0L,SEEK_SET);
fscanf(filepointer, "%f", &cal.calco2);
fscanf(filepointer, "%f", &cal.co2int);
    fclose(filepointer);
   printf("\n\n calphp = %f \n zoffhp = %f \n calco2 = %f \n co2int = %f\n\n",
       cal.calphp,cal.zoffhp,cal.calco2,cal.co2int);

printf("\nAre these values O.K. ? (y/n)   ");

scanf("%c",&achar);

if (!(achar == 121)) {

     printf("\n\nBailing out to allow for calibration\n\n");

   return(0);       /* kill this process if cal values are bad       */
  }

/*****************************************************************/
/*                                                               */
/*  Begin data entry                                             */
/*                                                               */
/*****************************************************************/

parlist=1;      /* Assignment to get while loop to execute at least once      */

while (parlist) {

    printf ("\n\nKey in data storage file name: eg. B: _____.dat   ");

     scanf("%s",param.filename);

    printf("\n\nLast name of diver: ");

    scanf("%s",param.name);

     printf("\n\nEnter date < %s > ",_strdate(param.date));

   while (!(kbhit()));

   result = getche();

  if (result > 40)  {

      param.date[0] = result;
      param.date[1] = '\0';
```

```
    scanf("%s",tempbuf);
    strcat(param.date,tempbuf);

    }

  printf("\n\nDepth in meters (integer) : ");

  scanf("%d",&param.idepth);

  printf("\n\nDepth under water (usually .5 meters) : ");

  scanf("%f",&param.ddepth);

  printf("\n\nBarometric pressure (mm Hg.) : ");

  scanf("%d",&param.ipamb);

  printf("\n\nBottle bath temp (degrees c) : ");

  scanf("%d",&param.botemp);

  printf("\n\nBottle size (20,50 or 80 cu.lft.) : ");

  scanf("%d",&param.botsiz);

  printf("\n\nWhat is the saturated water vapour pressure in the lab air :");
  printf("\n\nTemp (degrees c)      Pressure (mm.Hg.)");
 printf("\n\n20                  17.5");
 printf("\n21                 18.7");
 printf("\n22                 19.8");
 printf("\n23                 21.1");
 printf("\n24                 22.4");
 printf("\n25                  23.8\n\n");

 scanf("%f",&param.vp);

   printf("\n\nFilename: %s",param.filename);
  printf("\nName: %s",param.name);
  printf("\nDate: %s",param.date);
  printf("\nChamber depth: %d",param.idepth);
  printf("\nDiver (lung) depth: %f",param.ddepth);
  printf("\nBarometric Press.: %d",param.ipamb);
 printf("\nBottle bath temp.: %d",param.botemp);
  printf("\nBottle size: %d",param.botsiz);
  printf("\nVapour pressure: %f",param.vp);

scanf("%c",&achar);

  printf("\n\nAre these values correct? (y/n): ");


scanf("%c",&achar);

if (achar == 121)
```

```
        parlist=0;

    } /* end parameter list                                              */

/*****************************************************/
/*                                                                     */
/*  end parameter list entry     */
/*                                                                     */
/*****************************************************/

    if ((filepointer = fopen(param.filename,"w" )) == NULL) {/*  open file for writing */
       printf("\n\nCan't open file %s",param.filename);
       return(0);
       }

     fprintf(filepointer,"Diver's Last Name: %s\n",param.name);
     fprintf(filepointer,"Date of Dive: %s\n",param.date);
     fprintf(filepointer,"Depth of Dive: %d\n",param.idepth);
     fprintf(filepointer,"Depth Under Water: %f\n",param.ddepth);
     fprintf(filepointer,"Barometric Pressure %d\n",param.ipamb);
     fprintf(filepointer,"Bottle Bath Temp: %d\n",param.botemp);
     fprintf(filepointer,"Bottle Size: %d\n",param.botsiz);
     fprintf(filepointer,"Saturated V.P.: %f\n",param.vp);


/*****************************************************/
/*                                                                     */
/*  Begin data collection routine                                      */
/*                                                                     */
/*****************************************************/



printf("\n\nRemember co2 is on channel 6 ");
printf("\n\nand HP is on channel 2");
printf("\n\nHit return key to start test ");
scanf("%c",&achar);     /* clears kbd buffer                          */
scanf("%c",&achar);     /* clears kbd buffer                          */

printf("\n************ Aquisition started ***********\n");

/*****************************************************/
/* First 30 second sweep starts here                                   */
/*****************************************************/

par ->order[0] = 6;     /*  Put order of sweep information in here */
par ->order[1] = 2;

par ->gains[6] = 0;          /*     put your gain codes in here */
par ->gains[2] = 0;

LP(RESET);          /* Reset labpac arrays                             */
```

```
LP(AIINIT,ATOD,16,0,par ->gains);       /* Initialize analog sampling      */

LP(TIINIT,TIMER);              /*  Initialize  timer                      */

LP(TIST, 5, 15, 3);             /* start timer at 33 hz          */
LP(TIST, 1, 0, 0);              /* keep track of timer ticks   */

for(sweep=0; sweep<1000; ++sweep) {

    labpac(TISTAT,1,sweep);          /* wait 1/33 second           */

    buffer[sweep][0] = labpac(AIRAW, 6);  /* Save data in arrays     */
    buffer[sweep][1] = labpac(AIRAW, 2);

    buffer[sweep][0] = (cal.calco2 * buffer[sweep][0]) + cal.co2int;
    buffer[sweep][1] = cal.calphp * (buffer[sweep][1] - cal.zoffhp);
  }

total_sweeps = 1000;

/*************************************************************/
/*      Main 1 minute data aquisition loop (collects 45sec of data) */

/*************************************************************/

while (loops < 20) {

   LP(TIINIT,TIMER);
   LP(TIST, 5, 15, 3);
   LP(TIST, 1, 0, 0);
      fprintf(filepointer,"\n\nData segment number: %d \n",loops);

  printf("\n***************** File Save *****************\n");

     for(sweep=0;sweep<total_sweeps;++sweep){
       fprintf(filepointer,"%d   %d\n",buffer[sweep][0],buffer[sweep][1]);
       }

    LP(TISTAT, 1, 500);  /* waits 15 seconds while data saved to disc */
   if (kbhit()) {

       printf("\n\nData saved under filename: %s\n",param.filename);
       printf("\n************ End Data Aquisition ************\n");

       fclose(filepointer);
       return(0);  /* a way out of data aquisition at < 20 minutes */
       }


  printf("\n************* 15 SECOND WARNING ***\n");

  LP(TIST, 5, 15, 3);          /* start timer at 33 hz          */
```

```
    LP(TIST, 1, 0, 0);                  /* keep track of timer ticks    */

     for(sweep=0; sweep<300; ++sweep) {

        labpac(TISTAT,1,sweep);          /* wait 1/33 second             */

        buffer[sweep][0] = labpac(AIRAW, 6);    /* Save data in arrays      */
        buffer[sweep][1] = labpac(AIRAW, 2);
        buffer[sweep][0] = (cal.calco2 * buffer[sweep][0]) + cal.co2int;
        buffer[sweep][1] = cal.calphp * (buffer[sweep][1] - cal.zoffhp);
        }

   printf("\n************** 6 SECOND WARNING **************\n");

     for(sweep=300; sweep<500; ++sweep) {

        labpac(TISTAT,1,sweep);          /* wait 1/33 second             */

        buffer[sweep][0] = labpac(AIRAW, 6);    /* Save data in arrays      */
        buffer[sweep][1] = labpac(AIRAW, 2);
        buffer[sweep][0] = (cal.calco2 * buffer[sweep][0]) + cal.co2int;
        buffer[sweep][1] = cal.calphp * (buffer[sweep][1] - cal.zoffhp);
        }

   printf("\7\n************** CHANGE BOTTLES NOW! ***************************\n");

     for(sweep=500; sweep<1500; ++sweep) {

        labpac(TISTAT,1,sweep);          /* wait 1/33 second             */

        buffer[sweep][0] = labpac(AIRAW, 6);    /* Save data in arrays      */
        buffer[sweep][1] = labpac(AIRAW, 2);
        buffer[sweep][0] = (cal.calco2 * buffer[sweep][0]) + cal.co2int;
        buffer[sweep][1] = cal.calphp * (buffer[sweep][1] - cal.zoffhp);
        }

    loops++;

     total_sweeps = 1500;  /* this is the total no sweeps that will be written */
                    /* to filename, this is needed so all data is saved.*/

  }

/*****************************************************/
/*      End main while loop                          */
/*****************************************************/

printf("\n\nData saved under filename: %s\n",param.filename);
printf("\n\nTwenty Minute limit expired - End aquisition");
fclose(filepointer);
return(0);  /* a way out of data aquisition at 20 minutes */

}               /* End main code                                */
```

B. DATA ANALYSIS PROGRAM DIVEAN.C FOR PHYSIOLOGICAL DATA COLLECTED USING DIVE33D.C

```
/*****************************************************/
/*          DATA ANALYSIS PROGRAM FOR DATA COLLECTED  */
/*              USING THE PROGRAM DIVE33D             */
/*****************************************************/
#define DEBUG
# include <stdio.h>

      int data[1500][2];          /* data array size   */
float datasmooth[1500][2];   /* smoothed data array size  */

main() {
/*************************************************************/
/*      dive parameter information from data file            */
/*************************************************************/

struct diveparam {

  char fileip[20];    /*     name of file to be read   */
  char fileop[20];        /*       name of file for o/p     */
  char name[20];        /*         divers name             */
  char date[20];      /*    date of dive             */
  int botsiz;          /* bottle size cuft   */
  int botemp;          /* water bath temp          */
  int idepth;          /* depth of dive            */
  float vp;            /* vapour pressure          */
  int ipamb;           /* barometric pressure      */
  float ddepth;        /* depth under water        */
  }param;

/*****************************************************/
/*   variable declaration for main program           */
/*****************************************************/

struct timevals {

  int timehphi[21];     /* time of hp high peak     */
  int timehplo[21];     /* time of hp low peak      */
  float timedif;        /* time between hphi peak and hplo trough */
  }tval;

struct ventvals {
  float hphi[21];      /* hp peak value      */
  float hplo[21];      /* hp trough value          */
  float sumbf,          /* total no breaths for test  */
  meanbf;              /* mean breathing frequency */

  int framehi,         /* frame no. of highest hp val */
  framelo;             /* frame no. of lowest hp val  */
  int bf[20];          /* breathing frequency      */
  float hpdrp,         /* raw pressure drop val    */
  hpdrop,              /* corrected pressure drop val */
```

```
  viat,        /* Vi (ATPS)                */
  ventm,       /* Vi (BTPS)                */
  tidvol,      /* Vt (BTPS)                */
  sumtidvol,   /* sum of Vt for test       */
  sumventm,    /* sum of Vi for test       */
  meanvent,    /* mean Vi for test         */
  meantidvol;  /* mean Vt for test         */
 }vval;

struct co2val  {
  float pet,          /* end tidal co2 value      */
  btps,               /*     BTPS factor          */
  testsumpic,         /* sum of all petco2 vals for test  */
  testsumpet,         /* sum of all pico2 vals for test   */
  petco,              /* real petco2 val for each min     */
  pico,               /* real pico2 val for each min      */
  pic,                /* pico2 value              */
  avpet,              /* uncorrected petco2 val   */
  avpic,              /* uncorrected pico2  val   */
  sumpet,             /* raw val for sum of petco2 vals   */
  sumpic,             /* raw val for sum of pico2 vals    */
  meanpet,            /* average Petco2 for test          */
  meanpic,            /* average pico2 for test           */
  pdm,                /* total depth of diver (msw)       */
  pd,                 /* ambient pressure on diver (mmHg) */
  botvol;             /* bottle vol (litres)              */
  int low,            /* number of co2 troughs/min        */
  high;               /* number of co2 peaks/min          */
 }cval;

FILE  *fileinpointer;
FILE  *fileoutpointer;
int  parlist;
int  minstep;         /* total time for test */
int  loops;           /* loop counter        */
int  skp;             /* counter for no. of bad mins of hp data*/
int  sweep;           /* minute counter      */
float cotrig;         /* co2 trigger val     */
float hppktrig;       /* hp peak trigger     */
float hptrig;         /* hp trigger val      */
char garb[20];        /* garbage string      */
char achar;           /* for yes and no answers */

/*****************************************************/
/*                BEGIN MAIN ROUTINE                 */
/*****************************************************/
printf("\n\n\n ***************************************************");
printf("\n          This program analyses data collected by the\n");
printf("\n                aquisition program DIVE33D\n");
printf("\n                  v3.0 August 1988.");
printf("\n                Code by David Fothergill ");
printf("\n ***************************************************\n\n\n");

/*************************************/
```

```
/*          open data file for reading      */
/*************************************/
parlist=1; /* assignment to get while loop to execute at least once*/
while (parlist) {
   printf("\n\n Type in data file to be read: e.g. c:<filename>.dat ");
    scanf("%s",param.fileip);
    printf("\n\n\n Input number of full minutes of data collection period ");
    scanf("%d",&minstep);
    printf("\n\n Input co2 trigger value :");
    scanf("%f",&cotrig);
    printf("\n\n input high pressure trough trigger value :");
    scanf("%f",&hptrig);
    printf("\n\n input high pressure peak trigger value :");
    scanf("%f",&hppktrig);
   printf("\n\n\n Type in o/p file name: e.g. c:<filename>.dat ");
    scanf("%s",param.fileop);
    printf("\n\n input filename: %s ",param.fileip);
    printf("\n Total experiment time (min): %d",minstep);
   printf("\n Co2 trigger val : %f",cotrig);
   printf("\n Hp trigger val : %f",hptrig);
    printf("\n O/P filename: %s ",param.fileop);
    scanf("%c",&achar);
    printf("\n\n Is this info correct ? (y/n):");
    scanf("%c",&achar);
  if (achar==121)        /* ans=y */
  parlist=0;
 }
/* end of input loop  */

if ((fileinpointer = fopen(param.fileip,"r")) == NULL) {

   printf("\n Error: can't open i/p file: %s ",param.fileip);
  exit();
 }
else
 printf("\n\n Reading file:%s       ",param.fileip);

/*************************************************************/
/* Read in data header from input file                       */
/*************************************************************/


fscanf(fileinpointer,"%s  %s  %s  %s\n",garb,garb,garb,param.name);
fscanf(fileinpointer,"%s  %s  %s  %s\n",garb,garb,garb,param.date);
fscanf(fileinpointer,"%s  %s  %s  %d\n",garb,garb,garb,&param.idepth);
fscanf(fileinpointer,"%s  %s  %s  %f\n",garb,garb,garb,&param.ddepth);
fscanf(fileinpointer,"%s  %s  %d\n",garb,garb,&param.ipamb);
fscanf(fileinpointer,"%s  %s  %s  %d\n",garb,garb,garb,&param.botemp);
fscanf(fileinpointer,"%s  %s  %d\n",garb,garb,&param.botsiz);
fscanf(fileinpointer,"%s  %s  %f\n",garb,garb,&param.vp);

/*************************************************************/
/*                    Pressure   Calculations                */
```

```
/*****************************************************************/

cval.pdm =(param.idepth+param.ddepth); /*cval.pdm = total depth of diver (msw)*/
cval.pd =(cval.pdm*76.0);       /* cval.pdm(msw*76.0 mmHg/msw)

/*****************************************************************/
/*  Convert bottle size to bottle volume                         */
/*****************************************************************/
switch(param.botsiz)    {

   case 20:{
      cval.botvol=3.678;
     break;
    }
   case 50:{
      cval.botvol=7.854;
     break;
    }
   case 80:{
      cval.botvol=12.01;
     break;
    }
   default:{
      printf("ERROR: Invalid bottle size found");
     exit();
    }
  }
printf("\n\n  bottle  volume  :%f",cval.botvol);

/*****************************************************/
/*  open o/p file and store values in a header file  */
/*****************************************************/

if((fileoutpointer  =  fopen(param.fileop,"w"))==NULL)
                                    {
   printf("\n\n Can't open file %s",param.fileop);
  return(0);
 }
fprintf(fileoutpointer,"%s\n",param.name);
fprintf(fileoutpointer,"%s\n",param.date);
fprintf(fileoutpointer,"dive  depth  (mmHg):  %f\n",cval.pd);
fprintf(fileoutpointer,"barometric  pressure:  %d\n",param.ipamb);
fprintf(fileoutpointer,"bottle  bath  temp:  %d\n",param.botemp);
fprintf(fileoutpointer,"bottle  vol:  %f\n",cval.botvol);
fprintf(fileoutpointer,"sat  v.p.  :  %f\n",param.vp);

/*****************************************************/
/* scanning first 30 secs of data file               */
/*****************************************************/
sweep=0;

fscanf(fileinpointer,"%s  %s  %s  %s\n",garb,garb,garb,garb);
```

```
for (loops=0;loops<=1000;++loops) {

   fscanf(fileinpointer,"%d  %d\n",&data[loops][0],&data[loops][1]);
 }

for(loops=3; loops< 997; ++loops ) {

   datasmooth[loops][0] = (((data[loops-2][0])/13.0)+
    ((data[loops-1][0])*3.0/13.0)+((data[loops][0])*5.0/13.0)+
    ((data[loops+1][0])*3.0/13.0)+((data[loops+2][0])/13.0));

   datasmooth[loops][1] = (((data[loops-2][1])/13.0)+
    ((data[loops-1][1])*3.0/13.0)+((data[loops][1])*5.0/13.0)+
    ((data[loops+1][1])*3.0/13.0)+((data[loops+2][1])/13.0));
 }

/*********************************************************/
/*                                                       */
/*          loop routines to extract peaks and troughs           */
/*                                                       */
/*********************************************************/

cval.low=0;
cval.pic=0.0;
cval.pet=0.0;
cval.sumpic=0.0;
cval.sumpet=0.0;
cval.high=0;
loops=4;
while (loops<997)      {              /* while array is not overflowing */
                                      /* detect high point  */
   while (((datasmooth[loops][0]+cotrig) >=datasmooth[loops-1][0])

  && (loops<997)) {

    cval.pet = datasmooth[loops][0]; /* hold highest val */
   ++loops;
  }


  if ((!(loops==4) && (!(loops==997))))    { /* don't sum a high point */
                                             /* if pattern started on down */
                                             /* slope or if it is at end */
                                             /* of array  */
    ++cval.high;
     cval.sumpet = (cval.sumpet + cval.pet);
  }

#ifdef DEBUG
printf("\n co2 peak value : %f",cval.pet);
#endif
  /* detect low point */
   while (( (datasmooth[loops][0]-cotrig) <=datasmooth[loops-1][0])
```

```
  && (loops<997)) {

     cval.pic = datasmooth[loops][0];      /* hold lowest val */
    ++loops;
   }

 printf("\n trough value : %f",cval.pic);

   if (!(loops==997)) { /* don't sum low point if you are at
                           the end of the array */

     ++cval.low;
      cval.sumpic = (cval.sumpic + cval.pic);
   }
  }       /******* end of first minute data loop ***************/
++sweep;

/*************************************************/
/*              calculations for hp and pco2              */
/*************************************************/
cval.avpet=0.0;
cval.avpic=0.0;
vval.sumbf=0;
vval.hphi[sweep]=0.0;
loops=0;
vval.hphi[sweep] =((datasmooth[loops+3][1]+datasmooth[loops+4][1]
+datasmooth[loops+5][1])/3.0);
vval.bf[sweep]=0;
tval.timehphi[sweep]= 0.0;
vval.bf[sweep] = (cval.low + cval.high + 1);
vval.sumbf = (vval.sumbf + vval.bf[sweep]);
cval.avpic = (cval.sumpic/cval.low);
cval.avpet = (cval.sumpet/cval.high);
cval.btps = ((cval.pd+param.ipamb-47.0)*param.ipamb)/
          ((param.ipamb - param.vp) * 10000.0);
cval.pico = (cval.avpic*cval.btps);
cval.petco = (cval.avpet*cval.btps);
cval.testsumpic = (cval.testsumpic + cval.pico);
cval.testsumpet = (cval.testsumpet + cval.petco);


printf("\n                    Writing to output file");

/*************************************/
/* store values in o/p file & print to screen */
/*************************************/

printf("\n\nTime     Petco2  Pico2   bf       ");
printf("hphi          timhphi   hplo   timhplo\n");
printf("%d\t %2.2f\t %2.2f\t %d\t %4.1f\t %d\n",
       sweep,cval.petco,cval.pico,vval.bf[sweep],
       vval.hphi[sweep],tval.timehphi[sweep]);
fprintf(fileoutpointer,"\n\nTime      Petco2  Pico2   bf       hphi");
fprintf(fileoutpointer,"          timehphi   hplo   timehplo\n");
```

```
fprintf(fileoutpointer,"%d\t %2.2f\t %2.2f\t %d\t %4.2f\t %d\n",
        sweep,cval.petco,cval.pico,vval.bf[sweep],
        vval.hphi[sweep],tval.timehphi[sweep]);


printf("\n            Reading Data From Input File ");
++sweep;

/*************************************************/
/*      all other minutes data read in here      */
/*************************************************/

for(sweep=2; sweep<=minstep; ++sweep) {

   fscanf(fileinpointer,"%s %s %s %s\n",garb,garb,garb,garb);
          /* ignores file marker at top of data */

  for(loops=0;loops<=1500; ++loops)  /* read in data */  {
      fscanf(fileinpointer,"%d %d\n",&data[loops][0],&data[loops][1]);

       datasmooth[loops][0]=0.0;

         }

/*************************************************/
/*       set variables back to zero              */
/*************************************************/
cval.pic=0.0;
vval.bf[sweep]=0;
cval.pet=0.0;
cval.sumpet=0.0;
cval.sumpic=0.0;
cval.petco=0.0;
cval.pico=0.0;
vval.hplo[sweep]=0.0;
vval.hphi[sweep]=0.0;
tval.timehphi[sweep]=0.0;
tval.timehplo[sweep]=0.0;
vval.framehi=0;
vval.framelo=0;
cval.avpic=0.0;
cval.avpet=0.0;
cval.high=0;
cval.low=0;

/*****************************************/
/*    data smoothing loop                */
/*****************************************/

for(loops = 2; loops< 1497; ++loops) {

   datasmooth[loops][0] =(((data[loops-2][0])/13.0)+
   ((data[loops-1][0])*3/13.0)+((data[loops][0])*5/13.0)+
   ((data[loops+1][0])*3/13.0)+((data[loops+2][0])/13.0));
```

```
    datasmooth[loops][1] =(((data[loops-2][1])/13.0)+
    ((data[loops-1][1])*3/13.0)+((data[loops][1])*5/13.0)+
    ((data[loops+1][1])*3/13.0)+((data[loops+2][1])/13.0));


 }

/*************************************/
/* Loops for picking out troughs and peaks */
/*************************************/
loops=500;
while (loops<1496)                    {
   while(((datasmooth[loops][0] + cotrig)>=datasmooth[loops-1][0])

 &&(loops<1496)) {
    cval.pet = datasmooth[loops][0];
  ++loops;

  }
#ifdef DEBUG
printf("\nCo2 peak val : %f",cval.pet);
#endif
  if ((!(loops==500) && (!(loops==1496))))    {
    ++cval.high;
     cval.sumpet =(cval.sumpet + cval.pet);
  }
   /* detect low point */

   while (((datasmooth[loops][0] - cotrig)<=datasmooth[loops-1][0])

&&(loops<1496)) {
    cval.pic = datasmooth[loops][0];
   ++loops;
  }
#ifdef DEBUG
printf("\n   trough val : %f",cval.pic);
#endif


  if ((!(loops==500) && (!(loops==1496))))   {
   ++cval.low;
    cval.sumpic = cval.sumpic + cval.pic;
  }
  }  /* end of loop for co2 data */

    /* high pressure data loop */

loops=100;
while  (((datasmooth[loops][1]-hptrig)  <=datasmooth[loops-1][1])

 && (loops<1496)) {
```

```
    vval.hplo[sweep] = datasmooth[loops][1];
   ++loops;
}

printf("\n\n high pressure trough val : %f",vval.hplo[sweep]);

  if ((!(loops==100)) &&  (!(loops==1496))) {
   vval.framelo = loops;
  }
  if (loops==1496) { vval.framelo = 1496;}

printf("\nhp frame val : %d",vval.framelo);

   /* detect low point */
  while (((datasmooth[loops][1] + hppktrig) >=datasmooth[loops-1][1])

&&(loops<1496)) {
    vval.hphi[sweep] = datasmooth[loops][1];
   ++loops;
}

printf("\n\n hp peak val      : %f",vval.hphi[sweep]);

  if ((!(loops==10)) && (!(loops==1496))) {
   vval.framehi = loops;
  }
printf("\n\n hp value frame number : %d",vval.framehi);


/*******************************************************/
/*                  Begin Co2 calculations             */
/*******************************************************/
cval.avpic = cval.sumpic/cval.low;
cval.avpet = cval.sumpet/cval.high;
cval.pico = cval.avpic*cval.btps;
cval.petco = cval.avpet*cval.btps;
cval.testsumpic = cval.testsumpic + cval.pico;
cval.testsumpet = cval.testsumpet + cval.petco;
vval.bf[sweep] = cval.low + cval.high + 1;
vval.sumbf =vval.sumbf + vval.bf[sweep];

tval.timehphi[sweep] =((sweep * 60.0) + ((vval.framehi - 500.0)/33.3)-60.0);
tval.timehplo[sweep] =((sweep * 60.0) + ((vval.framelo - 500.0)/33.3)-60.0);

/*******************************************/
/*     write values to o/p file & screen   */
/*******************************************/

printf("\n\nTime    PetCo2   Pico2   bf      hphi ");
printf("         timehphi   hplo   timehplo\n");
printf("%d\t %2.2f\t %2.2f\t %d\t %4.1f\t\t %d\t %4.1f\t %d\n",
sweep,cval.petco,cval.pico,vval.bf[sweep],vval.hphi[sweep],
tval.timehphi[sweep],vval.hplo[sweep],
tval.timehplo[sweep]);
```

```
fprintf(fileoutpointer,"%d\t %2.2f\t %2.2f\t %d\t %4.2f\t %d\t %4.1f\t %d\n",
sweep,cval.petco,cval.pico,vval.bf[sweep],vval.hphi[sweep],
tval.timehphi[sweep],vval.hplo[sweep],
tval.timehplo[sweep]);
}
    /**** END OF SAMPLING and CRUNCHING LOOP ****/

printf("\n\nTime   Vi (BTPS)   Vt (BTPS)\n");
fprintf(fileoutpointer,"\n\nTime   Vi (BTPS)   Vt (BTPS)\n");

/*********************************************/
/*   final calculations for ventilation      */
/*********************************************/
vval.sumtidvol=0.0;
vval.sumventm=0.0;
skp=0;

for (sweep=1; sweep<=minstep; ++sweep) {

   if (vval.hphi[sweep] > 1.0)   {

   tval.timedif  = ((tval.timehplo[sweep+1] - tval.timehphi[sweep])/60.0);

       if (tval.timedif > 0.0)      {
   vval.hpdrp  = ((vval.hphi[sweep] - vval.hplo[sweep+1])/tval.timedif);

  /* correct pressure drop using heat effect reg equatns  */

   vval.hpdrop = (0.759*vval.hpdrp + 0.00012);

  /* calculate vol of air used in mins from corrected pressure drop */

  vval.viat = (vval.hpdrop * cval.botvol/14.70); /* ATPS  */
  vval.ventm = vval.viat * 760.0/(cval.pd + param.ipamb - 47.0)*310.0/
               (273.0 + param.botemp);
  vval.tidvol = (vval.ventm / vval.bf[sweep]);
  vval.sumtidvol = (vval.sumtidvol + vval.tidvol);
  vval.sumventm = (vval.sumventm + vval.ventm);


    /* send values to screen and o/p file */

    printf("%d\t  %3.2f\t  %1.2f\n",sweep,vval.ventm,vval.tidvol);
    fprintf(fileoutpointer,"%d\t  %3.2f\t  %1.2f\n",sweep,vval.ventm,vval.tidvol);
    }
 }
   if (vval.hphi[sweep]== 0.0 || tval.timedif < 0.0) { ++skp; }
  } /* end of for-sweep=1 to minstep */

vval.meanvent  =  vval.sumventm/(minstep-skp);
vval.meantidvol  =  vval.sumtidvol/(minstep-skp);
cval.meanpet=  cval.testsumpet/minstep;
cval.meanpic=  cval.testsumpic/minstep;
vval.meanbf  =  vval.sumbf/minstep;
```

```
/*** write these values to bottom of o/p file ****/

printf("\n\n Mean Petco2 value for test (mmHg): %2.2f\n",cval.meanpet);
printf(" Mean Pico2 value for test (mmHg): %2.2f\n",cval.meanpic);
printf(" Mean Vi for test l per min(BTPS): %3.2f\n",vval.meanvent);
printf(" Mean bf for test brths/min : %2.1f\n",vval.meanbf);
printf(" Mean Vt for test l(BTPS): %1.2f\n",vval.meantidvol);
fprintf(fileoutpointer,"mean petco2 value for test (mmHg): %2.2f\n",cval.meanpet);
fprintf(fileoutpointer,"mean pico2 value for test (mmHg): %2.2f\n",cval.meanpic);
fprintf(fileoutpointer,"mean Vi for test (l per min) : %3.3f\n",vval.meanvent);
fprintf(fileoutpointer,"mean bf for test (brths per min) : %2.1f\n", vval.meanbf);
fprintf(fileoutpointer,"mean Vt for test (litres): %1.2f",vval.meantidvol);

/***************** close files **********************/

 printf("\n Analysis completed and stored in file: %s",param.fileop);

 fclose(fileinpointer);
 fclose(fileoutpointer);

 } /* End of program */
```

APPENDIX 4:

SUMMARY TABLES SHOWING CO_2 AND VENTILATORY RESPONSES FOR INDIVIDUAL SUBJECTS UNDER ALL EXPERIMENTAL CONDITIONS.

Table 6.1: **Mean end-tidal PCO_2 tensions while completing the performance test battery at 1 and 6 ATA under the low (non-rebreathing condition), medium and high levels of hypercapnia. Values are means ± S.E.M. (mmHg).**

	1 ATA			6 ATA		
	Level of Hypercapnia					
	LOW	MEDIUM	HIGH	LOW	MEDIUM	HIGH
Subject Number						
1	28.10 ±0.53	47.58 ±0.44	55.24 ±0.32	29.24 ±1.01	49.92 ±0.38	58.08 ±0.48
2	23.28 ±1.09	47.03 ±0.22	56.43 ±0.45	24.13 ±0.97	49.41 ±0.64	60.01 ±0.64
3	30.76 ±0.56	47.72 ±0.25	54.40 ±1.02	33.03 ±0.50	48.79 ±0.61	60.34 ±0.48
4	30.15 ±0.80	46.24 ±0.46	54.30 ±0.53	28.60 ±0.98	47.24 ±0.68	58.19 ±0.61
5	21.88 ±1.01	44.84 ±0.37	56.14 ±0.37	29.39 ±0.72	48.21 ±0.41	57.87 ±0.35
6	22.27 ±0.53	47.24 ±0.41	55.38 ±0.52	25.82 ±1.31	46.48 ±0.41	58.07 ±0.37
7	36.07 ±0.67	47.31 ±0.37	56.25 ±0.45	30.51 ±0.50	47.01 ±0.25	59.29 ±0.57
8	34.57 ±0.54	44.88 ±0.33	55.23 ±0.65	25.53 ±0.69	45.89 ±0.41	57.01 ±0.41
9	25.50 ±0.58	45.47 ±0.30	54.48 ±0.50	24.44 ±0.48	47.10 ±0.55	58.04 ±0.60
10	33.86 ±0.45	45.67 ±0.58	49.96 ±0.72	35.29 ±0.69	48.96 ±0.50	59.92 ±0.53
11	28.72 ±1.50	49.11 ±0.34	56.95 ±0.50	34.00 ±0.75	48.39 ±0.52	58.93 ±0.72
12	32.85 ±1.38	48.90 ±0.48	56.83 ±0.32	29.15 ±0.66	48.60 ±0.43	57.64 ±0.53
Mean	29.00	46.83	55.13	29.09	47.92	58.62
S.E.M.	1.42	0.42	0.54	1.05	0.33	0.31

Table 6.2: **Mean inspired PCO_2 tensions while completing the performance test battery at 1 and 6 ATA under the low (non-rebreathing condition), medium and high levels of hypercapnia. Values are means ± S.E.M. (mmHg)**

	1 ATA			6 ATA		
	End-tidal CO_2 tension (mmHg)					
	LOW 29.0*	MEDIUM 46.8*	HIGH 55.1*	LOW 29.1*	MEDIUM 47.9*	HIGH 58.6*
Subject Number						
1	3.36 ±0.26	42.21 ±0.65	46.29 ±0.15	3.34 ±0.17	25.46 ±0.87	43.01 ±1.35
2	1.49 ±0.37	37.84 ±0.97	53.24 ±0.41	3.23 ±0.50	24.14 ±1.38	26.93 ±2.13
3	1.21 ±0.02	36.92 ±0.75	44.18 ±0.60	4.78 ±0.82	15.78 ±0.63	17.51 ±1.12
4	1.08 ±0.06	39.28 ±0.86	51.27 ±0.81	2.67 ±0.22	12.24 ±2.09	13.06 ±1.27
5	5.27 ±0.50	40.00 ±0.71	52.17 ±0.43	3.96 ±1.31	32.83 ±1.69	47.51 ±1.08
6	1.18 ±0.06	41.67 ±0.29	50.12 ±0.86	2.50 ±0.19	12.83 ±1.04	19.08 ±1.97
7	1.06 ±0.04	34.81 ±0.48	51.89 ±0.52	3.01 ±0.82	17.90 ±0.97	32.56 ±1.22
8	2.38 ±1.73	37.23 ±0.76	49.30 ±1.05	2.24 ±0.15	17.10 ±1.01	17.03 ±1.73
9	0.33 ±0.01	39.86 ±0.55	48.35 ±0.65	2.08 ±0.10	24.18 ±2.23	44.04 ±1.52
10	2.02 ±1.10	36.36 ±0.53	43.85 ±0.78	2.74 ±0.25	14.58 ±0.79	35.08 ±1.27
11	2.70 ±0.78	38.20 ±0.70	53.58 ±0.50	2.85 ±0.22	21.87 ±0.43	21.46 ±1.15
12	1.41 ±0.06	42.59 ±0.76	51.84 ±0.66	2.95 ±0.10	21.48 ±1.47	30.42 ±0.91
Mean	2.00	38.61	49.67	3.03	20.06	28.97
S.E.M.	0.42	0.73	0.97	0.22	1.75	3.40

* Mean $P_{ET}CO_2$ tensions recorded for the 12 subjects (*cf.*, Table 6.1)

Table 6.3: **Mean inspired minute ventilation response to various levels of hypercapnia at 1 and 6 ATA of pressure while completing the performace test battery. Values are means ± S.E.M. (liters/minute BTPS).**

	1 ATA			6 ATA		
	End-tidal CO_2 tension (mmHg)					
	LOW	MEDIUM	HIGH	LOW	MEDIUM	HIGH
	29.0*	46.8*	55.1*	29.1*	47.9*	58.6*
Subject Number						
1	6.19 ±1.40	42.49 ±2.24	69.09 ±2.07	17.84 ±0.80	34.20 ±2.34	38.46 ±2.19
2	13.84 ±2.10	31.47 ±1.97	43.15 ±2.91	21.48 ±2.49	31.62 ±2.22	35.29 ±3.53
3	4.59 ±0.47	24.57 ±2.00	54.91 ±2.34	8.07 ±1.16	19.25 ±1.41	35.57 ±1.90
4	6.16 ±1.26	51.98 ±5.26	107.5 ±7.07	12.40 ±1.77	36.65 ±4.71	46.97 ±2.35
5	7.73 ±1.57	57.90 ±2.12	79.36 ±2.09	10.92 ±0.85	43.39 ±2.50	48.83 ±1.65
6	11.37 ±1.62	32.99 ±1.57	47.12 ±3.71	16.22 ±2.56	33.10 ±2.14	37.76 ±3.14
7	10.62 ±1.43	40.74 ±2.72	50.61 ±2.70	10.45 ±1.16	30.04 ±1.93	40.83 ±3.78
8	6.13 ±1.03	36.74 ±2.58	75.52 ±1.52	14.13 ±1.08	26.83 ±2.70	39.33 ±2.91
9	15.91 ±1.35	56.85 ±2.32	82.66 ±1.91	10.19 ±1.54	29.64 ±2.40	37.76 ±2.10
10	6.90 ±1.77	23.81 ±2.27	34.60 ±1.98	5.86 ±1.27	24.79 ±2.50	38.23 ±2.07
11	12.01 ±2.19	19.70 ±1.35	37.89 ±2.39	10.46 ±2.42	24.23 ±1.65	33.97 ±1.77
12	5.34 ±0.68	21.26 ±1.83	54.06 ±2.75	7.85 ±0.63	33.65 ±2.92	38.29 ±2.37
Mean	8.90	36.71	61.37	12.16	30.56	39.27
S.E.M.	1.09	3.89	6.22	1.29	1.84	1.29

* Mean $P_{ET}CO_2$ tensions recorded for the 12 subjects (*cf.*, Table 6.1)

Table 6.4: **Mean breathing frequencies in response to various levels of hypercapnia at 1 and 6 ATA of pressure while completing the performance test battery. Values are means ± S.E.M. (breaths/min).**

	1 ATA			6 ATA		
	End-tidal CO_2 tension (mmHg)					
	LOW 29.0*	MEDIUM 46.8*	HIGH 55.1*	LOW 29.1*	MEDIUM 47.9*	HIGH 58.6*
Subject Number						
1	22.2 ±0.70	31.4 ±0.82	33.7 ±0.56	22.1 ±2.26	26.3 ±1.35	27.44 ±0.48
2	15.9 ±0.35	22.5 ±1.15	17.3 ±0.55	15.1 ±0.58	19.5 ±0.96	17.6 ±0.73
3	14.0 ±1.57	16.3 ±0.53	23.4 ±0.55	11.9 ±0.52	14.1 ±0.48	19.9 ±0.84
4	12.1 ±0.67	24.2 ±2.28	29.3 ±1.91	8.63 ±0.45	14.1 ±0.68	15.1 ±0.43
5	22.7 ±0.84	27.6 ±1.20	32.7 ±0.91	13.4 ±0.63	24.5 ±0.92	24.8 ±0.46
6	15.5 ±0.51	17.4 ±0.82	18.6 ±1.20	11.3 ±1.41	18.0 ±1.01	17.7 ±1.12
7	14.5 ±0.79	26.6 ±0.84	23.6 ±0.43	13.1 ±0.98	16.7 ±0.63	21.9 ±0.51
8	15.6 ±0.74	25.5 ±1.87	22.6 ±0.76	13.1 ±0.48	15.0 ±0.87	19.2 ±0.94
9	16.7 ±0.76	26.6 ±0.95	34.6 ±1.81	11.8 ±0.58	20.7 ±1.50	27.1 ±1.45
10	9.3 ±0.68	21.7 ±1.22	18.9 ±1.73	6.1 ±0.48	14.1 ±1.09	24.6 ±1.04
11	18.3 ±0.78	14.9 ±0.51	18.8 ±0.78	13.2 ±0.63	16.7 ±0.41	19.9 ±0.50
12	21.8 ±1.54	20.4 ±0.99	26.0 ±0.69	16.6 ±0.54	22.6 ±0.68	20.8 ±0.40
Mean	16.5	22.9	25.0	13.0	18.5	21.3
S.E.M.	1.21	1.51	1.80	1.13	1.21	1.13

* Mean $P_{ET}CO_2$ tensions recorded for the 12 subjects (*cf.*, Table 6.1)

Table 6.5: **Mean tidal volume values in response to various levels of hypercapnia at 1 and 6 ATA of pressure while completing the performace test battery. Values are means ± S.E.M. (liters BTPS).**

	1 ATA			6 ATA		
	End-tidal CO_2 tension (mmHg)					
	LOW	MEDIUM	HIGH	LOW	MEDIUM	HIGH
	29.0*	46.8*	55.1*	29.1*	47.9*	58.6*
Subject Number						
1	0.28 ±0.06	1.36 ±0.07	2.07 ±0.04	1.14 ±0.37	1.32 ±0.05	1.41 ±0.07
2	0.80 ±0.11	1.43 ±0.11	2.58 ±0.10	1.39 ±0.15	1.65 ±0.08	2.09 ±0.15
3	0.42 ±0.08	1.52 ±0.10	2.37 ±0.19	0.74 ±0.18	1.40 ±0.12	1.83 ±0.09
4	0.56 ±0.14	2.28 ±0.15	3.68 ±0.22	1.51 ±0.22	2.45 ±0.25	3.08 ±0.17
5	0.35 ±0.07	2.04 ±0.03	2.43 ±0.03	0.83 ±0.11	1.77 ±0.06	1.85 ±0.06
6	0.74 ±0.10	1.98 ±0.08	2.56 ±0.17	1.21 ±0.08	1.90 ±0.12	2.25 ±0.12
7	0.80 ±0.08	1.57 ±0.11	2.17 ±0.11	0.79 ±0.02	1.80 ±0.07	1.87 ±0.14
8	0.37 ±0.06	1.48 ±0.10	3.30 ±0.11	1.11 ±0.14	1.86 ±0.07	2.11 ±0.13
9	0.95 ±0.07	2.12 ±0.04	2.49 ±0.11	0.84 ±0.10	1.39 ±0.06	1.40 ±0.06
10	0.78 ±0.19	1.06 ±0.10	1.76 ±0.12	0.97 ±0.06	1.69 ±0.10	1.67 ±0.12
11	0.64 ±0.12	1.34 ±0.07	2.05 ±0.12	0.94 ±0.23	1.48 ±0.10	1.69 ±0.12
12	0.31 ±0.01	1.05 ±0.07	2.08 ±0.13	0.49 ±0.04	1.50 ±0.11	1.86 ±0.09
Mean	0.58	1.60	2.46	1.00	1.68	1.93
S.E.M.	0.07	0.11	0.15	0.08	0.09	0.13

* Mean $P_{ET}CO_2$ tensions recorded for the 12 subjects (*cf.*, Table 6.1)

APPENDIX 5:

FIGURES FOR SUCCESSIVE PRACTICE TRIAL SCORES ON THE COGNITIVE AND PSYCHOMOTOR TESTS EMPLOYED IN THE TEST BATTERY.

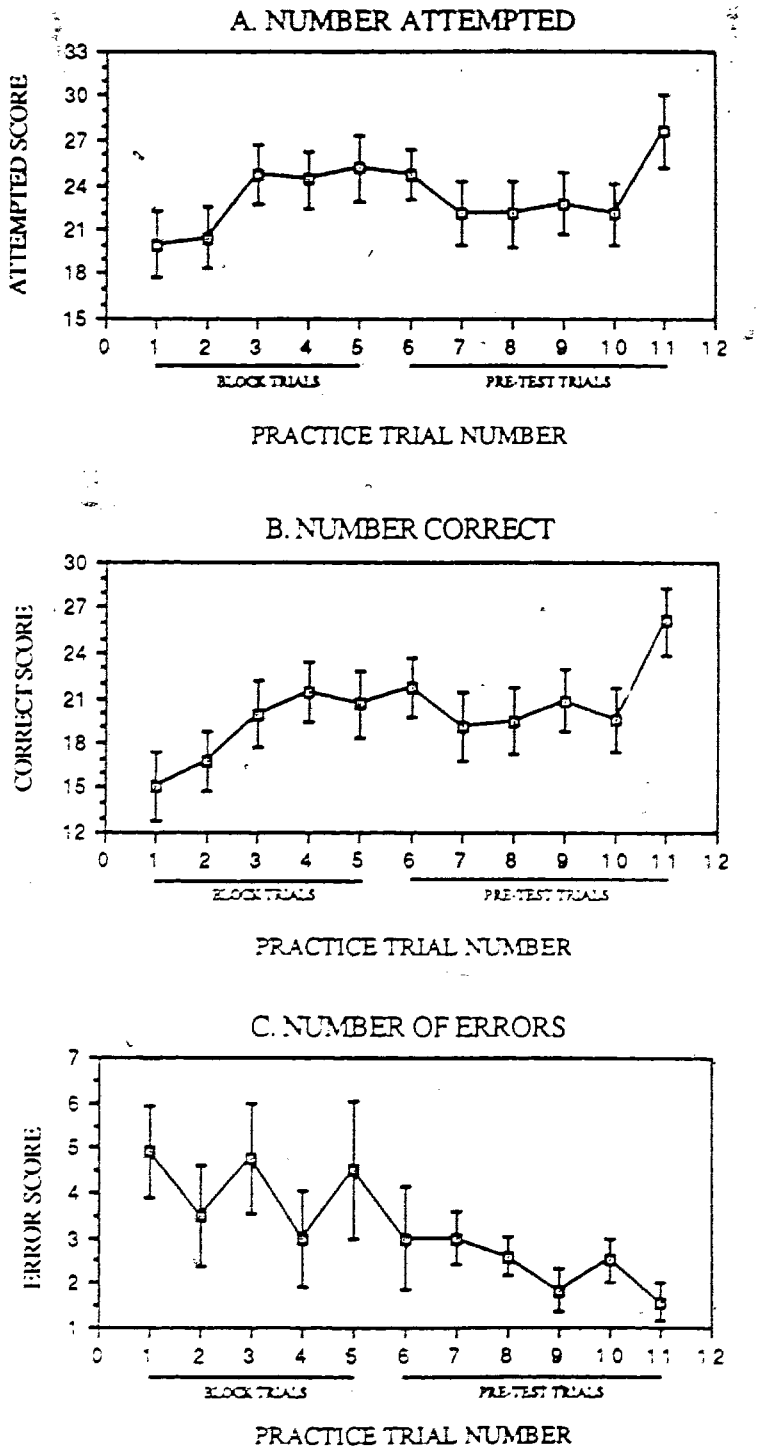


Figure 6.1 Mean scores for the number of problems correct (Fig. 6.1(A)), the number of problems attempted (Fig. 6.1(B)) and the number of errors (Fig. 6.1(C)) on successive practice tests of the Math task. Bars represent SEM.

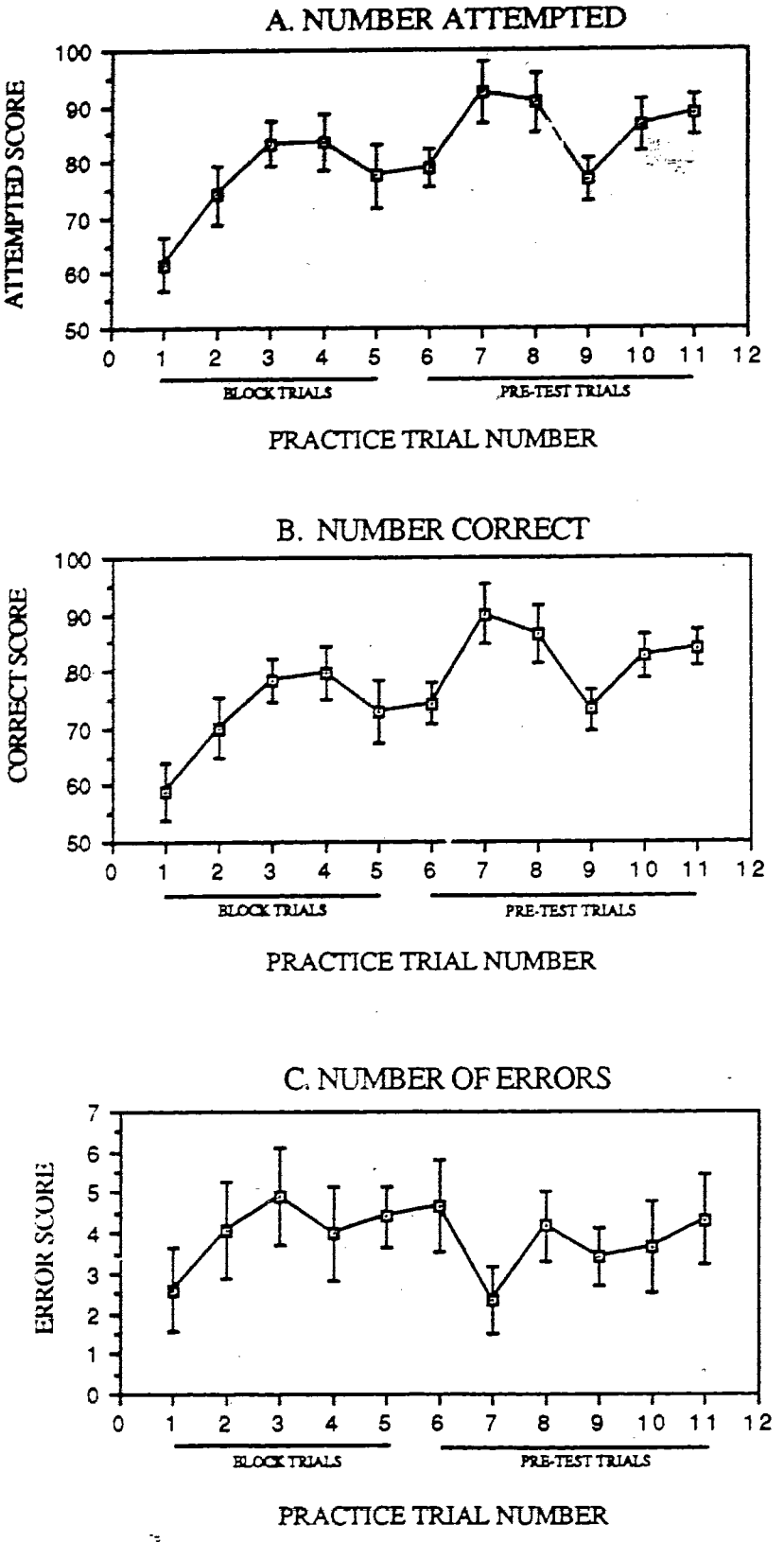


Figure 6.2 Mean scores for the number/portion of figures attempted (Fig. 6.2(A)), correct (Fig. 6.2(B)) and incorrect (Fig. 6.2(C)) on successive practice tests of the Copying task. Bars represent SEM.

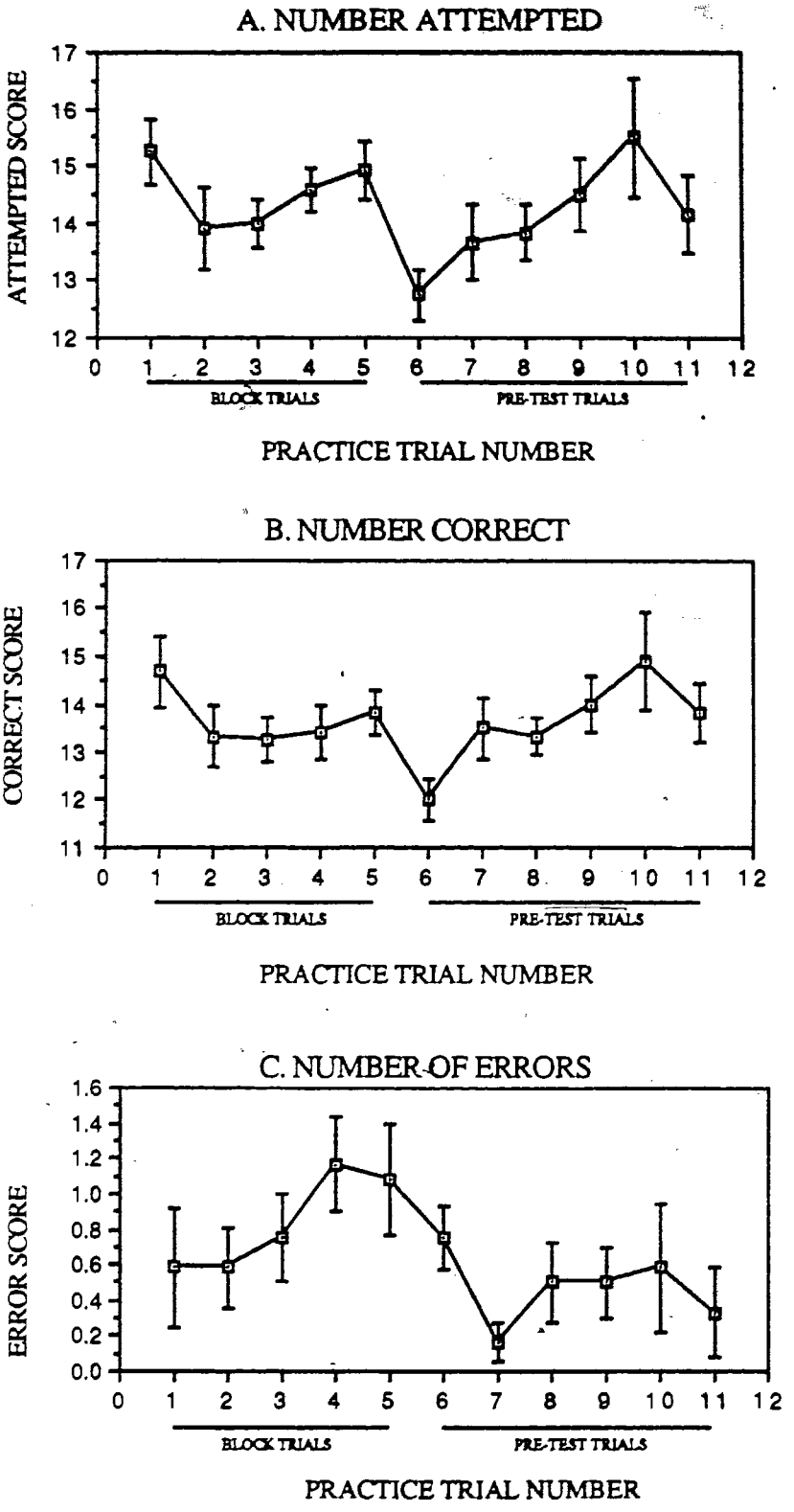


Figure 6.3 Mean scores for the number of digit sets attempted (Fig. 6.3(A)), correct (Fig. 6.3(B)) and incorrect (Fig. 6.3(C)) on successive practice tests of the Number comparison test. Bars represent SEM.

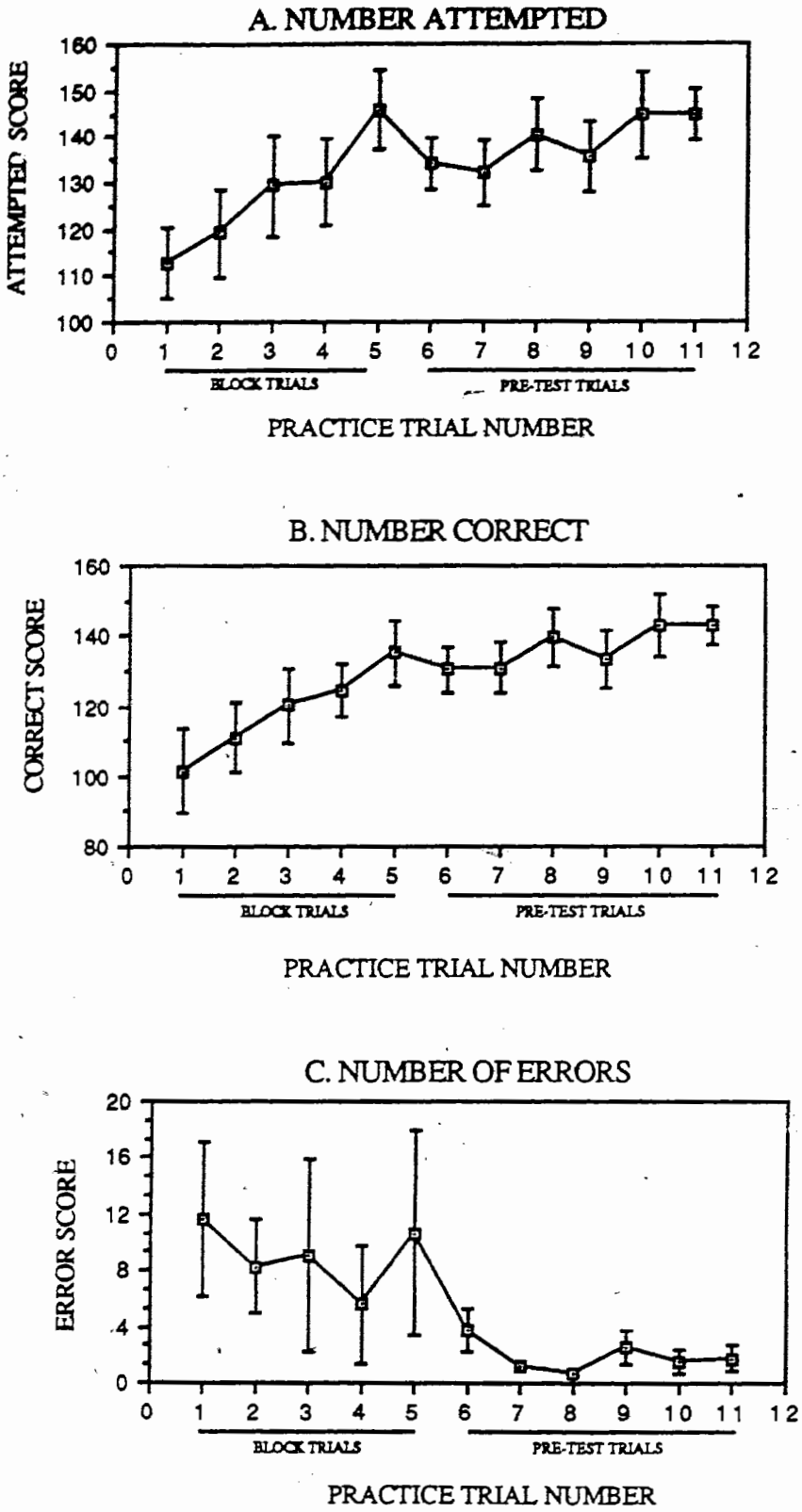
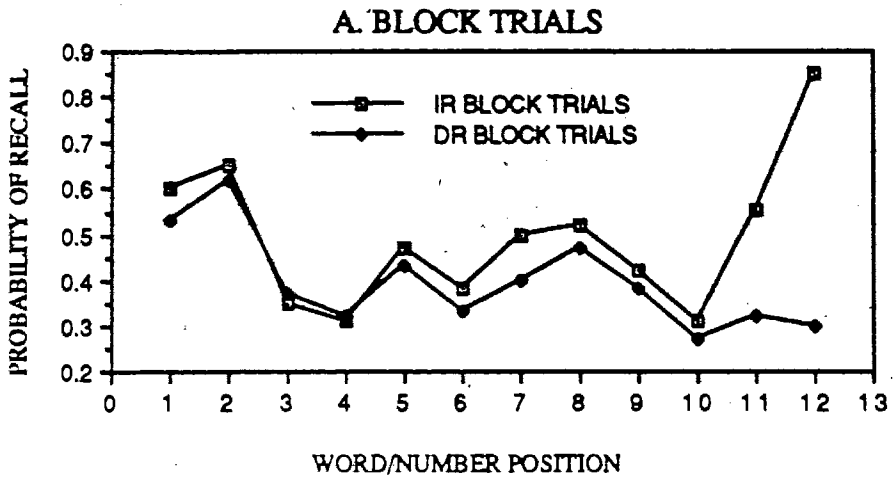
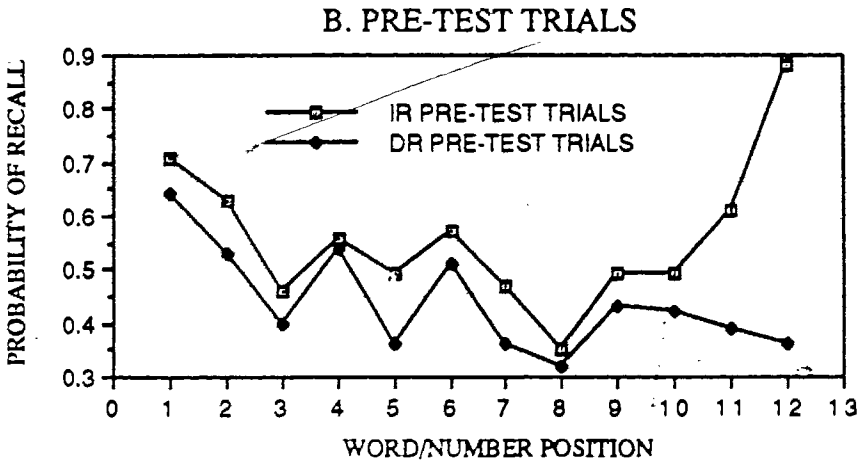


Figure 6.4 Mean scores for the number of letters attempted (Fig. 6.4(A)), correct (Fig. 6.4(B)) and incorrectly crossed out (Fig. 6.4(C)) on successive practice tests of the Letter cancellation task. Bars represent SEM.





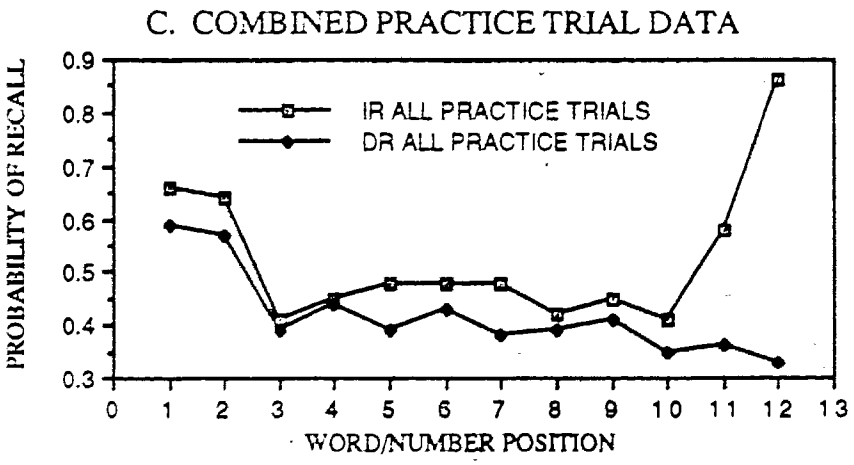


Figure 6.5 Probability of immediate and delayed recall vs serial position for combined successive block practice trials (A), pre-test trials (B) and total practice trials (C), on the paired association memory recall task.

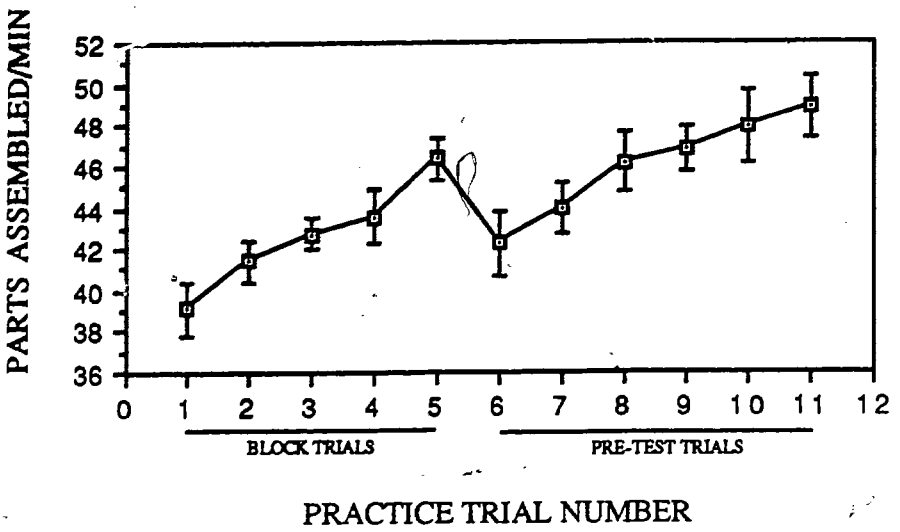


Figure 6.6 Mean scores for the number/portion of assemblies attempted over successive practice trials on the Purdue pegboard task. Bars represent SEM.

APPENDIX 6:

SUMMARY STATISTICS FOR PERFORMANCE SCORES FOLLOWING 2-WAY ANOVA ANALYSIS.

Table 6.6A: **Summary statistics for the main effect of CO_2 on the cognitive and psychomotor performance tests (data collapsed across N_2 levels).**

Performance Test	SSE	MSE	F	p	
Copying Test					
(No. Attempted)	1727	78.51	13.53	0.0003	*
(Error score)	149	6.78	0.87	0.4340	NS
(Correct score)	1807	82.14	14.28	0.0002	*
Math Test					
(No. Attempted)	122	5.53	26.08	0.0000	*
(Error score)	68	3.11	0.78	0.4741	NS
(Correct score)	213	9.66	20.31	0.0000	*
NComp Test					
(No. Attempted)	55	2.48	4.39	0.0243	*
(Error score)	10	0.46	1.30	0.2935	NS
(Correct score)	71	3.23	4.99	0.0160	*
Stroop Test					
(No. Attempted)	4838	219.89	14.14	0.0002	*
(Error score)	320	14.54	0.56	0.5826	NS
(Correct score)	5947	270.30	12.65	0.0004	*
IR					
(Total score)	100	4.54	4.87	0.0174	*
(Primacy)	24	1.08	0.73	0.4957	NS
(Recency)	8	0.39	1.55	0.2334	NS
DR					
(Total score)	110	5.00	3.36	0.0522	NS
(Primacy)	26	1.18	0.58	0.5749	NS
(Recency)	10	0.46	1.55	0.2326	NS
Purdue Pegboard					
(Prts assbld/min)	311	14.15	17.14	0.0001	*

df = 2, 22
n = 12
* $p<0.05$
Abbreviations: IR = Immediate recall, DR = Delayed recall, Ncomp = Number comparison test.
NS = Not significant.
SSE = Sum of Squares for the error term
MSE = Mean square of the error term (within-group variance estimate).

Table 6.6B: **Summary statistics for the CO_2–N_2 interaction on cognitive and psychomotor performance.**

Performance Test	SSE	MSE	F	p	
Copying Test					
(No. Attempted)	1819	82.69	0.06	0.9315	NS
(Error score)	161	7.31	0.29	0.7564	NS
(Correct score)	2051	93.24	0.11	0.8943	NS
Math Test					
(No. Attempted)	364	16.53	0.39	0.6839	NS
(Error score)	68	3.11	0.78	0.4741	NS
(Correct score)	447	20.33	0.21	0.8141	NS
NComp Test					
(No. Attempted)	37	1.70	0.29	0.7509	NS
(Error score)	11	0.51	3.99	0.0323	•
(Correct score)	50	2.29	0.19	0.8293	NS
Stroop Test					
(No. Attempted)	2649	120.41	0.82	0.4572	NS
(Error score)	424	19.27	0.53	0.6019	NS
(Correct score)	3505	159.34	0.87	0.4373	NS
IR					
(Total score)	98	4.48	0.69	0.5153	NS
(Primacy)	18	0.81	1.77	0.1928	NS
(Recency)	9	0.42	0.64	0.5436	NS
DR					
(Total score)	78	3.56	0.38	0.6940	NS
(Primacy)	26	1.18	0.58	0.5749	NS
(Recency)	8	0.37	1.04	0.3715	NS
Purdue Pegboard					
(Prts assbld/min)	286	13.01	0.08	0.9118	NS

df = 2, 22
n = 12
• $p<0.05$
Abbreviations: IR = Immediate recall, DR = Delayed recall, Ncomp = Number comparison test.
NS = Not significant.
SSE = Sum of Squares for the error term
MSE = Mean square of the error term (within-group variance estimate).

APPENDIX 7:

SUMMARY TABLES FOR POST HOC MULTIPLE COMPARISON TESTS.

Title: **Copying Test (Attempted Score)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	88.62	——	8.24*	13.16*
Medium CO_2	80.38		——	4.92
High CO_2	75.46			——

Tukey's critical range = 6.42
*= significant at the 0.05 level.

Title: **Copying Test (Correct Score)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	83.12	——	8.41*	13.87*
Medium CO_2	74.71		——	5.46
High CO_2	69.25			——

Tukey's critical range = 6.57
*= significant at the 0.05 level.

Title: **Math Test (Attempted Score)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	21.38	——	1.50	4.80•
Medium CO_2	19.88		——	3.30•
High CO_2	16.58			——

Tukey's critical range = 1.70
• = significant at the 0.05 level.

Title: **Math Test (Correct Score)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	19.21	——	2.17	5.67•
Medium CO_2	17.04		——	3.50•
High CO_2	13.54			——

Tukey's critical range = 2.25
• = significant at the 0.05 level.

Title: **Number Comparison Test (Attempted Score)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	14.08	——	0.50	1.33*
Medium CO_2	13.58		——	0.83
High CO_2	12.75			——

Tukey's critical range = 1.14
* = significant at the 0.05 level.

Title: **Number Comparison Test (Correct Score)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	13.42	——	0.63	1.63*
Medium CO_2	12.79		——	1.00
High CO_2	11.79			——

Tukey's critical range = 1.30
* = significant at the 0.05 level.

Title: **Stroop Test (Attempted Score)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	140.71	——	12.09*	22.75*
Medium CO_2	128.62		——	10.66
High CO_2	117.96			——

Tukey's critical range = 10.75
* = significant at the 0.05 level.

Title: **Stroop Test (Correct Score)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	138.75	——	12.17*	23.87*
Medium CO_2	126.58		——	11.71
High CO_2	114.88			——

Tukey's critical range = 11.91
* = significant at the 0.05 level.

Title: **Purdue Pegboard (parts assembled/min)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	46.29	——	1.91	6.21•
Medium CO_2	44.38		——	4.30•
High CO_2	40.08			——

Tukey's critical range = 2.73
• = significant at the 0.05 level.

Title: **Immediate Recall (Total Score)**

Level of Hypercapnia	Marginal Means	Low CO_2	Medium CO_2	High CO_2
Low CO_2	5.79	——	1.04	1.91•
Medium CO_2	4.75		——	0.87
High CO_2	3.88			——

Tukey's critical range = 1.54
• = significant at the 0.05 level.

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