THE EFFECTS OF EXERCISE ON THE SLEEP OF UNFIT INDIVIDUALS

SUSAN J. GRIFFIN (née Paxton)

Submitted in partial fulfilment of the requirements for the degree of Master of Psychology.

UNIVERSITY OF TASMANIA

1978.

THE EFFECTS OF EXERCISE ON THE SLEEP OF UNFIT INDIVIDUALS.

ACKNOWLEDGEMENTS

With deep gratitude I would like to acknowledge the invaluable guidance and support of Iain Montgomery, my supervisor, and Dr. John Trinder.

The collection of data in sleep research is laborious and exhausting for both those monitoring equipment and subjects alike. Thus, I sincerely thank Alison Bowling, Dot Bruck and again John Trinder and Iain Montgomery, for running nights in the laboratory, and all my subjects for sleeping and exercising there.

TABLE OF CONTENTS

LIST OF TABLES

A B S T R A C T

A facilitative effect of daytime exercise on SWS has been cited as evidence in support of the bodily restorative theory of sleep. However, this effect has not been observed in studies using unfit individuals. It was hypothesized that the failure of earlier studies to find a facilitative effect of exercise on SWS in unfit subjects was due to a stress induced by the exercise sessions which disrupted sleep and counteracted the facilitative effect.

Steps were taken to minimize stress by using afternoon exercise, a range of exercise levels and a number of exercise sessions for each subject. Thus the experiment consisted of a 4 x 4 factorial design with repeated measures on one factor (N = 24). The first variable consisted of 4 exercise levels (no exercise, 50% VO_{2max} for 45 mins., 75% VO_{2max} for 60 mins., and maximum output for 60 mins.) while the second variable represented 4 measurement occasions (baseline and three nights following 1, 3 and 5 consecutive exercise days). It was hypothesized that a facilitative effect of exercise would be observed under conditions in which the stress factor was minimized.

No effect of exercise on SWS was found in any condition, nor was there any evidence that a hypothetical stress factor accounted for the results. These findings are inconsistent with present statements of the bodily restorative theory of SWS. It is possible, however, that SWS levels may be responsive to long term rather than short term alterations in activity levels.

INTRODUCTION

The answer to the question "Why do people sleep?" has proven remarkably elusive, although it has dominated sleep research for several decades. It has become evident that the development of our understanding of the role of sleep requires a considerable extension of the descriptive and empirical data base in the area. However, research to develop a satisfactory data base in the area of sleep is most likely to be productive if it is theoretically based. Many theories of sleep have been proposed. A long standing, but nonetheless promising, theoretical notion is that sleep serves a restorative role.

The precise specification of restorative processes affected by sleep varies between theorists, though there is a general agreement by these theorists that during sleep there is a reduction in catabolic processes and an increase in anabolism. Most theorists attribute different restorative processes to various types or stages of sleep, which are identified on the basis of several physiological variables, most notably the electroencephalogram (EEG). As a consequence much of the data in support of a restorative theory evaluates the distribution of sleep stages following manipulations which would be expected to vary catabolism and thus vary the need for anabolic activity during the sleep period. In this context the effect of physical exercise on sleep is of considerable theoretical significance. At present, the results of these studies appear somewhat contradictory, but they do indicate a possible link between physical exercise and the distribution of sleep stages. Further clarification of this relationship is important to the evaluation of the restorative role of sleep. The

present experiment investigates one aspect of this relationship - the effects of different levels of physical exercise on the sleep of individuals unused to exercising.

CHAPTER 1.

THE NATURE OF SLEEP

In this chapter a general description of the nature of sleep is provided, including a consideration of the stages of sleep, its physiological concomitants, the phylogeny and ontogeny of sleep, and the effects of total and selective sleep deprivation.

EEG Sleep Stages

Sleep is generally classified into a number of stages on the basis of brain activity, muscle tonus and eye movements (Rechtschaffen and Kales, 1968). The major distinction is between Non Rapid Eye Movement (NREM) and Rapid Eye Movement (REM) sleep. (These two types of sleep have been given a variety of labels by different authors, although NREM and REM will be used throughout this thesis.)

NREM sleep is characterized by high amplitude, low frequency EEG activity. In primates this stage is further divided into four substages, identified as Stages 1 through 4. The major distinction is the increase in amplitude and decrease in frequency of the EEG. Thus Stage 1 is scored in the absence of EEG activity of an amplitude greater than 75 μv and a frequency of less than 2 Hz. Stage 2 contains up to 20%, Stage 3 20-50%, and Stage 4 greater than 50% EEG activity of greater than 75 μv and less than 2 Hz. Stages 3 and 4, when combined, are referred to as Slow Wave Sleep (SWS).

REM sleep is characterized by relatively low amplitude, mixed frequency EEG activity, episodic rapid eye movements and a relative low tonic EMG (Rechtschaffen and Kales, 1968).

NREM and REM periods recur cyclically through the night, sleep always commencing with a NREM period except in certain pathological conditions and in the newly born (Feinberg, 1974). In humans, the NREM-REM cycle generally recurs 4 or 5 times a night, each cycle lasting approximately 90 mins. (Dement and Kleitman, 1957; Feinberg, 1974). The internal structure of the sleep cycle changes across the night, the length of the NREM periods decreasing, and the REM periods increasing in the latter part of the night. There is also a decrease in SWS and an increase in minutes of Stage 2 in successive NREM periods (Feinberg, 1974). Thus a large proportion of SWS occurs early in a night's sleep and consequently SWS levels are not greatly affected by total sleep time (TST).

Two slightly different scoring systems for the EEG sleep stages are currently in use, both based on a system described by Dement and Kleitman (1957). The most widely used is a standardization of the original system proposed by Dement and Kleitman (1957) (Rechtschaffen and Kales, 1968). The second has been developed by two laboratories within the University of Florida (Williams, Karacan and Hursch, 1974). The systems differ in the minimum amplitude specifications for SWS activity, and the time constant used in recording. As a result direct comparison of SWS levels obtained using the different systems is difficult.

Physiological Concomitants of Sleep

Before sleep, while the body is resting, oxygen consumption, rectal temperature, heart rate and respiratory rate become lower. There is no abrupt discontinuity either at sleep onset or on waking (Snyder, 1971). There is a progressive decrease in rectal temperature

during the early hours of sleep and a rise towards morning (Snyder, 1971). This is also the case with oxygen consumption, however, superimposed on this curve, there are gross periodic variations which parallel changes in sleep stages (Brebbia and Altschuler, 1965). The highest rate of oxygen consumption occurs during REM sleep, the least in Stage 3 and 4, and intermediate levels during Stage 2 (Brebbia *et al.*, 1965). Heart rate and respiratory rate decrease through the night, however during REM sleep there are slight increases in average level (Snyder, Hobson, Morrison and Goldfrantz, 1964; Aserinsky and Kleitman, 1953) and marked increases in short term variability (Snyder *et al.*, 1964). The decrease in these functions appears to reflect a decrease in metabolic rate during sleep. During REM sleep there is also an increase in cerebral blood flow (Kety, 1967) and in brain temperature (Rechtschaffen, Cornwell and Zimmerman, 1965).

As mentioned, during REM sleep there are periodic bursts of rapid eye-movements and a complete loss of muscle tonus in the major muscle systems. During NREM sleep there is general muscle relaxation in humans and body mobility is low (Oswald, Berger, Jaramillo, Keddie, Olley and Plunkett, 1963), however, tonic muscle potentials are not entirely abolished (Berger, 1961).

Of particular interest, is the secretion of human growth hormone (HGH) during SWS. Peaks of HGH secretion appear to be dependent on the occurrence of SWS rather than merely occurring at the same time in the circadian cycle (Takahashi, Kipnis and Daughaday, 1968; Parker, Sassin, Mace, Gotlin and Rossman, 1969; Honda, Takahashi, Takahashi, Azumi, Irie, Sakuma, Tsushima and Shizume, 1969; Sassin, Parker, Johnson, Rossman, Mace and Gotlin, 1969a, 1969b). Three other hormones which regulate tissue development are dependent on sleep for their

release; prolactin, luteinizing hormone and testosterone (Boyar, Finkelstein, Roffwarg, Kapen, Weitzman and Hellman, 1972; Sassin, Frantz, Kapen and Weitzman, 1973).

The Phylogeny of Sleep

Prolonged periods of inactivity, organized on a circadian basis appear to be of very old phylogenetic origin being observed in reptiles, amphibia, fish and insects (Meddis, 1975). However, clear electrophysiological signs of sleep, i.e., high voltage, slow wave, electrical activity in the cortex, did not develop phylogenetically until the early mammals and birds (Allison and Van Twyver, 1970).

The most primitive animal, phylogenetically speaking, to show EEG signs of sleep, is the echidna, a representative of the nontherians. The nontherians have both distinctive mammalian features, maintaining constant body temperature and nursing their young, and distinctive reptilian features, the most striking being that the young are hatched from eggs. EEG studies of the echidna indicate that it sleeps about 12 hrs. a day, its sleep being entirely SWS. Thus, it appears that SWS was the earliest form of sleep to emerge, probably developing about 180 million years ago (Allison and Van Twyver, 1970).

One of the most primitive surviving therian mammals is the opossum which has both SWS and REM sleep, indicating that REM sleep developed in the early therian mammals about 130 million years ago. Both SWS and REM sleep have been retained in marsupial and placental mammals (Allison and Van Twyver, 1970).

There appears to be no really primitive form of bird surviving, so the development of sleep in birds cannot be traced. However, while the number of birds studied using electrophysiological techniques

is small, there are clear indications of SWS in all species, although REM sleep appears to exist in only a primitive and transitory form. Since the birds developed independently of the mammals it appears that sleep evolved separately in the mammals and birds (Allison and Van Twyner, 1970).

The Ontogeny of Sleep in Man

NREM and REM sleep develop in the human fetus by about the 34th week following conception, a large proportion of sleep being REM sleep. At birth, REM sleep has fallen to 50% of total sleep time (TST) (TST being approximately 18 hours)(Dreyfus-Brisac, 1964) and by 6 months and 1 year the REM sleep component makes up about 40% and 30% of TST resepctively (Hartmann, 1967). After the initial drop in REM levels early in life percentages of REM sleep stablize at about 25% of TST and remain at this level until old age when they again fall slightly (Williams *et al.*, 1974).

In neonates REM sleep precedes NREM sleep in the sleep cycle, however, by the age of 3 months NREM begins to precede REM sleep, as it normally does for the rest of life (Parmalee and Stern, 1972).

NREM levels as a percentage of TST remain constant after childhood, however the proportion of SWS appears to fall consistently as a function of age, Stage 4 dropping out before Stage 3 (Williams *et al.*, 1974). The major normative studies (Feinberg, 1974; Williams *et al.*, 1974) agree that there is a decrease in SWS with age, however there is some disagreement as to the rate of this change, Williams *et al.* (1974) finding a more rapid decline in SWS as a function of age than Feinberg (1974).

The Effects of Sleep Deprivation

Extended periods of sleep deprivation in dogs has been reported to result in death (Kleitman, 1963), however, this finding may be confounded by procedures used to keep the animals awake. In man even prolonged periods of sleep deprivation result in few consistent psychological and physiological changes (Kollar, Pasnau, Rubin, Naitoh, Slater and Kales, 1969; Kuhn, Meltzer, Wyatt and Snyder, 1970; Naitoh, Johnson and Lubin, 1971). The main psychological effect of sleep deprivation in man, apart from drowsiness, appears to be a lowering of performance on tasks requiring focused attention and vigilance (Hartmann, 1973; Hockey, 1970; Wilkinson, 1965). Performances on cognitive, perceptual and psychomotor tasks have also been observed to decrease (Pasnau, Naitoh, Stier and Kollar, 1968) and after five days of deprivation, disorientation, misperceptions and impairment of reality testing have been found to become more frequent (Pasnau *et al.*, 1968). However, it is important to note that during mild sleep deprivation (up to 48 hours) performance deficits are difficult to demonstrate, although, of course, subjects report being sleepy.

Following sleep deprivation, sleep time is extended and there is a marked increase in levels of SWS over baseline levels. Following extended periods of sleep deprivation, SWS remains elevated on the second recovery night although not to the same degree, while percentage of REM sleep is increased and REM latency decreased (Kales, Tan, Kollar, Naitoh, Preston and Malmstrom, 1970). Thus it appears that following sleep deprivation recovery of SWS takes priority over REM sleep recovery.

Selective deprivation of Stage 4 results in an increase in Stage 4 during the recovey night (Agnew, Webb and Williams, 1964).

However, extended periods of Stage 4 deprivation have been found to produce no clear cut physiological or behavioural changes although there is a suggestion of an increase in lethargy and depression over the period (Agnew, Webb and Williams, 1967).

Selective REM deprivation does not consistently result in a REM rebound on the recovery night (Cartwright and Monroe, 1968; Moses, Johnson, Naitoh and Lubin, 1975) and the observation of gross psychological changes as a result of REM deprivation reported by Dement (1960) has not been replicated in subsequent studies. There do appear to be minor psychological changes during REM deprivation, however, e.g., increased irritability (Greenberg, Pearlman, Fingar, Kantrowitz and Kawliche, 1970). Partial sleep deprivation, a procedure which primarily limits REM and Stage 2 time, has been shown to result in increases in Stage 4 (Dement and Greenberg, 1966) and Stage 4 and REM sleep (Webb and Agnew, 1965) on recovery nights.

CHAPTER 2.

THEORIES OF THE FUNCTION OF SLEEP

The following description of sleep theories classifies models into six categories.

1. Immobilization Theory
2. Energy Conservation Theory
3. Vigilance Function of REM Sleep
4. Central Nervous System Stimulation Theories
5. Information Processing Theories
6. Restorative Theories.

Before discussing each category several general points will be considered. Firstly, most recent theoretical approaches to sleep have focused on restricted aspects of the sleep process. In particular many models have proposed functions associated with either NREM or REM sleep, or alternatively, have ignored the distinction. As a consequence there are few comprehensive theories as to the functions of sleep. A second consideration is that many theories have been primarily based on a particular set of data. For example, the immobilization theory depends almost entirely on phylogenetic data for its support. A consequence of these first two points has been that many theories are compatible with each other and indeed in some instances are complementary. Finally, most theoretical approaches have been strongly influenced by evolutionary considerations. Thus, there has been a general acceptance of the view that sleep must have adaptive evolutionary significance. This approach has been influenced by the observation that sleep developed early phylogenetically, and

has been retained, and in fact intensified in most recently evolved species (Meddis, 1975).

Immobilization Theory

The main proponents of the immobilization theory are Meddis (1975) and Webb (1971, 1974). They propose that the prime function of sleep is to maintain immobility at times when immobility might be expected to improve an animal's chances of survival. All mammals have periodic immobility, or rest-activity cycles, organized on a circadian basis which it is assumed maximize activity levels during periods most favourable to the species. Thus food gathering is restricted to those phases of the circadian cycle when food is most available and when the species' physiological attributes are most effective. It is argued that high levels of activity at other times would both be less efficient and expose the organism unnecessarily to predation. The function of sleep is to ensure immobility and thus maintain the inactive periods.

The major evidence in support of the model is derived from phylogenetic data which indicates that there is both a positive correlation between sleep time and the security of the species' sleeping arrangements and a significant negative relationship between sleep time and the amount of time required for food gathering (Allison and Van Twyver, 1970).

The model maintains an interesting distinction between the function of sleep and the physiological mechanisms responsible for inducing it. (The mechanisms are assumed to be central nervous system structures located primarily in the brain stem.) There are two important implications of this distinction. Firstly, sleep is viewed

as instinctive, a concept supported by Tinbergen (1951) and Moruzzi (1969, 1972). Secondly, the deleterious effects of experimental manipulations of sleep, such as sleep deprivation, are considered to be due to the disruption of the mechanism rather than the function. As a result sleep disruption experiments are not viewed as highly significant evidence of function. Indeed, Meddis (1975), in particular, does not consider sleep to have any physiological function or unique physiological advantage other than that gained from periodic immobilization. As evidence for this view he points to species such as the shrew and Dall's Porpoise, presumably animals with much the same physiological requirements as other mammals, which do not appear to have clearly defined sleep.

This model in its most extreme form appears a plausible, if not testable, account of the appearance of sleep in early phylogenetic species. It becomes less convincing in explaining why sleep should have been retained by more recent species, particularly in predators such as carnivores. Carnivores have particularly high total sleep levels despite the fact that they appear to have little need for prolonged immobility (Zepelin and Rechtschaffen, 1974). It is possible that the adaptive implications of sleep have changed with phylogenetic development. Thus Webb (1974), in particular, is sympathetic to the view that sleep may have acquired other functions, such as energy conservation, which may not have been present in more primitive species.

One final difficulty with the immobilization hypothesis is its sterility with respect to experimental verification. It depends almost entirely upon phylogenetic considerations and predicts negative effects with respect to the particular importance of physiological

processes during sleep. As such it cannot be proven and presumably can only gain acceptance by default.

Energy Conservation Theory

The energy conservation model proposes that the main function of sleep is to lower metabolic requirements and thus conserve energy supplies (Allison and Ciccetti, 1976; Berger, 1975; Snyder, 1966; Zepelin and Rechtschaffen, 1974). This view has received considerable support from phylogenetic data though it is not entirely dependent upon such evidence. For example, Zepelin and Rechtschaffen (1974) have correlated a number of physiological and sleep variables over 53 species and found that metabolic rate estimated from body weight correlated .65 with TST and .73 with SWS. They concluded that the main function of sleep is to enforce a state of rest and so adaptively regulate energy expenditure. Thus those species whose waking activity is relatively expensive in terms of metabolic rate spend a greater proportion of the day asleep and have, as a consequence, less time to engage in metabolically expensive activity.

In support of the energy conservation model Berger (1975) points to the observation that the complete physiological and behavioural manifestations of sleep are unique to homeotherms and possibly evolved in parallel to homeothermy and the resulting need for energy conservation. The ontogenetic development of SWS and homeothermy also appear closely correlated (Berger, 1975).

A number of other studies relating sleep to metabolic rate support the energy conservation model.

1. In humans thyroid function (which influences metabolic rate) seems to influence the amount of SWS, as suggested by high levels of

SWS in cases of hyperthyroidism (Dunleavy, Oswald, Brown and Strong, 1974) and low levels in hypothyroid patients (Kales, Heuser, Jacobson, Kales, Hamley, Zweizig and Paulson, 1967).

2. An increase in SWS has been observed in adult humans after fasting (McFadyen, Oswald and Lewis, 1973; Karacan, Rosenbloom, Londono, Salis, Thornby and Williams, 1973) which is interpreted as reflecting a response to the need for energy conservation when food is limited.

3. In humans, there is a decrease in SWS (the stage during which energy conservation is at a minimum) with increasing age, and it is suggested that this occurs due to the parallel decrease in metabolic rate with age, and thus a decreased need for energy conservation (Berger, 1975).

Most of the theorists in favour of an energy conservation model of sleep have attributed this function primarily to NREM (as opposed to REM) sleep. Thus Allison and Ciccetti (1976) argue, on the basis of phylogenetic correlations, that energy conservation relates to NREM sleep while predator vs. prey and the security of sleeping arrangements is more closely associated with REM sleep. Similarly Zepelin and Rechtschaffen (1974) did not find a significant relationship between metabolic rate and REM sleep after NREM sleep had been partialed out. In contrast, Horne (1977) suggests that REM sleep may also serve an energy conservation function. He presents evidence to indicate that thermoregulation, particularly heat dissipating mechanisms, are suspended during REM sleep thus conserving energy.

It is generally recognized that there are several problems facing the energy conservation model of sleep.

1. Negative correlations between metabolic rate and sleep time have been found within several orders (Zepelin and Rechtschaffen, 1974).

2. Sleep quotas among some animals, e.g., the carnivores, are greater than would be predicted, while among others, e.g., small insectivores, they are lower than would be predicted by the conservation hypothesis (Zepelin and Rechtschaffen, 1974).

3. The occurrence of relaxed wakefulness in species with greater cortical development may require a modification of the energy conservation model of sleep as it suggests that energy conservation may be achieved in states other than sleep (Horne, 1977).

Vigilance Functions of Sleep

While Snyder (1966) suggested an energy conservation role for SWS, the role he proposes for REM sleep is that of a "sentinel". He notes that the major problem with sleep is that it renders animals helpless. (It is interesting to note that the immobilization model argues that sleep has virtually the opposite effect of making the animal safe.) The function of REM sleep is to combat this helplessness by periodically restoring responsiveness to the external world, and preparing the animal physiologically for possible defensive action. To add to the efficiency of this mechanism there is a brief period of arousal following REM in many species which serves a vigilance or "sentinel" function. It is further proposed that, in the case of animals under severe environmental threat, even this mechanism is insufficient. Thus REM sleep is replaced by longer and more frequent wake time (Snyder, 1966). This accounts for the finding that animals with insecure sleeping arrangements have low levels of REM sleep (Allison and Van Twyver, 1970).

Freemon (1970) also proposes an environmental surveillance model of sleep, but it applies both to REM and NREM sleep, and assumes other overriding functions of sleep. He proposes that the main function of sleep is CNS restoration and this restoration cannot take place in neurons at the same time as they are involved in analyzing ongoing environmental events. Yet some environmental surveillance is required continually, for the safety of the animal, even if it is at a low level. Thus during REM one set of neurons undergoes renewal while another provides a low level of environmental surveillance, while during NREM the roles of the groups of neurons are reversed.

Central Nervous System Stimulation Theories

One group of theories of REM sleep proposes that REM provides endogenous CNS stimulation during sleep either to establish and maintain neural pathways or to maintain an adaptive level of cortical activity (Roffwarg, Muzio and Dement, 1966; Berger, 1969; Ephron and Carrington, 1966). Roffwarg *et al.* (1966) hypothesize that a certain amount of afferent stimulation above that provided by external stimulation is necessary for the proper development of neural circuits in the mammalian cortex and this is supplied by REM sleep. The main evidence used to support this theory is the very high level of REM sleep found in the mammalian fetus and newborn. It is argued that since young mammals sleep a great deal they need more stimulation to the cortex than can be provided by external stimulation while awake. This situation is exaggerated in the womb when external sensory stimulation is minimal. Thus REM sleep levels are at their highest during these periods as most CNS stimulation must be attained through this source. As the animal grows older and remains awake longer, less endogenous stimulation is required, thus REM levels drop.

Berger (1969) proposes that the internal stimulation provided by REM sleep enables the establishment and maintenance of neuromuscular pathways serving binocular co-ordinated eye movements. In support of this hypothesis Berger (1969) cites the positive correlation between the percentage of total sleep time spent in REM and the amount of partial decussation at the optic chiasma in various species, particularly mammals. Another piece of evidence is the finding that performance on tasks requiring binocular co-ordination is higher immediately following a REM rather than a NREM period (Berger and Scott, 1971; Berger and Walker, 1972).

Finally, Ephron and Carrington (1966) suggest that during NREM sleep there is a progressive loss of cerebral vigilance and cortical deafferentation which must be maintained within adaptively appropriate limits. When a critical level of deafferentation is reached, REM is triggered and it supplies the necessary endogenous stimulation to raise cortical excitation, or cortical 'tonus' to a necessary level. Ephron and Carrington (1966) use the following evidence to support their theory.

1. Sleep is cyclic in nature, NREM preceding REM sleep.

2. During NREM sleep animals are relatively unresponsive to external stimuli.

3. During REM sleep most areas of the neocortex are at least as active as during the waking state.

The CNS stimulation models are not necessarily incompatible with each other. The various arousal functions of the REM state may represent both the different requirements of the organism at different stages in ontogenetic development and the various requirements of different subsystems. As such, these theories support

Hartmann's (1973) argument that it is probably inappropriate to be looking for *the* function of sleep as opposed to identifying its *functions*.

Information Processing Theories

Information processing theories of REM sleep propose that during REM new material, both factual and emotional, may be integrated into existing or new cognitive structures or programmes, and old information may be reprocessed and integrated into these structures or discarded. Thus, it is suggested that REM sleep is involved in memory, learning, and information and emotion organizing processes (Breger, 1967; Dewan, 1970; Greenberg, Pearlman, Fingar, Kantrowitz and Kawliche, 1970; Hawkins, 1966; Feinberg and Carlson, 1968; Feinberg and Evarts, 1969; Newman and Evans, 1965).

Computer analogue models have been used to describe the function of REM (Dewan, 1970; Newman and Evans, 1965). It has been proposed that the dream process might be likened in function to the systematic programme clearance which is necessary where computer programmes are being continually evolved to meet changing circumstances. The greater the change in circumstances being programmed for, the greater must be the amount of programme evolution and the more urgent the programme clear-out. REM sleep allows such a clearing process to get underway without interference from external information (Newman and Evans, 1965). Dewan (1970) proposes that during sleep and especially REM, reprogramming itself takes place.

Feinberg and Carlson (1968) and Feinberg and Evarts (1969) propose that REM sleep is involved specifically in cognitive processing such as learning and memory, while SWS provides the necessary

substrates for the processes occurring during REM. The main lines of evidence presented to support this theory are:

1. In normal older subjects the absolute amount of REM, and % REM of TST, are negatively correlated with age and positively correlated with scores on the Performance Scale of the WAIS (Feinberg, Koresco and Heller, 1967).

2. The total amount of REM sleep and eye movement activity is lower when intellectual functioning or ability is low (Feinberg, 1968).

3. SWS almost always precedes REM.

Other theories have emphasized the processing of emotional material during REM (Breger, 1967; Hawkins, 1966; Greenberg *et al.*, 1970). These theories emphasize the relationship between REM sleep and reports of dreaming. They suggest that during the day some events recall past emotionally arousing material to the preconscious, or are in themselves emotionally arousing. Dreaming serves to integrate affectively aroused material and unconscious impulses, into structures which have previously proved satisfactory in dealing with similar material. Breger (1967) proposes that dreams are uniquely adaptive insofar as they provide the conditions allowing for the integration of aroused material that is not so readily integrated during the waking state. These conditions consist of the greater availability of stored information, the greater fluidity of associational processes, the freedom from 'critical' processing for social acceptability, and a greater variety of means of manipulating symbols or processing and transforming stored information. The availability of these means would allow creative integration of material.

Restorative Theories of Sleep

One major problem for restorative theories has been the difficulty in specifying the precise nature of the hypothesized restorative processes occurring during sleep. These processes have typically been described in very general terms. Thus, it has been proposed that sleep is a particularly important time for anabolism and biosynthetic processes such as the maintenance and growth of cells. It has also been argued that the function of sleep is the replacement of a depleted substance or the depletion of a noxious substance (Feinberg, 1974; Hartmann, 1973; Oswald, 1974).

Restorative theories differ as to the proposed site of restoration during SWS and REM sleep. Feinberg (1974) maintains that both stages of sleep, but especially NREM, are involved in CNS restoration, while Oswald (1974) suggests that CNS repair is restricted to REM sleep and during SWS restoration takes place primarily in the periphery. Hartmann (1973) supports a CNS restorative role for REM and proposes that both CNS and peripheral repair take place during SWS.

Feinberg (1974) proposes that sleep serves to reverse a yet unknown neuronal-metabolic consequence of waking. He postulates two neuronal-metabolic states; State 1 is present at the completion of sleep but this state is reduced by waking to a depleted or otherwise altered state, State 2. During NREM sleep State 2 is converted to State 1. This process is most intense during SWS.

$$\text{State 1} \underset{\text{NREM}}{\overset{\text{Waking}}{\rightleftharpoons}} \text{State 2}$$

REM sleep functions to maximize the occurrence of NREM, and thus to maximize the process, State 2 $\xrightarrow{\text{NREM}}$ State 1, by producing

a cofactor or other substrate required for the transformation of State 2 to State 1. REM is supposed to produce a substrate similar to that produced by waking but to differ from waking in that State 1 is not depleted. It is hypothesized that perhaps the consumption of State 1 is associated with formation of new memory traces, a process which does not occur during sleep, despite periods of intense neuronal activity.

The first NREM period makes a substantial conversion of State 2 to State 1 but does not achieve optimal levels of the latter. REM sleep then occurs producing a cofactor or substrate which permits further NREM, again raising the level of State 1. This process is repeated until optional levels of State 1 are achieved. Completion of the transformation is indicated by NREM consisting entirely of Stage 2 and by a lessened duration of the later NREM periods.

This model is consistent with a number of observations.

1. NREM and REM periods occur cyclicly throughout sleep (Feinberg, 1974).

2. Greater amounts of NREM sleep, and especially SWS, occur early in the night (Feinberg, 1974).

3. Following total and partial sleep deprivation there are greater increases in SWS than REM sleep (Webb and Agnew, 1965).

4. Late afternoon naps contain a large SWS component (Karacan, Williams, Finley and Hursch, 1970).

5. Finally, Feinberg (1974) argues that both CNS activity, such as information acquisition and processing, and SWS, decrease with increasing age.

Feinberg's (1974) model which argues for a relationship between SWS and CNS restorative processes, while denying any similar

role for sleep in peripheral restoration, faces two major contrary findings. The first is a positive correlation between REM, not SWS, and level of cognitive functioning in aged normal and senile subjects reported by Feinberg (Feinberg *et al.*, 1967). The second is that manipulations of a number of factors which most certainly effect peripheral functions, have been shown to influence SWS. These findings will be discussed later in this chapter, the most relevant to the present study being the possible effect of physical exercise on SWS. These effects would not be predicted by Feinberg's model but rather lend support for a peripheral restorative function for SWS as proposed by Oswald (1974) and Hartmann (1973).

The restorative theories of sleep proposed by Oswald (1974) and Hartmann (1973) have stressed the bodily restorative role of SWS. Oswald (1974) suggests that SWS is important for the growth and repair of bodily tissues while the main function of REM sleep is the restoration of the brain. Similarly, Hartmann (1973) proposes that SWS can be considered an anabolic phase of sleep in which macromolecules, such as proteins and RNA, are synthesized. Hartmann (1973) also emphasizes the role of REM sleep in the restoration and formation of connexions in the cortex involved in learning, focused attention and emotional functions.

The bodily restorative theory of SWS predicts increases in SWS following increased catabolism and heavy demands on body tissues, and decreases in SWS when catabolism is reduced. In addition, SWS should provide an optimal environment for anabolic processes. On the basis of these predictions the following evidence has been cited in support of the bodily restorative theory of SWS (Adam and Oswald, 1977; Hartmann, 1973; Oswald, 1974).

1. Higher levels of SWS have been found following daytime exercise, and presumably higher levels of catabolism, than following no daytime exercise, in athletes (Baekeland, and Lasky, 1966) in cats (Hobson, 1968) and rats (Matsumoto, Nishisho, Suto, Sadahiro and Miyoshi, 1968).

2. Nocturnal peaks of human growth hormone (HGH) secretion depend upon the presence of SWS (Sassin *et al.*, 1969ab; Schure, Raskin and Lipman, 1971; Takahashi *et al.*, 1968; Honda *et al.*, 1969). Since HGH is an anabolic hormone, its secretion during SWS would ensure a suitable environment for restorative processes. Growth hormone stimulates amino acid uptake into tissues, promotes protein and RNA synthesis (Korner, 1965) and has wide interactions such as stimulating red blood cell formation indirectly through erythropoietin (Peschle, Rappaport, Sasso, Gordon and Condorelli, 1972). It also raises blood free fatty acid levels, whose subsequent degradation is a source of cellular energy, thereby saving amino acids from catabolism and increasing their availability for protein synthesis during sleep (Adam and Oswald, 1977).

3. Corticosteroids, hormones which stimulate catabolism (Friedman and Strang, 1966), are lowest at the time when HGH secretion is highest. Consequently there is even greater net protein synthesis during human sleep.

4. Three other anabolic human hormones are sleep dependent; prolactin (Sassin *et al.*, 1973), luteinizing hormone and testosterone (Rubin, Poland and Tower, 1976; Boyar, Finkelstein, Roffwarg, Kaper, Weitzman and Hellman, 1972).

5. Daytime exercise, as opposed to no daytime exercise, results

in increased levels of sleep associated HGH and decreased levels of corticosteroids (Adamson, Hunter, Ogunremi, Oswald and Percy-Robb, 1974). These hormonal changes would appear to provide ideal circumstances for the enhanced reparative processes required following increased catabolism.

6. High SWS levels and sleep associated HGH secretion are found in hyperthyroid patients (Dunleavy, Oswald, Brown and Strong, 1974) in whom catabolism takes place at high levels. Thus the need for anabolic processes would be correspondingly high. Conversely, low levels of SWS are found in hypothyroid patients (Kales *et al.*, 1967) in whom catabolism takes place at a low level. After days when normal men have had higher thyroxine secretion they also get more SWS (Johns, Masterton, Paddle-Ledinek, Patel, Winikoff, Malinek, 1975).

7. Acute starvation increases both SWS (McFadyen *et al.*, 1973) and HGH secretion (Parker, Rossman and Vanderlaan, 1972; Karacan *et al.*, 1973) at a time when protein-sparing processes would be especially important. Weight loss in patients using femfluramine (an amphetamine derivative) is also associated with increased SWS (Lewis, Oswald and Dunleavy, 1971).

In contrast to the effects of acute starvation, chronic starvation results in a decrease in SWS levels in humans (Crisp and Stonehill, 1976). It is unclear as to why the duration of the deprivation period should have this effect, though it is noteworthy that during refeeding following long term deprivation periods SWS levels increase over the deprivation period (Lacey, Crisp, Kalucy, Hartmann and Chen, 1975). This increase is possibly related to tissue rebuilding.

8. Longer periods awake produce both increased levels of SWS and sleep associated HGH (Berger and Oswald, 1962; Williams, Hammack, Daly, Dement and Lubin, 1964; Jacoby, Smith, Sassin, Greenstein and Weitzman, 1975; Karacan, Rosenbloom, Londono, Williams and Salis, 1975; Beck, Březinová, Hunter and Oswald, 1974).

9. Selective deprivation of SWS produces in subjects feelings of physical discomfort (Agnew, Webb and Williams, 1967).

10. Periods of rapid physical growth are accompanied by high levels of SWS and HGH, while both fall in old age when growth and regeneration of cells decreases (Hartmann, 1967; Williams *et al.*, 1974).

11. There is a relationship between basal metabolic rate and sleep duration. Across species total sleep duration and NREM sleep time are positively correlated with basal metabolic rate (Zepelin and Rechtschaffen, 1974). In man, individual differences in sleep duration correlate positively with waking body temperature and thus possibly with waking metabolic rate (Taub and Berger, 1976). It has been suggested that the higher day time metabolism produces greater levels of degradation requiring higher levels of compensatory synthesis and thus longer sleep (Adam and Oswald, 1977).

12. Finally, Adam and Oswald (1977) argue that an optimal environment for protein synthesis is created during SWS by the lowered energy requirements of other physiological systems. Synthetic processes, like protein building, require energy. When the general demands for energy are high the energy charge (EC)[1] of cells falls and biosynthesis

---

1. Atkinson (1968) has quantified the energy level of cells in terms of the relative levels of adenosine triphosphate (ATP), adenosine diphosphate (ADP) and adenosine monophosphate (AMP). The resulting value, which has a range of 0 to 1, is referred to as the energy charge (EC) of the cell. Thus

$$EC = \frac{ATP + ADP/2}{ATP + ADP + AMP}.$$

is inhibited. In contrast, when energy supplies within cells are high biosynthesis is stimulated. During SWS, when overall energy requirements are particularly low, as indicated by decreased metabolic rate, EC is maintained at a high level and thus increased energy is diverted to synthetic cellular processes, which then occur at a greater rate.

Oswald (1974) and Hartmann (1973) propose that REM sleep is involved in CNS restoration, particularly in the form of protein synthesis in the brain. These proteins may be used to restore, reorganize or form connections in the cortex required for focused attention and possibly learning and memory associated with focused attention. REM sleep may also provide recuperation for the catecholamine-dependent neuronal systems involved in learning, memory, attention, emotional integrity and social adaption, during the day. This aspect of Hartmann's (1973) and Oswald's (1974) restorative models is similar in many ways to the information processing theories described above. Evidence presented in favour of this view includes the following points.

1. REM sleep levels are elevated for up to 2 months following drug overdoses or periods of amphetamine administration. This time period corresponds with the length of time required to replace neuronal proteins and it is argued that REM sleep is enhanced during the recovery period to provide optimum conditions for the extra protein replacement required following such an insult to the brain (Haider and Oswald, 1970; Oswald, 1970).

2. During REM sleep blood flow in the brain rises above waking levels (Kety, 1967) and there is an accompanying rise in brain temperature (Rechtschaffen, Cornwell and Zimmerman, 1965). This may reflect increased protein synthesis.

3. Just before and after birth, when rates of brain development are high, levels of REM sleep are likewise high (Hartmann, 1967). In senility, when brain renewal becomes less, there is an accompanying reduction in REM sleep time (Feinberg *et al.*, 1967).

4. Mentally retarded infants have less REM sleep than normal infants (Oswald, 1974) and in older people there is a positive correlation between amounts of REM sleep and performance scores (Feinberg *et al.*, 1967).

5. Performance on tasks requiring focused attention deteriorates with sleep deprivation (Wilkinson, 1965). Irritability, poor social presence and lowered self-confidence are often observed following REM deprivation (Hartmann, 1973; Greenberg *et al.*, 1970).

6. Within individuals more REM sleep occurs in times of stress and change in environment. Also studies of short and long sleepers suggest that long sleepers, who presumably have more REM sleep, have more stressful lifestyles (Hartmann, Baekeland and Zwilling, 1972; Hartmann, Baekeland, Zwilling and Hay, 1971).

7. When catecholamine levels are low, high levels of REM sleep are observed, however, when they are high, low levels of REM sleep occur (Hartmann, 1973).

While the weight of evidence currently available appears to suggest that sleep has a restorative function a number of the experimental results listed by both Oswald (Oswald, 1974; Adam and Oswald, 1977) and Hartmann (1973) have not been replicated. Possibly the most controversial finding has been the relationship between physical exercise and SWS. This relationship is an important component of the evidence in support of the bodily restorative role of SWS as it is

the manipulation which most clearly has peripheral, as opposed to CNS effects. As such it is important evidence for Oswald's and Hartmann's theories and is possibly crucial evidence in evaluating these models as opposed to Feinberg's. The literature on the effects of physical exercise on sleep is reviewed in Chapter 4.

CHAPTER 3.

PHYSIOLOGICAL EFFECTS OF EXERCISE

While some restorative theories predict that physical exercise will have consequences for sleep, the mechanisms by which this effect is brought about have not been elucidated. However, the effects of exercise on a wide range of other physiological systems have been extensively studied. The purpose of this chapter is not to speculate on the nature of the mechanisms relating sleep to exercise, but rather to provide background information as to the general physiological effects of exercise.

Immediate Responses to Exercise

As soon as a person starts exercising, changes take place in his body, primarily to enable him to meet the increased energy requirements of the active muscle cells. The only direct source of energy within the cell is adenosine triphosphate (ATP) which releases energy when it splits into adenosine diphosphate (ADP) and phosphoric acid (Pi) (Margaria, 1976). However, there is insufficient ATP stored within a cell to support contraction for more than a fraction of a second. Consequently ATP must constantly be resynthesized in the cell if contraction is to continue (Karpovich and Sinning, 1971).

The most immediate process is the cleavage of creatine phosphate (CP) into creatine (C) and Pi. These two reactions are in series and thus the term phosphagen is used to refer to the energy supplied by ATP and its continual resynthesis by CP. However, as the supply of CP in muscles is only about three times as great as

ATP, the phosphagen process is not sufficient to provide for prolonged activity. If muscular activity is to continue energy must be supplied in the system so that ATP can be continually resynthesized.

The main fuel source for the resynthesis of ATP is glucose. When exercise begins glucose is released from the liver into the blood to be carried to active muscle cells where it is broken down, releasing energy for ATP synthesis. There are two ways in which the glucose can be broken down, one requiring oxygen (O_2) - aerobic metabolism, and one which does not require O_2 - anaerobic metabolism. In aerobic metabolism glucose is converted to pyruvic acid. During this process two molecules of ATP are formed from the metabolism of each molecule of glucose. The pyruvic acid then enters the mitochondria where it undergoes oxidation to carbon dioxide (CO_2) and water (H_2O) with the formation of 36 molecules of ATP per molecule of glucose metabolized. Fats and protein can likewise be oxidized in the mitochondria with the production of ATP (Karpovich and Sinning, 1971).

Under submaximal exercise conditions the amount of O_2 will be sufficient to maintain the phosphagen of muscles in equilibrium. However, when the level of activity is such that the O_2 supply is inadequate to provide all the energy needed by the cell from aerobic metabolism, the balance of the energy required is derived from anaerobic metabolism. In this case the pyruvic acid formed by glycolysis is broken down with the formation of lactic acid, and two molecules of ATP per molecule of glucose. The formation of lactic acid enables H_2 to be removed from the energy releasing cycle, which is necessary for the cycle to continue (Åstrand and Rodahl, 1970). There are a number of disadvantages of anaerobic metabolism:

1. The yield of ATP is low.

2. Only carbohydrate, which is in limited supply, can be used as fuel.

3. Lactic acid is formed in the process. Since it is only slowly resynthesized to glycogen it accumulates within the cells and limits their capacity for contraction.

These factors limit the time cells can rely on anaerobic metabolism to about 30 secs. depending upon the actual level of exercise. Despite these drawbacks, it is a very important process since it is used to provide energy at the beginning of exercise before the supply of O_2 has been increased and also when O_2 is insufficient to provide all the energy required for exercise (Morehouse and Miller, 1971).

As mentioned, for sustained exercise the O_2 supply to the active cells must be increased to enable increased aerobic metabolism. This is accomplished by an increased respiratory rate, and, as O_2 is transported via the blood, there is an increase in heart rate and stroke volume and consequent blood flow through the body. Systolic blood pressure is increased and blood flow redistributed so that an adequate amount of blood reaches active areas (Brouha and Radford, 1960). These physiological changes also allow waste products, primarily CO_2, heat and to some extent lactic acid, to be removed from the cells. CO_2 is breathed out while heat is dissipated from capillaries near the surface of the skin. Lactic acid stays in the blood and when exercise finishes it is broken down in the liver (Åstrand and Rodahl, 1970).

Each individual has a maximum oxygen intake (VO_{2max}) or aerobic power, which is limited by such factors as maximum HR and maximum speed of O_2 uptake mechanisms at a cellular level (Kreuzer, 1964). If a person exercises at a submaximal level O_2 supply to

cells can meet O_2 demands and a 'steady state' is said to exist (Åstrand and Rodahl, 1970). Submaximal work can continue until terminated by some other factor like exhaustion of glycogen stores (Morehouse and Miller, 1971).

Metabolites in the blood, like lactate, pyruvate, glycerol, free-fatty-acids (FFA) and ketone bodies increase during exercise (Johnson, Walton, Krebbs and Williamson, 1974), while some hormones, such as thyroxine, decrease (Irvine, 1974).

During severe exercise changes may take place in muscle cells. Fat droplets are deposited in the cells, probably due to the increased fat utilization in prolonged exercise, and focal areas of inflammation and necrosis of skeletal muscle cells develop. Other muscle changes observed after exercise include fragmentation and atrophy of scattered muscle fibres, indistinct striation, marked proliferation of muscle nuclei, formation of multinucleated giant cells and infiltration by various types of inflammatory cells. Exhaustive exercise also causes an increase of granulae in myocardial sacroplasma and mitochondria of muscle cells. It seems likely that histological and histochemical changes also take place in CNS cells. Moderate exercise probably causes milder cellular changes of this kind (Simonson, 1971).

Recovery from Effects of Exercise

The speed of recovery from exercise naturally depends on the degree of severity of the work performed. However, different changes due to exercise require different time periods to be reversed. Indirect evidence suggests that ATP, ADP and AMP ratios (the energy charge of a cell (Atkinson, 1968)), remains low for longer than

6 hours after strenuous exercise (Adam and Oswald, 1977). Basal metabolic rate is increased the day after strenuous exercise and may remain high up to 72 hours following severe exercise, probably as the result of some metabolic stimulation produced by sympathetic nervous system arousal (Simonson, 1971).

The term of 'oxygen debt' is used to describe the amount of O_2 required in the post exercise recovery period, above basal levels, to restore the body to its pre-exercise condition (Åstrand and Rodahl, 1970). A number of factors contribute to the delayed return of O_2 uptake levels following exercise (the payment of the O_2 debt). O_2 is required

1. to refill the O_2 content of the body,
2. to support an elevated metabolic rate due to an increase in tissue temperature and a possible increased output of adrenalin,
3. to supply increased oxygen demand to activated respiratory muscles,
4. to remove anaerobic metabolites.

The former three factors contribute to an 'alactacid' oxygen debt, while the last contributes to a 'lactacid' oxygen debt (Åstrand and Rodahl, 1970). The payment of the debt follows a logarithmic form (Simonson, 1971). The alactacid portion takes only about three minutes to pay while the lactacid debt may take up to an hour and a half depending on the severity of the exercise (Karpovich and Sinning, 1971).

As O_2 demand increases, respiratory rate, HR and circulation rate decrease. After moderate work these functions return to basal levels following several minutes while after severe exercise this process takes longer and in fact these functions may remain slightly elevated

above basal levels for many hours due to increased sympathetic nervous system activation. The restoration of the normal phase-coupling between HR and respiration is much slower than the recovery of either of these functions separately (Simonson, 1971).

Blood lactate, pyruvate and glycerol levels return to pre-exercise levels within three hours after terminating exercise, while FFA and blood-ketone-body levels may remain elevated for longer than this (Johnson *et al.*, 1974).

The reversible histological changes in muscle, heart and CNS after work may require a period of up to several days to recover, depending on the severity of change (Simonson, 1971).

In summary, most of the effects of moderate exercise are reversed within a few hours, however if work is severe recovery processes take longer, metabolic rate being elevated for up to three days and histological recovery also requiring several days to complete.

Responses to Exercise in Fit and Unfit Individuals

There are marked differences in the responses to, and recovery from exercise between fit and unfit individuals. At the cellular level, the level of enzymes essential for aerobic reactions and the size and number of mitrochrondria increase with increasing fitness. This enables ATP, and therefore energy, to be supplied in working cells at a greater rate (Karpovich and Sinning, 1971; Thomas 1975).

Oxygen transport systems also become more efficient with training; depth of breathing, coronary capacity and muscle tissue capilliarization being increased (Karpovich and Sinning, 1971; Thomas, 1975). These factors contribute to an increased VO_{2max} in fit people which is reflected in lower HRs on the same exercise task than that

of unfit people and a greater capacity for work at VO_{2max}. For this reason work capacity at a specific HR and derived estimates of VO_{2max} are used as indications of degree of fitness (Åstrand and Rodahl, 1970).

In unfit people the O_2 debt after exercise is repaid more slowly than in the fit. Recovery of basal HR, blood pressure, and basal metabolic rate is also slower in the unfit (Kreuzer, 1964).

Removal of wastes is less efficient in unfit people after exercise. In particular, lactate is disposed of more slowly, probably as a result of slower oxidation in the muscle or in the liver (Johnson *et al.*, 1974).

Training causes hypertrophy of latent muscle cells and an increase in the number of myofybrils in skeletal and respiratory muscles thus producing an increase in muscle strength and endurance (Karpovich and Sinning, 1971). Training also decreases the likelihood of muscle damage, lesions or inflammation as a consequence of exercise (Simonson, 1971).

In conclusion, it is evident that, by comparison with fit individuals, untrained individuals can do less work at a particular HR. This is primarily due to the facts that their ATP resynthesis, oxygen uptake and waste removal systems are less efficient, and their muscles not as strong. Not only can they do less work but their recovery rates for most functions affected by exercise are slower.

CHAPTER 4.

EFFECTS OF EXERCISE ON SLEEP

The effects of exercise on sleep have been cited in support of Oswald's (1974) and Hartmann's (1973) version of the restorative theory of SWS. The predicted relationship is an increase in SWS levels following exercise, due to the increased catabolism in muscle tissue and the consequent increased need for anabolism. However, a complete review of the literature indicates that many reports are contradictory and in some instances results appear to be inconsistent with the model. To some extent the contradictory nature of the literature is due to a failure to recognize the existence of two separate effects of exercise (Griffin and Trinder, 1978). One is the effect of habitual exercise (physical fitness), and the other is due to the immediate effects of a particular exercise session. The former appears to result in a sustained increase in SWS levels while the latter may produce a transitory increase on the immediately succeeding night. The following review of this literature evaluates the evidence for each of these effects.

Physical Fitness and Sustained[1] SWS Levels

Three studies have evaluated the effects of habitual exercise on human sleep, independently of immediately preceding exercise. All three found higher levels of SWS in fit subjects though only one

1. The term "sustained increase in SWS" is used to indicate an increase in SWS on nights following days in which no specific exercise is administered. As such it is assumed to represent an effect of physical fitness or habitual exercise, rather than the immediate effects of exercise itself (though see point 2, page 38).

(Griffin and Trinder, 1978) included a non-fit control group, while the other two (Baekeland and Lasky, 1966; Zloty, Birdick and Adamson, 1973) compared their data with other published studies. Two studies have compared the relationship between chronically high activity levels and sleep in animals, one of which found positive effects on SWS levels (Reite, Stynes, Vaughn, Pauley and Short, 1976), while the other (Webb and Friedman, 1969) found no effect on total sleep time (TST).

A comparison of SWS levels of fit and unfit subjects by Griffin and Trinder (1978) indicated that fit subjects had significantly more SWS, due to increased Stage 3, than unfit subjects, independent of a particular day's exercise. The mean levels of SWS for fit and unfit subjects were 119.7 and 94.0 mins., respectively.

Baekeland and Lasky (1966) reported that, following a day of no exercise, the mean SWS level of trained athletes (117 mins.) was considerably higher than that (87.6 mins.) obtained by Williams, Agnew and Webb (1964) with their largely sedentary and nonathletic subjects.

Zloty *et al.* (1973) reported a mean SWS level of 104.7 mins. (23% of sleep period time (SPT)) in long distance runners. This level was compared with a mean control night level of SWS of 13.5% SPT found by Scott (1972) and a mean SWS level of regular exercisers following daytime exercise of 59.5 mins. reported by Baekeland (1970). Zloty *et al.* (1973) concluded that distance runners have significantly more SWS than other regular exercisers or non exercisers.

A significant positive correlation between amount of locomotor behaviour and SWS in infant monkeys was reported by Reite *et al.* (1976). Finally, Webb and Friedmann (1969) compared the sleep of habitually exercised and sedentary rats and found no difference in total sleep time or diurnal distribution of sleep, however NREM and REM levels were

not reported.

The failure of Baekeland and Lasky (1966) and Zloty *et al.* (1973) to run the appropriate unfit control groups limits the usefulness of their data. However, the general trend of the studies is consistent with the hypothesis that habitual exercise produces a sustained increase in SWS. Griffin and Trinder (1978) suggest four possible explanations for this finding.

1. Physical fitness could be correlated with SWS levels because of a physiological adaptation to the habitual exercise.

2. The differences in SWS levels between fit and unfit individuals may represent a residual effect of exercise and not a chronic difference between the groups, i.e., anabolic processes initiated by exercise may persist for several days with the result that SWS levels would not return to baseline levels until these processes were completed.

3. SWS levels may be correlated with a group of physiological factors, e.g., VO_{2max}, which are largely genetically determined, and which are known to be related to physical fitness.

4. Different dietary practices may exist between fit and unfit individuals which could affect their sleep patterns.

Exercise and Immediately Succeeding Sleep

Ten studies have reported the effects of exercise on human sleep during the immediately succeeding night. Three studies have investigated the same effect in animals. Four of the experiments using human subjects (Baekeland and Lasky, 1966; Griffin and Trinder, 1978; Maloletnev, Telia and Tchatchanashvili, 1977; Shapiro, Griesel, Bartel

and Jooste, 1975) and all three animal studies (Boland and Dewsbury, 1971; Hobson, 1968; Matsumoto, Nishisho, Suto, Sadahiro and Miyoshi, 1968) reported facilitative effects of exercise on SWS. Seven studies failed to find this effect in humans (Adamson, Hunter, Ogunremi, Oswald, and Percy-Robb, 1974; Baekeland, 1970; Desjardins, Healey and Broughton, 1974; Griffin and Trinder, 1978; Hauri, 1968; Horne and Porter, 1975; Zir, Smith and Parker, 1971). With one exception (Baekeland, 1970) the discrepancy in the human literature can be accounted for by the probable level of physical fitness of the subjects used. Thus four of the five studies which used physically fit subjects found the facilitative effect. In contrast, six of the seven studies reporting negative effects used unfit subjects, subjects of average fitness or subjects whose physical fitness was not specified. It is likely that the typical level of fitness in the latter group of studies was relatively low. The following review of this literature categorizes studies according to whether humans or animals were used and as to the physical fitness of the human subjects.

Studies on Fit Human Subjects: Baekeland and Lasky (1966) studied the sleep of 10 athletes. An analysis of the first 6 hours of sleep indicated that the mean level of SWS was significantly higher on nights following afternoon exercise (40.1%, 144.4 mins.) than on nights following no exercise (32.5%, 117 mins.). SWS levels were intermediate following evening exercise (35.4%, 127.4 mins.) and not significantly different from SWS levels following either of the other exercise conditions. The mean level of Stage 1 was significantly greater on no exercise nights (7.3%) and on evening exercise nights (6.1%) than following afternoon exercise (4.1%) and time awake was significantly greater following evening exercise (.78%) than following

afternoon exercise (.14%). It was suggested (Baekeland and Lasky, 1966) that following evening exercise there is more disturbed sleep, possibly due to CNS activation, opposing a more general (perhaps metabolic) effect of exercise increasing SWS requirements. Thus evening exercise may act as a 'stressor'.

Shapiro *et al.* (1975) assessed the sleep of two highly trained young men following six morning exercise sessions of increasing severity. The absolute level of SWS increased in both subjects with increasing severity of exercise sessions, Subject A having 80 and 220 mins. of SWS and Subject B having 110 and 160 mins. of SWS following the no exercise condition and the fifth exercise condition (160 mins. at 75% VO_{2max}), respectively. SWS increased at the expense of both REM sleep and Stage 2. Following the most severe exercise session (3 hours at 50% VO_{2max} in a hot box) there were slight decreases in the amount of SWS as compared with the fifth experimental night. Total sleep time (TST) increased with increasing severity of the exercise condition and in the two most severe conditions Subject A had increased levels of wake time. The decrease in SWS in both subjects and the increased wake time in Subject A, following the most strenuous work load, may reflect a stress response to the unusually severe exercise.

The effect of intensive afternoon exercise on the sleep of 15 athletes was studied by Maloletnev *et al.* (1977) and a significant increase in Stage 4 was reported. This study was published in Russian, however, so further details are difficult to ascertain.

Griffin and Trinder (1978) compared the sleep of eight fit and unfit subjects following late afternoon exercise and a day of minimal exercise. They found that Stage 3 increased in fit (53.9 to 66.8 mins.) and decreased in unfit (42.6 to 32.9 mins.) subjects following

exercise, the interaction being significant. A similar trend for total SWS was found, however this did not reach significance, fit subjects having a mean of 114.2 mins. and 125.1 mins., and unfit subjects having a mean of 97.2 and 90.7 mins. SWS, following no exercise and exercise respectively. The facilitative influence of exercise on SWS in fit subjects and the negative effect in unfit subjects were also reflected in a decreased SWS onset latency (latency to Stage 3) in fit and an increased SWS onset latency in unfit subjects as a function of exercise. Finally, a measure of disturbed sleep indicated a significant interaction between fitness level and exercise. Disturbed sleep increased in the unfit and decreased in the fit subjects as a function of exercise. This effect was interpreted as a stress response to the unaccustomed exercise in the unfit subjects.

In an experiment investigating the effects of exercise deprivation on subjects who exercised three or four times a week, Baekeland (1970) failed to find a facilitative effect of exercise on SWS. A non-significant trend towards higher levels of SWS (due to higher Stage 4 levels) on the second night following daytime exercise (59.5 mins. SWS) as compared to the first exercise deprivation night (46.2 mins. SWS), was found. Over the one month exercise deprivation period, however, SWS rose to predeprivation levels. Baekeland (1970) suggests that the failure to obtain a significant effect of exercise, as in his previous study (Baekeland and Lasky, 1966), could have been due to one or both of two factors. Firstly, the subjects in this experiment exercised less strenuously and frequently than in the earlier study. Secondly, the sequential design of the experiment made it impossible to control for possible

increase in SWS due to adaptation to laboratory conditions.

Studies on Unfit Human Subjects: Those experiments which have used subjects who have been identified as unfit, of average fitness, or when samples have been selected without consideration of the fitness of subjects, have uniformly failed to find a facilitative effect of exercise on SWS.

Horne and Porter (1975) assessed the sleep of eight young adult subjects, described as healthy and of average build and fitness, following two no exercise control days, a morning and evening exercise condition and the morning and evening exercise carryover nights. Exercise was standardized for all subjects at 45% VO_{2max} for two 42 min. periods with a 15 min. break in between. No significant differences in whole night percentages of each sleep stage were found between exercise and no exercise conditions. There were some half night differences however. After evening exercise there was a significant increase in Stage 3 for the first half of the night, although there was a non-significant decrease in Stage 3 during the last half of the night. Combined State 2ii (10-20% SW activity), 3 and 4 increased significantly prior to the first REM period. After morning exercise there was a significant decrease in Stage 2ii in the first half of the night and before the first REM period. Horne and Porter (1976) concluded that if exercise is taken late in the day ensuing wakefulness may not be sufficient for complete recovery, thus recovery processes may intrude into the earlier part of sleep. The temporal displacement of sleep stages following evening exercise may represent some sleep disruption, possibly due to disturbance of various physiological processs like basal metabolism (Horne and Porter, 1976).

Hauri (1968) investigated the effects of three presleep activities on a variety of physiological variables during the first 3½ hours of subsequent sleep. Fifteen subjects of unspecified age and fitness were used. Sleep was assessed following 6 hours physical and mental relaxation, 6 hours intense studying and 6 hours of strenuous exercise (bicycle riding and weight lifting). None of the presleep conditions significantly influenced the amount of time spent in any sleep stage. Exercise increased HR throughout the 3½ hour assessment period, and the elevation was significant for all stages except REM. The number of rapid fluctuations of skin potential was also increased after exercise, but only during SWS and only during the first 2 hours of sleep.

Hauri (1968) suggests that waking activities could have a delayed effect on the sleep cycle rather than an immediate one and the physiological imbalance after heavy physical exercise might have to subside before the sleep cycle can be influenced. Since only the first 3½ hours of sleep were recorded such an effect may have been missed. These results have been interpreted by other investigators (Baekeland and Lasky, 1966) as being due to a stress effect caused by evening exercise, as opposed to afternoon exercise. The data could also be viewed as a result of stress caused by prolonged exercise in a largely unfit population.

Desjardins *et al.* (1974) studied the sleep of six males, whose fitness levels were not reported, after a no exercise control day and after early evening high (HR = 185-205 bpm) and low (HR = 140-148 bpm) intensity treadmill exercise. No significant increase in SWS levels was found following exercise, although there was a significant decrease in REM after both levels of exercise which Desjardins *et al.*

(1974) hypothesize may represent a stress effect. In addition, HR was significantly elevated up to 60 mins. after going to bed.

The effect of exercise on EEG sleep variables and daytime secretion of HGH and corticosteroids in twelve males subjects, was investigated by Adamson *et al.* (1974). Five of the subjects participated regularly in sports but were not trained athletes while the remaining seven took little exercise. Following strenuous but not exhausting afternoon exercise, subjects fell asleep slightly more quickly, had slightly less SWS, less REM, and sleep was more broken, than following a no exercise control day, but none of these differences were significant. However, after exercise there was a significant increase in HGH and a significant decrease in corticosteroids. Adamson *et al.* (1974) comment that it is possible that after exercise some increase in intrasleep restlessness opposes any exercise induced tendency to increase SWS.

Zir *et al.* (1971) investigated the effects of exercise on HGH secretion and SWS levels in 10 male subjects, "in good condition". No consistent pattern of SWS or HGH augmentation was found following light or moderate afternoon exercise. There was, however, an overall increase in mean sleep time following exercise conditions.

Finally, as mentioned in the previous section, Griffin and Trinder (1978) compared the sleep of unfit subjects following no exercise and afternoon exercise conditions. They found a decrease in Stage 3 and SWS levels, an increase in SWS latency and an increase in combined MT, wake time and Stage 1, following exercise.

Animal Studies: A number of animal studies have provided support for a facilitative effect of exercise on SWS. Hobson (1968) made continuous

EEG recordings from cats following 2 hours subtotal sleep deprivation and 2 hours moderate treadmill exercise. Following exercise the cats went almost immediately to sleep, while after sleep deprivation they engaged in waking activities for about an hour before sleeping. In the first five hours of recording there was a significantly higher level of SWS and later REM sleep onset following the exercise condition.

Pilot studies on 6 cats indicated that 2 hours of very severe exercise resulted in exhaustion and subsequent restless inactivity, hypervigilance and EEG desynchronization (Hobson, 1968). Hobson (1968) proposes that exercise within critical limits produces somnolence, however when the critical level is exceeded sleep disturbance results. It is suggested that there is probably an intermediate level of exercise which causes the two effects to be in balanced opposition, producing no changes in sleep patterns. The arousing effect might be due to the activation of emergency or stress mechanisms, such as the pituitary-adrenocortical system.

Matsumoto *et al.* (1968) compared sleep recordings from eight rats for 24 hours after 4 hours of food, water and sleep deprivation, and 4 hours of treadmill exercise. SWS was accentuated by the exercise: it appeared earlier and there was significantly more of it. REM sleep levels were within the normal range although they appeared significantly later after exercise. These effects were not observed in four younger rats after similar experimental procedures, however they did not appear as tired as the older rats.

In a comparable study, Boland and Dewsbury (1971) recorded the EEG of 12 rats for 4 hours following fast wheelrunning for 12 secs. in

every minute for 2½ hours and following 2½ hours of being undisturbed. They found that the rats had significantly higher levels of SWS, shorter latency to SWS and shorter mean length of awake episodes following exercise.

Two other studies (Ryback and Lewis, 1971; Webb and Agnew, 1973) have adopted somewhat different approaches to investigating the relationship between physical activity and sleep, and thus are difficult to integrate with the studies described above. Webb and Agnew (1973) assessed the effects of two sleep deprivation conditions on recovery night sleep in eight subjects in good physical health. In a bedrest condition subjects remained in bed but awake for three days and two nights while in an exercise condition subjects exercised for 15 mins. every other hour and were awake an equivalent period. During the recovery nights there was a significant increase in Stage 4 over baseline levels, however there were no significant differences in sleep variables between the two conditions.

Ryback and Lewis (1971) analyzed the sleep of two groups of subjects before, during, and after five weeks bed-rest. One group received no exercise while the other exercised on a total body ergometer while confined to bedrest. In the combined group there were significant increases in SWS and Stage 4 and decreases in Stages 1 and 2 during the bedrest period when compared with baseline levels. The increase in SWS and Stage 4 levels remained in the recovery phase but this was not significant in the case of SWS. When the levels of SWS during bedrest of the exercise and nonexercise groups were compared separately with baseline levels, only the nonexercise group showed a significant increase, although there was a progressive increase in SWS

in the exercise group over the bedrest period. Ryback and Lewis (1971) proposed that physiological restoration is needed when muscles are being used considerably less than usual to combat disuse atrophy, and that SWS may increase during bedrest to aid this reparative process, especially in the no exercise condition. The continued high levels of Stage 4 during recovery may be due to stress on muscles as they recover from "atrophied" to "normal".

Conclusions

A review of the literature indicates that there are possibly two distinct effects of exercise on sleep. Firstly, fit subjects have higher levels of SWS than unfit subjects independent of daytime exercise. Secondly, daytime exercise facilitates subsequent SWS levels in fit but not unfit subjects. The findings that habitual exercise results in a sustained elevation of SWS levels and that fit subjects show an increase in SWS following daytime exercise, are compatible with a bodily restorative theory of SWS. In contrast, however, the failure of unfit subjects to show an increase in SWS levels following exercise does not appear compatible with the restorative model since it would be predicted that all subjects, independent of physical fitness, would require increased anabolism following exercise, and thus increased levels of SWS. However, an explanation for the failure of exercise to increase SWS in unfit subjects, which is compatible with the bodily restorative theory of SWS has been hypothesized.

This explanation of the data argues that unfit subjects when subjected to unaccustomed exercise, experience a physiological

stress[1] effect which results in sleep disturbance and a counteraction of the facilitative effect of exercise on SWS levels. The possibility that physiological stress may offset the effect of exercise has been suggested by a number of other writers (Adamson *et al.*, 1974; Baekeland and Lasky, 1966; Desjardins *et al.*, 1974; Griffin and Trinder, 1978; Hauri, 1968; Hobson, 1968; Horne and Porter, 1975). Both those studies concerned with the relationship between exercise and sleep and those concerned with the effect of exercise on physiological systems offer evidence in support of the stress effect hypothesis. The former will be discussed first.

A number of studies have reported results that may be interpreted as indicative of a stress effect in unfit subjects in response to exercise. Two studies report increases in levels of combined wake-time, movement time (MT) and Stage 1 (Adamson *et al.*, 1974; Griffin and Trinder, 1978). Sleep disturbance, as indicated by temporal displacement of sleep stages, has been reported by Griffin and Trinder (1978) who found increases in SWS latency. Reduced levels of SWS and REM sleep in response to exercise are also consistent with the stress hypothesis. Thus slight decreases in SWS have been reported in unfit subjects by Adamson *et al.* (1974) and Griffin and Trinder (1978). Decreases in REM sleep have been shown by Adamson *et al.* (1974) and Desjardins *et al.* (1974).

---

1. The term "stress" is one which has a large number of controversial interpretations in the psychological literature. The term is used in the present context in a rather general sense to summarize a number of diverse observations in this literature. While it is highly likely that the phenomenon involves a large physiological arousal component (possibly, as suggested by Hobson (1968), it is a response of the sympathetic-adrenal system), it is believed that it is too early to specify the exact nature of the physiological or psychological process involved.

Indications of physiological arousal during sleep in studies which failed to observe facilitative effects of exercise are also relevant. Two studies have reported heart rate increases (Desjardins *et al.*, 1974; Hauri, 1968) and one rapid fluctuations in skin potential (Hauri, 1968). However, it should be noted that none of the studies reporting positive effects measured this type of variable in fit subjects.

It would be predicted by the stress effect hypothesis that fit people would also show a stress response if they performed unusually severe exercise or exercise from which they had no time to recover before sleep. Two studies of fit subjects support this prediction. Shapiro *et al.* (1975) reported lower levels of SWS after extreme as opposed to moderate levels of exercise. Baekeland and Lasky (1966) reported lower levels of SWS and increased levels of wake time and Stage 1 following evening exercise rather than afternoon exercise.

Finally, one study from the animal literature provides support for the stress effect hypothesis. Hobson (1968) reports that while moderate exercise facilitates sleep in cats, severe exercise results in hypervigilance and restlessness.

Consistent with the stress effect hypothesis is the fact that strenuous exercise, such as that performed by unfit subjects in many of the studies described, has been reported to result in physiological arousal. For example, as discussed in Chapter 3, following exhausting exercise, HR and metabolic rate take several hours to return to basal levels due to sustained sympathetic nervous system arousal (Simonson, 1971). This recovery process is prolonged in unfit individuals (Kreuzer, 1964), and thus is more likely to impinge into

sleep time and act as a stressor in unfit than in fit individuals.

Both the available evidence and general opinion strongly support the view that the failure to find a facilitative effect of exercise on SWS in unfit subjects is due to a stress response induced by the exercise programmes used in earlier studies. This hypothesis would predict that the possibility of observing an exercise effect in an unfit population would be maximized if the physiological stress of exercise were minimized while still maintaining a sufficiently high level of catabolism. The present study was designed to test this hypothesis by providing the appropriate conditions to minimize stress.

The previous review of the literature suggests that three factors are relevant in minimizing stress effects present during sleep following exercise.

1. There is considerable argument in favour of late afternoon as opposed to evening exercise (Baekeland and Lasky, 1966; Desjardins *et al.*, 1974; Hauri, 1968; Horne and Porter, 1976). Thus to minimize stress and maximize the facilitative effect of exercise on SWS, exercise should be conducted during the afternoon.

2. It has been suggested that there may be an inverted U-shaped relationship between exercise level and subsequent SWS, such that a level of exercise insufficient to cause a change in catabolism results in no increase in SWS, a level of exercise sufficient to cause catabolism but insufficient to cause sleep disturbance results in an increase in SWS and a level of exercise severe enough to cause both catabolism and sleep disturbance results in no increase in SWS (Hobson, 1968). Thus, an appropriate level of exercise relative to fitness level may be required to produce a facilitative effect on SWS (Griffin and Trinder, 1978). In order to identify a level of

exercise sufficient to produce the necessary catabolism but at the same time insufficient to produce disturbance of sleep, it would be necessary to parametrically vary exercise level.

3. Three of the six studies which have evaluated the effects of exercise on sleep in unfit subjects have used only a single exercise session (Griffin and Trinder, 1978; Hauri, 1968; Zir *et al.*, 1971) and the remaining three used only two exercise sessions a number of days apart (Adamson *et al.*, 1974; Desjardins *et al.*, 1974; Horne and Porter, 1976). It seems likely that this sudden introduction of exercise would be stressful to subjects. In contrast, after a number of consecutive days of exercise subjects would adapt to the exercise level and experience less stress. Therefore, to minimize stress, subjects should exercise on a number of consecutive days.

Two previous studies, Desjardins *et al.* (1974) and Zir *et al.* (1971) have varied the level of exercise performed by unfit subjects. However, Desjardins *et al.* (1974) used evening exercise and only one testing occasion at each exercise level. Zir *et al.* (1971) used two exercise levels described as light and moderate exercise, on one occasion at each level. In the moderate exercise condition subjects performed a variety of exercises for 6 hours while in the light exercise condition subjects exercised for 2 hours. While it is difficult to determine the exercise levels in terms of percentage of VO_{2max}, both these exercise levels appear quite demanding and may have been sufficient to cause physiological stress. Thus, neither study used appropriate conditions to minimize the hypothesized stress effects of exercise.

The hypothesis tested in the present study was that the failure of earlier studies to find a facilitative effect of exercise on SWS

in unfit subjects was due to stress induced by the exercise sessions which disrupted sleep and counteracted the facilitative effect of exercise on SWS levels. It was predicted that following mild or moderate exercise levels, as compared to no exercise or severe exercise, SWS levels in unfit subjects would be facilitated and the stress effect would be minimized. This effect would be particularly evident after a number of consecutive days of exercise.

CHAPTER 5.

METHOD

Subjects

Twenty four unfit subjects (10 female and 14 male) were recruited, largely from the university community. Eight subjects were paid for their participation. The mean age was 21.67 years (SD = 3.27). The physical fitness of subjects, defined in terms of predicted maximal oxygen uptake (VO_{2max}), was assessed using a submaximal exercise nomogram method described by Åstrand and Rodahl (1970). Subjects were considered unfit if they had a VO_{2max} of equal to or less than 2.1 litres for females and 2.8 l. for males in accordance with data published by Åstrand (1960). The mean VO_{2max} levels were 1.65 l. (SD = .25) and 2.22 l. (SD = .30) for females and males respectively. In addition it was required that subjects did not engage in any regular physical exercise.

Six subjects were assigned to each of four groups, such that the groups were matched as closely as possible for age and baseline SWS levels. This matching procedure was carried out to ensure equivalence of the groups with respect to initial SWS levels. The group means for age and baseline SWS are shown in Table 1. Because of the concern for these two variables it was not possible to have the same distribution of males and females in each group.

While unfit, according to our criteria, subjects were all healthy and were not on medication, they were also within the normal weight range for their physical builds, the mean weights being 56.10 kg. (SD = 7.80) and 68.79 kg. (SD = 8.47) for females and males respectively. Group and individual information as to the sex, weight, pre- and

TABLE 1.

Group means and SDs for age (in years) and baseline SWS levels (in mins.)

	Age (yrs)		Baseline SWS (mins)	
	Mean	SD	Mean	SD
Group 1	20.67	2.49	101.25	23.22
Group 2	22.17	4.06	99.75	34.86
Group 3	22.50	3.73	94.67	36.05
Group 4	21.33	2.05	92.50	19.32

TABLE 2.

Mean VO_{2max} values before and after exercise as a function of exercise severity (means adjusted for missing values)

	Pre-Exercise	Post-Exercise
Group 1	1.90	1.72
Group 2	1.82	1.93
Group 3*	2.25	2.63
Group 4	1.90	2.30

* Mean values are higher in this group as it contains a higher proportion of male subjects.

post-exercise VO_{2max} levels, age and baseline SWS levels of subjects are recorded in Appendix I.

Design

A 4 x 4 factorial design with repeated measures on one factor was used. The first factor consisted of four experimental groups which differed according to the level of exercise they were required to perform during the experiment. Thus Gp. 1 did not exercise, Gp. 2 exercised at 50% VO_{2max} for a total of 45 mins., Gp. 3 at 75% VO_{2max} for a total of 60 mins., and Gp. 4 at as close to their maximal level as possible for a total of 60 mins. The exercise levels were selected on the basis of pilot work, as representing a gradation of severity from no exercise to relatively exhausting exercise.

The second factor consisted of four assessment nights, one before and three during the exercise period. Thus each subject in Gps. 2, 3 and 4 exercised in the afternoon (between 1500 and 1800 hours) on five consecutive days. A baseline (B) measure was recorded four nights prior to the first exercise session. There were then three recordings (E1, E3, and E5) during the exercise period on nights following the first, third and fifth exercise session respectively. Two nights prior to the baseline night, subjects were given an adaptation night to eliminate possible "first night" effects (Agnew, Webb and Williams, 1966). Subjects in Gp. 1, who did not exercise, had their sleep assessed on the same schedule as subjects in other groups.

Procedure

Physical Fitness Assessment: Physical fitness levels were assessed using Åstrand's (Åstrand, 1960) submaximal exercise work test. Testing was typically conducted several days before the adaptation night.

During the test, subjects rode a bicycle ergonometer at a submaximal work load (one that required a heart rate (HR) between 150-175 beats/min.) for 6 mins. This was sufficiently long for the HR to have been stable for two or three minutes. HR was continually monitored during the test by means of a cardiotachometer and the mean HR for the final minute of bicycle riding was designated the working pulse for the given work load. Having determined the subject's working pulse for a specific work load, his VO_{2max} was calculated using Åstrand's (Åstrand, 1960) nomogram method. The resulting predicted VO_{2max} was used as an indication of the subject's degree of physical fitness. As indicated above, unfit subjects were defined as those with a VO_{2max} of equal to or less than 2.1 litres for females and 2.8 litres for males.

A marked increase in physical fitness following the five day exercise period, while unlikely, would confound the interpretation of any increase in SWS due to the experimental manipulation, as it could be attributed to either fitness or to the immediate effects of exercise. Therefore, to monitor for such a change a second submaximal exercise work test was given to subjects three days following the final exercise session. Four post-exercise fitness scores are not available, however, as the subjects failed to keep their appointments for this test.

A further fitness test was given to most subjects before and after the exercise period to ensure that any change in fitness recorded on the bicycle ergonometer submaximal work test could not be attributed merely to a mechanical efficiency factor acquired during the five bicycle riding sessions. This test was a modified version of the Harvard Step Test (Åstrand and Rodahl, 1970). HR was recorded

as subjects stepped on to and down from a step 33 cm. high at a rate of 20 steps/min. for females and 30 steps/min. for males, for 5 mins. The working HR on the Step Test was used directly as a measure of the subject's fitness.

VO_{2max} levels and HRs on the Step Test of groups, before and after the exercise period, were compared by means of a 4(groups) x 2(pre- and post-exercise scores) analysis of variance, with repeated measures on the second variable. The statistic of interest was the interaction effect between exercise severity and occasion of fitness testing. This interaction was statistically significant for both VO_{2max} levels ($F(3,20) = 3.27$, $p < .05$) and HR scores ($F(3,20) = 4.60$, $p < .01$). Thus while VO_{2max} and HR scores of Gps. 1 and 2 remained relatively constant, VO_{2max} scores increased and HR scores decreased in Gps. 3 and 4 following the exercise period (VO_{2max} levels are recorded in Table 2).

While the change in fitness levels was statistically significant it was small in magnitude. This is illustrated by the fact that when compared with the normative material published by Åstrand (1960) all subjects remained in the low fitness categories. The level of significance attained was most likely due to the uniformity of the sample which resulted from the selection procedures. As the magnitude of the effect is small it would be unlikely that it would result in SWS changes as a function of physical fitness. Nevertheless, the change indicates the effectiveness of the exercise program in placing demands on the subjects in the two more severe exercise groups.

The main effect of groups was not significant for VO_{2max} levels $F(3,20) = 3.00$, $p > .05$) or for HR scores ($F(3,20) = 1.73$, $p > .05$).

However, the main effect of testing occasion was significant in the case of VO_{2max} levels ($F(1,20) = 5.76$, $p < .05$) although not in the case of HR scores ($F(1,20) = 2.51$, $p > .05$).

Exercise Sessions: All exercise was performed on a stationary bicycle ergonometer. There were a number of reasons for selecting this form of exercise.

1. It enabled the work load demanded of each subject to be closely controlled in terms of % VO_{2max}.

2. Subjects unused to exercising were less likely to sustain injuries while riding a stationary bicycle than while running or playing an unfamiliar sport.

3. Unlike many forms of exercise, riding a bicycle ergonometer is not affected by inclement weather conditions.

The second two issues were of some concern as it was necessary to complete all exercise sessions within a specified schedule.

Subjects in the three exercise groups rode the bicycle ergonometer, under supervision, between 1500 and 1800 hours, on five consecutive days. Subjects in Gp. 2 exercised at 50% VO_{2max}. That is at a level maintaining a HR of 138 beats/min. for females and 128 beats/min. for males (Åstrand and Rhyming, 1954), for 45 mins. divided into 15 min. sessions by 10 min. rests. The mean work loads for the exercise sessions were 263 kpm/min. (SD = 75) and 450 kpm/min. (SD = 0) for females and males respectively.

Subjects in Gp. 3 were required to work at 75% VO_{2max} for 60 mins. divided into 15 mins. sessions by 10 min. rests. This work load required subjects to maintain a working pulse of 160 beats/min. (Åstrand and Rhyming, 1954). The mean work load for this group was 725 kpm/min.

(SD = 113). (N.B. All subjects were male.)

Subjects in Gp. 4 were asked to exercise at a work load as close to their maximal level as possible for a total of 60 mins. divided into 15 min. sessions by 10 min. rests. The mean work load for Gp. 4 was 500 kpm/min. (SD = 87) for females and 875 kpm/min. (SD = 156) for males. None of the subjects in this group could complete their exercise sessions in the specified schedule on the first exercise day. On this day subjects required at least twice the number of scheduled rests. However, by the fifth day all subjects found the exercise considerably easier, 4 being able to complete their exercise sessions according to schedule.

Sleep Assessment: On adaptation and sleep recording nights, subjects reported to the sleep laboratory an hour before their usual bedtime. The sleep laboratory consists of two separate, sound attenuated bedrooms (thus enabling two subjects to be run concurrently), with an adjacent room for sleep monitoring equipment. During the hour prior to the subject's normal bedtime, the subject prepared for bed and the recording electrodes were attached. Electroencephalogram (EEG), electromyogram (EMG) and electrooculogram (EOG) information was collected according to the standardized procedures described by Rechtschaffen and Kales (1968). One exception to these procedures was used in that a single bipolar EOG was recorded rather than two monopolar channels (Wells, Allen and Wagman, 1977). All recordings were made on a Bechman 411 Dynograph. EEG amplification was set at 100μV/cm with a time constant of 1 sec. The paper speed was set at 10 mm/sec.

The light was turned out at approximately the subject's normal bedtime. Subjects were not disturbed during the night but were woken

at their normal rising time.

Scoring: The polygraph sleep records were scored blind by two scorers with an inter-rater reliability of greater than 90%. Each 30 sec. epoch was scored a particular stage according to the criteria described by Rechtschaffen and Kales (1968).

Measurement of Stress: As discussed in Chapter 4 the term "stress response" has been used by a number of authors in a general sense to account for certain effects of exercise on sleep. Its status has been explanatory rather than involving a specific physiological or psychological response. The reasons for proposing the concept have varied but most frequently have involved measures of sleep disturbance following certain experimental manipulations of exercise. While the aim of the present study was to explore the effects of exercise on SWS following experimental manipulations designed to minimize this hypothetical response it would have been an advantage to measure it in some way. However, without knowing the exact nature of the stress reponse it was difficult to decide how best to measure it. Neither the sleep and exercise literature, nor the physiological literature on exercise gave any clear indication as to which physiological system to monitor. In this situation it seemed most appropriate to use measures of sleep disturbance itself as indications of the stress response.

There were two factors supporting this decision. Firstly, as mentioned, sleep disturbance measures figured prominently in the original arguments in support of the stress response hypothesis. Secondly, it is possible that the hypothesized effect of the stress on SWS is mediated by a general disturbance of sleep. Thus, a number of sleep variables, which are generally accepted as indicators of disturbed or poor sleep, were analyzed.

TABLE 3.

F values and significance levels (P) of NREM sleep variables

		Days of Exercise	Level of Exercise	Days x Level of Exercise
Stage 2 (mins)	F	1.84 (3,60)*	.35(3,20)	.94 (9,60)
	P	.15	.79	.50
Stage 2(%TST)	F	1.81	.36	1.65
	P	.15	.78	.12
Stage 3 (mins)	F	.12	1.05	1.04
	P	.95	.39	.42
Stage 3(%TST)	F	.15	1.05	1.00
	P	.93	.35	.44
Stage 4 (mins)	F	.29	.42	1.32
	P	.83	.74	.24
Stage 4 (%TST)	F	.50	.51	1.38
	P	.69	.68	.22
SWS (mins)	F	.18	.13	.47
	P	.91	.94	.88
SWS(%TST)	F	.52	.10	.36
	P	.67	.96	.95
Stage 2+3+4 (mins)	F	1.12	.23	.99
	P	.35	.88	.45
SWS latency (mins)	F	2.35	1.27	1.79
	P	.08	.31	.09

* d.f.

CHAPTER 6.

RESULTS

Each sleep variable was analyzed by means of a 4 (levels of exercise) x 4 (days of exercise) analysis of variance with repeated measures on the second factor. As baseline values for variables other than SWS tended to vary between groups a second analysis was also conducted on each variable using a 4 (levels of exercise) x 3 (days of exercise) analysis of covariance in which the baseline level was used as the covariate for each exercise day (Winer, 1971, p.796). As the results were unaffected by the nature of the analysis only the former method is reported here. Condition means for all sleep variables analyzed are presented in Appendix 2.

NREM Sleep Variables

The F ratios and probability levels for a number of NREM sleep variables are reported in Table 3. The stress effect hypothesis would predict a significant main effect of the level of exercise for SWS variables, with the moderate exercise level groups (Groups 2 and 3) showing greater positive effects than the zero or maximal exercise groups. This hypothesis would also predict an increase in SWS in exercise groups over days which may appear as a significant interaction between exercise level and days or even as a main effect of days.

As indicated in Table 3, no significant differences were found in any NREM variable as a function of either exercise level or exercise duration. Further analysis of Stage 3, 4 and SWS (3 + 4) by the combination of various groups, for example, Group 2 plus 3 compared with Group 1, and with Group 4, gave results identical to the initial analysis. The independence of SWS levels from the independent variables is clearly indicated in Table 4. Thus, there is no evidence to

TABLE 5.

F values and significance levels (P) of sleep variables indicative of poor and disturbed sleep

		Days of Exercise	Level of Exercise	Days x Level of Exercise
Sleep Onset Latency (mins)	F	.95 (3,60)*	.21(3,20)	.94(9,60)
	P	.42	.89	.50
Awake in SPT (mins)	F	1.55	.32	1.1
	P	.21	.81	.37
Awake in SPT (%SPT)	F	1.47	.44	1.06
	P	.23	.73	.41
Total Time Awake (mins)	F	.82	.27	1.11
	P	.49	.84	.37
Total Time Awake (%TiB)	F	.68	.32	1.16
	P	.57	.81	.34
No. of Awakenings	F	1.99	1.18	1.09
	P	.12	.34	.39
Stage 1 (mins)	F	.37	.11	1.02
	P	.78	.95	.44
Stage 1 (%TST)	F	.47	.32	1.17
	P	.71	.81	.33
MT (mins)	F	.48	1.01	1.28
	P	.70	.41	.27
MT (%TST)	F	.39	.97	1.24
	P	.76	.43	.29
Awake + MT + Stage 1 (%SPT)	F	1.18	.01	1.04
	P	.33	.99	.42
Awake + MT + Stage 1 (%TiB)	F	.54	.16	1.42
	P	.66	.92	.20

* d.f.

TABLE 4.

Mean SWS Levels (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	101.25	96.75	105.50	94.08	99.40
Group 2	99.75	106.08	98.08	108.00	102.98
Group 3	94.67	97.83	99.42	101.08	98.25
Group 4	92.50	97.67	95.50	95.58	95.31
Mean	97.04	99.58	99.63	99.69	98.99

support the restorative theory, and the stress hypothesis does not appear to account for the failure of exercise to facilitate SWS in unfit subjects.

Sleep Disturbance Measures

The results of the analyses of variance performed on a number of measures of poor or disturbed sleep are summarized in Table 5. As can be seen from this table, no significant differences were found in any of these variables. The absence of exercise effects is illustrated in Table 6 in which combined values of time awake, movement time and Stage 1 as a percent of total time in bed, are presented.

Sleep Length Variables

Analyses of variance were performed on three sleep length variables, time in bed (TiB), sleep period time (SPT) and total sleep time (TST).

TABLE 6.

Mean Group levels of time awake + Stage 1 + movement Time expressed as a percent of TiB, for each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	16.49	15.62	12.33	17.30	15.44
Group 2	15.55	14.81	18.89	15.71	16.24
Group 3	13.60	17.83	13.53	12.12	14.27
Group 4	18.10	15.28	12.65	18.33	16.09
Mean	15.94	15.89	14.35	15.87	15.51

These results are reported in Table 7. No significant differences were found as a result of level or days of exercise.

TABLE 7.

F values and significance levels (P) of sleep duration variables

		Days of Exercise	Level of Exercise	Days x Level of Exercise
Time in Bed (mins)	F	1.41(3,60)*	.48(3,20)	.17(9,60)
	P	.25	.70	.99
Sleep Period Time (mins)	F	.86	.21	.50
	P	.47	.89	.87
Total Sleep Time (mins)	F	.39	.36	.44
	P	.76	.78	.91

* d.f.

REM Sleep Variables

The results of analyses of variance performed on REM sleep duration and REM latency are shown in Table 8. No significant differences were found in these variables, either as a function of exercise level or duration.

TABLE 8.

F values and significance levels (P) of REM sleep variables

		Days of Exercise	Levels of Exercise	Days x Level of Exercise
Stage REM (mins)	F	.50(3,60)*	1.01(3,20)	1.04(9,60)
	P	.69	.41	.42
Stage REM (%TST)	F	1.02	.73	1.48
	P	.39	.54	.18
REM Latency (mins)	F	.71	.60	1.16
	P	.56	.62	.34

CHAPTER 7.

DISCUSSION

The results of the present study offer no support for the bodily restorative theory of SWS since they provide no evidence that afternoon exercise can produce an increase in SWS in unfit subjects. The data further suggest that the failure of this study to demonstrate the effect cannot be accounted for by a stress effect counteracting an increase in SWS.

The failure to find an increase in SWS under conditions designed to minimize the effects of stress was unexpected as there is a considerable amount of evidence in the literature consistent with this widely argued view. Because this hypothesis is appealing, it was necessary to consider whether other aspects of the experiment may have mitigated against the effect.

It might be argued that the sample size was insufficient to demonstrate a significant effect. Two factors make this improbable. Firstly, the sample size was comparable with other studies from this laboratory which have produced positive effects. Thus, Griffin and Trinder (1978) used two groups of eight subjects for two nights each (df = 31), while Bruck (1978) used four groups of six subjects for three nights (df = 71). Secondly, the SWS data of the present study did not suggest even the slightest trend or suggestion of an effect (see Table 3).

It could also be argued that the failure to observe an increase in SWS following exercise was due either to the low intensity conditions being too demanding, or the high intensity conditions being

insufficiently demanding, in other words, that the various exercise levels did not encompass a sufficient range of conditions. The former was viewed to be highly unlikely. To begin with, it is difficult to consider an exercise level of 50% VO_{2max} for 45 mins. to be in any way demanding. Secondly, the absence of any indication of sleep disturbance in the most severe exercise group makes it unlikely that the less severe conditions would be stressful to subjects. Finally, it could be argued that if the low exercise condition, Gp. 2, was a difficult task for the subjects it would be unlikely that subjects in Gp. 4 would have been able to complete the task at all.

It was more likely that all three exercise levels were insufficiently demanding. This interpretation would be consistent with the failure to show sleep disturbance under any conditions in the experiment. However, for two reasons, this alternative was not considered probable.

1. There was a small but statistically significant increase in fitness levels in subjects in the more severe exercise conditions following only five days of exercise. This, in itself, was rather compelling evidence that the higher exercise levels were sufficient to make catabolic demands on the subjects.

2. As mentioned in the Method, subjects in the most severe exercise condition were unable to complete their exercise according to the usual schedule on the first three days, requiring a greater number of rest periods.

If it was accepted that the range of exercise levels was indeed appropriate to the aims of the experiment then the failure to show any indication of sleep disturbance needs to be considered. While it was true that the aim of the experimental design was to minimize

stress it had been expected that on the first exercise night, particularly in Gp. 4, there would have been some sleep disturbance. The most plausible explanation for the failure to do so, would be that the more intense exercise levels were sufficiently severe to produce changes in catabolism, as indicated by the effect on fitness levels, but insufficiently severe to stress the subjects and thus disturb sleep. This is possible as the literature reviewed in Chapter 3 suggests that prolonged and excessive psychological arousal following exercise occurs most prominently with very severe and exhausting exercise. The particular exercise levels used in this study, in combination with afternoon as opposed to evening exercise, could have meant that any stress produced would have been dissipated by bed time.

It is also possible that the absence of effects of exercise on sleep disturbance measures was due to the particular nature of the exercise used, riding a stationary bicycle. Those studies which have reported increases in sleep disturbance measures used exercise such as running (Adamson *et al.*, 1974; Baekeland and Lasky, 1966; Griffin and Trinder, 1978) and ballgames (Adamson *et al.*, 1974; Baekeland and Lasky, 1966). Moderate bicycle ergonometer exercise resulted in no change in sleep disturbance measures in a study by Horne and Porter (1975).

To this point the argument has assumed that the hypothetical stress response would be reflected in, or measured by, sleep disturbance measures. As discussed in the Method this assumption has rested on two grounds.

1. The stress concept itself developed out of a number of observations relating sleep disturbance to experimental manipulations which

were thought likely to produce physiological stress.

2. It is most likely that the hypothetical stress response would effect both SWS and the quality of sleep and thus sleep disturbance measures. However, it remains logically possible that the effect of stress could be to reduce SWS and leave the level of disturbed sleep unaffected. In this case, a stress response could be operating, undetected by sleep disturbance measures, counteracting the effect of exercise on SWS. However, if this were so there still seems no reason why the level of this response would not be proportional to the intensity of the exercise with corresponding changes in SWS.

The preceding section indicates that there are no obvious reasons for the failure of the present data to support the hypothesis. However, in the case of a negative result it must always be considered possible that unapparent factors were involved. Nevertheless, there appears to be no compelling reason to modify the conclusion that exercise in unfit individuals does not increase SWS levels and that, in this study at least, the failure is not due to a stress effect.

The failure to find an effect of exercise on SWS in unfit subjects both in the present study, in which a range of exercise loads and an extended number of exercise sessions were used, and in a number of other studies, provides strong evidence that exercise does not have a facilitative effect on SWS in this population. This conclusion is incompatible with present statements of the bodily restorative theory of SWS and thus supports those theorists who have either denied a restorative role for SWS or restricted its effect to the CNS.

Two recent studies (Bruck, 1978; Walker, Floyd, Fein, Cavness, Lualhati and Feinberg, 1978), only available subsequent to the collection of the present data, have elvaluated other aspects of the relationship

between exercise and SWS. Both of these studies, one having been conducted in the same laboratory as the present study (Bruck, 1978), failed to replicate the exercise effect in fit subjects. That is, they found no difference in SWS levels on nights following afternoon exercise as opposed to nights following no exercise, in fit subjects. In addition, both studies tested the effect of physical fitness on SWS levels. Bruck (1978) replicated earlier studies which had found chronically higher levels of SWS in fit as compared to unfit individuals. Walker *et al.* (1978) failed to observe the effect in SWS (Stages 3 + 4) but did find differences in NREM sleep (Stages 2 + 3 + 4). This study is the first published report which does not find high levels of SWS in physically fit individuals.

It is clear that no firm conclusions can yet be reached as to the relationship between exercise, physical fitness and sleep in human subjects. However, a number of tentative conclusions can be drawn.

1. In the light of results from the present study it can be argued with some degree of confidence that exercise does not affect SWS on the immediately succeeding night in unfit subjects.

2. It is also possible that this is true for fit subjects although the positive effects reported in several studies (Baekeland and Lasky, 1966; Griffin and Trinder, 1978; Shapiro *et al.*, 1975) would suggest that the facilitative effect of exercise on SWS exists in fit subjects under particular circumstances yet to be identified.

3. Despite the recent results of Walker *et al.* (1978), the most robust finding is the sustained elevation of SWS as a function of physical fitness.

The bodily restorative theory of SWS requires some revision in view of the conclusions stated above. In particular, the theory requires modification to account for the finding that SWS appears to be more closely related to chronic activity levels than day to day variations in activity.

Thus it could be argued that SWS fulfils a peripherally restorative function in which anabolic activity would be set according to long term requirements. This version of the model would predict changes in SWS levels only in response to sustained alterations in activity levels. Considering the relatively stable nature of sleep patterns and other biological rhythms this version of the restorative model has some intrinsic appeal. Such a concept would also be consistent with other well known physiological consequences of physical exercise. Thus habitual exercise causes a variety of long term physiological and functional changes which enhance performance of and recovery from exercise (see Chapter 3). A sustained elevation of SWS may be a similar adaptation enabling the more efficient restoration or maintenance of muscle tissues on which there are heavy demands.

It could also be speculated, if it should be found that fitness is related to a facilitative effect of exercise on SWS, that increased fitness results in an increased flexibility of the SWS system under some circumstances. This flexibility may enable an immediate facilitative effect of exercise on SWS to occur.

As a final point it should be noted that the apparent relationship between chronic activity levels, as defined by physical fitness, and SWS is open to at least two alternative interpretations, which,

if either were valid, would further compromise restorative theory.

1. SWS levels may be correlated with other physiological factors, such as oxygen uptake and athletic ability, which are to a considerable extent genetically determined, or are a relatively permanent result of early training and which are likely to result in physical fitness. Thus the selection of high activity subjects on the basis of physical fitness, or athletic ability, may involve the assessment of a genetic or early training factor, unrelated to current activity levels.

2. Different dietary practices between fit and unfit individuals may result in different sleep patterns in the two groups as nutritional level has been shown to affect sleep (Crisp and Stonehill, 1976).

The role of habitual exercise or fitness on both the sustained elevation of SWS and the immediately facilitative effect of exercise on SWS would be resolved by an experiment in which sleep was monitored on at least two occasions within the same subject, once before and once after a physical fitness programme designed to make physically unfit subjects fit. Diet would need to be simultaneously measured. Such a study would have relevance to both the validity of these phenomena and the nature of the factors determining them.

REFERENCES

Adam, K. and Oswald, I. Sleep is for tissue restoration. *Journal of The Royal College of Physicians*, 1977, *11*, 376-388.

Adamson, L., Hunter, W.M., Ogunremi, O.O., Oswald, I. & Percy-Robb, I.W. Growth hormone increase during sleep after daytime exercise. *Journal of Endocrinology*, 1974, *62*, 473-78.

Agnew, H.W., Webb, W.B. and Williams, R.L. Comparison of Stage 4 and 1 - REM sleep deprivation. *Perceptual and Motor Skills*, 1967, *29*, 47-56.

Agnew, H.W., Webb, W.B. & Williams, R.L. The effects of Stage four sleep deprivation. *Electroencephalography and Clinical Neurophysiology*, 1964, *17*,68-70.

Agnew, H.W., Webb, W.B. & Williams, R.L. The first night effect: an EEG study of sleep. *Psychophysiology*, 1966, *2*, 263-266.

Allison, T. & Ciccetti, D.V. Sleep in mammals: Ecological and Constitutional correlates. *Science*, 1976, *194*, 732-734.

Allison, T. & Van Twyver, H. The evolution of sleep. *Natural History*, 1970, *79*, 57-65.

Aserinsky, E. & Kleitman, N. Regularly occurring periods of eye-motility and concomitant phenomena during sleep. *Science*, 1953,*118*, 273-274.

Åstrand, I. Aerobic work capacity in Men and Women with special reference to Age. *Acta Physiologie Scandinavia*, 1960, *49*, Suppl. 169.

Åstrand, P.-O. & Rhyming, S. A nomogram for calculation of aerobic capacity (physical fitness) from pulse rate during submaximal work. *Journal of Applied Physiology*, 1954, *7*, 218-222.

Åstrand, P.-O. & Rodhal, K. *Textbook of Work Physiology*. New York: McGraw-Hill Book Co., 1970.

Atkinson, D.E. The energy charge of the adenylate pool as a regulatory parameter. Interaction with feedback modifiers. *Biochemistry*, 1968, *7*, 4030-4034.

Baekeland, F. Exercise deprivation, sleep and psychological reactions. *Archives of General Psychiatry*, 1970, *22*,365-369.

Baekeland, F. & Lasky, R. Exercise and sleep patterns in college athletes. *Perceptual and Motor Skills*, 1966, *23*, 1203-1207.

Beck, U., Březinová, V., Hunter, W.M. & Oswald, I. Plasma growth hormone and slow wave sleep increase after interrupted sleep. *Journal of Clinical Endocrinology and Metabolism*, 1974, *40*, 812-815.

Berger, R.J. Bioenergetic functions of sleep and activity rhythms. *Federation Proceedings*, 1975, *34*, 97-102.

Berger, R.J. Oculomotor control: A possible function of REM sleep. *Psychological Review*, 1969, *76*, 144-164.

Berger, R.J. Tonus of extrinsic laryngeal muscles during sleep and dreaming. *Science*, 1961, *134*, 840-841.

Berger, R.J. & Oswald, I. Effects of sleep deprivation on behaviour, subsequent sleep and dreaming. *Journal of Mental Science*, 1962, *108*, 457-459.

Berger, R.J. & Scott, T.D. Increased accuracy of binocular depth perception following REM sleep periods. *Psychophysiology*, 1971, *8*, 763-768.

Berger, R.J. & Walker, J.M. Oculomotor co-ordination following REM and Non-REM sleep periods. *Journal of Experimental Psychology*, 1972, *94*, 216-224.

Boland, B.D. & Dewsbury, D.A. Characteristics of sleep following sexual activity and wheel-running in rats. *Physiology and Behaviour*, 1971, *6*, 145-149.

Boyar, R., Finkelstein, J., Roffwarg, H., Kaper, S., Weitzman, E. & Hellman, L. Synchronization of augmented luteinizing hormone secretion with sleep during puberty. *New England Journal of Science*, 1972, *287*, 582-585.

Brebbia, D.R. & Altschuler, K.Z. Oxygen Consumption Rate and electroencephalographic stage of sleep. *Science*, 1965, *150*, 1621-1623.

Breger, I. Functions of Dreams, *Journal of Abnormal Psychology*, 1967, *72*, 1-28.

Brouha, L. & Radford, E. The Cardiovascular System in Muscular Activity. In W.R. Johnson (Ed.), *Science and Medicine of Exercise and Sport*. New York: Harper and Bros.,1960.

Bruck, D. *Fitness, Exercise, Age and Human Sleep.* Honours Thesis submitted at the University of Tasmania, 1978.

Cartwright, R.D. & Monroe, L.J., Relation of Dreaming and REM sleep: The effects of REM deprivation under two conditions. *Journal of Personality and Social Psychology*, 1968, *10*, 169-174.

Crisp, A.H. & Stonehill, E. *Sleep, Nutrition and Mood.* London: John Wiley and Sons, 1976.

Dement, W.C. The effect of dream deprivation. *Science*,1960, *131*, 1705-1707.

Dement, W.C. & Greenberg, S. Changes in total amount of Stage Four sleep as a function of partial sleep deprivation. *Electroencephalography and Clinical Neurophysiology*, 1966, *20*, 523-526.

Dement, W.C. & Kleitman, N. Cyclic variations in EEG during sleep and their relation to eye movements, body motility and dreaming. *Electroencephalography and Clinical Neurophysiology*, 1957, *9*, 673-690.

Desjardins, J., Healey, T. & Broughton, R. Early evening exercise and sleep. *Sleep Research*, 1974, *3*, 31.

Dewan, E.M. The programming 'P' hypothesis for REM sleep. In E. Hartmann (Ed.), *Sleep and Dreaming*, Boston: Little Brown, 1970.

Dreyfus-Brisac, C. The electroencephalogram of the premature infant and fullterm newborn: Normal and Abnormal development of waking and sleeping patterns. In P. Kellaway & I. Peterson (Eds.), *Neurological and Electroencephalographic Correlative Studies in Infancy*. New York: Grune and Stratton, 1964.

Dunleavy, D.L.F., Oswald, I., Brown, P. & Strong, J.A. Hyperthyroidism, sleep and growth hormone. *Electroencephalography and Clinical Neurophysiology*, 1974, *36*, 259-263.

Ephron, H.S. & Carrington P. Rapid eyemovement sleep and cortical homeostasis. *Psychological Review*, 1966, *73*, 500-526.

Feinberg, I. Changes in Sleep Cycle patterns with age. *Journal of Psychiatric Research*, 1974, *10*, 283-306.

Feinberg, I. Eye movement activity during sleep and intellectual functioning in mental retardation. *Science*, 1968, *159*, 1256-1258.

Feinberg, I. & Carlson, V.R. Sleep variables as a function of age in man. *Archives of General Psychiatry*, 1968, *18*, 239-250.

Feinberg, I. & Evarts, E.V. Changing concepts of the function of sleep: Discovery of intense brain activity during sleep calls for revision of hypotheses as to its function. *Biological Psychiatry*, 1969, *1*, 331-348.

Feinberg, I., Koresco, R.L. & Heller, N. EEG sleep patterns as a function of normal and pathological aging in man. *Journal of Psychiatric Research*, 1967, *5*, 107-113.

Freemon, F.R. Reciprocal environmental surveillance model of sleep. *Journal of Theoretical Biology*, 1970, *27*, 339-340.

Friedman, M. & Strang, L.B. Effect of Longterm corticosteroids and corticotrophin on the growth of children. *Lancet*, 1966, *2*, 568-570.

Greenberg, R. & Pearlman, C. REM sleep and the analytic process: A psychophysiological bridge. Report to the American Psychoanalytic Association. New York, December 1972.

Greenberg, R., Pearlman, C., Fingar, R., Kantrowitz, J. & Kawliche, S. The effects of dream deprivation:Implications for a theory of the psychological functions of dreaming. *British Journal of Medical Psychology*, 1970, *43*, 1-11.

Griffin, S.J. & Trinder, J. Physical fitness, exercise and human sleep. *Psychophysiology*, 1978, *15*, 447-450.

Haider, I. & Oswald, I. Late brain recovery processes after drug overdose. *British Medical Journal*, 1970, *2*, 318-322.

Hartmann, E.L. *The Biology of Dreaming*. Illinois: Charles C. Thomas, 1967.

Hartmann, E.L. *The Functions of Sleep*. New Haven: Yale Uni. Press, 1973.

Hartmann, E.L., Baekeland, F. & Zwilling, G. Psychological differences between long and short sleepers. *Archives of General Psychiatry*, 1972, *26*, 263-268.

Hartmann, E.L., Baekeland, F., Zwilling, G. & Hay, P. Sleep need: how much sleep and what kind? *American Journal of Psychiatry*, 1971, *127*, 1001-1008.

Hauri, P. Effects of evening activity on early night sleep. *Psychophysiology*, 1968, *4*, 267-277.

Hawkins, D.R. A review of psychoanalytic dream theory. *British Journal of Medical Psychology*, 1966, *39*, 85-104.

Henane, R., Bugeret, A., Roussel, B. & Bittel, J. Variations in evaporation and body temperatures during sleep in man. *Journal of Applied Physiology*, 1977, *4*, 50-55.

Hobson, J.A. Sleep after exercise. *Science*,1968, *162*, 1503-1505.

Hockey, G.R. Changes in attention allocation in a multicomponent task under loss of sleep. *British Journal of Psychology*, 1970, *61*, 473-480.

Honda, Y., Takahashi, K., Takahashi, S., Azumi, K. Irie, M., Sakuma, M., Tsushima, T. & Shizume, K. Growth Hormone secretion during nocturnal sleep in normal subjects. *Journal of Clinical Endocrinology*, 1969, *29*, 20-25.

Horne, J.A. Factors relating to energy conservation during sleep in mammals. *Physiological Psychology*, 1977, *5*, 403-408.

Horne, J.A. & Porter, J.M. Exercise and Human Sleep. *Nature*, 1975, *256*, 573-575.

Horne, J.A. & Porter, J.M. Time of day effects with standardized exercise upon subsequent sleep. *Electroencepahlography and Clinical Neurophsiology*, 1976, *40*, 178-184.

Irvine, C.H.G. Effect of exercise on thyroxine degradation in athletes and non-athletes. In K.D. Rose, R.J. Shephard, & W.P. Leary (Eds.), *Sport Medicine: Physiology*. New York: M.S.S. Information Corporation, 1974.

Jacoby, J.H., Smith, E., Sassin, J.F., Greenstein, M. & Weitzman, E.D. *Neuroendocrinology*, 1975, *18*, 9. Adam, K. & Oswald, I., Sleep is for tissue restoration. *Journal of the Royal College of Physicians*, 1977, *11*, 376-388.

Johns, M.W., Masterton, J.P., Paddle-Ledinek, J.E., Patel, Y.C., Winikoff, D. & Malinek, M. *Clinical Science and Molecular Medicine*, 1975, *49*, 629. Adam, K., & Oswald, I., Sleep is for tissue restoration. *Journal of the Royal College of Physicians*, 1977, *11*, 376-388.

Johnson, R.H., Walton, J.C., Krebbs, H.A. & Williamson, D.H. Metabolic fuels during and after severe exercise in athletes and non-athletes. In K.D. Rose, R.J. Shephard, & W.P. Leary (Eds.), *Sport Medicine: Physiology*. New York: M.S.S. Information Corporation, 1974.

Kales, A., Heuser, G., Jacobson, A., Kales, J.D., Hanley, J., Sweizig, J.R. & Paulson, M.J. All-night sleep studies in hypothyroid patients, before and after treatment. *Journal of Clinical Endocrinology and Metabolism*, 1967, *27*, 1593-1599.

Kales, A., Tan, T., Kollar, E., Naitoh, P., Preston, T. & Malmstrom, E. Sleep Patterns following 205 hours of sleep deprivation. *Psychosomatic Medicine*, 1970, *32*, 189-200.

Karacan, I., Rosenbloom, A.L., Londono, J.H., Williams, R.C. & Salis, P.J. Growth hormone levels during morning and afternoon naps. *Behavioural Neuropsychiatry*, 1975, *6*, 67-70.

Karacan, I., Rosenbloom, A.L., Londono, J.H., Salis, P.J., Thornby, J.I. & Williams, R.L. Sleep and the sleep-growth hormone (G.H.) response during

acute fasting. *Psychosomatics*, 1973, *14*, 33.

Karacan, I., Williams, R.L., Finley, W.W. & Hursch, C.J. The Effects of Naps on nocturnal sleep: Influence on the need of Stage 1-REM and Stage 4 sleep. *Biological Psychiatry*, 1970, *2*, 391.

Karpovich, P.V. & Sinning, W.E. *Physiology of Muscular Activity*. Philadelphia: W.B. Saunders Co., 1971.

Kety, S.S. Relationship between energy metabolism in the brain and functional activity. In S.S. Kety, E.G. Evarts & H.L. Williams, (Eds.), *Sleep and Altered States of Consciousness*. Baltimore: Williams and Wilkins, 1967.

Kleitman, N. *Sleep and Wakefulness*. Chicago: Uni. of Chicago Press, 1963.

Kollar, E.J., Pasnau, R.O., Rubin, R.T., Naitoh, P., Slater, G.G. & Kales, A. Psychological, psychophysiological and biochemical correlates of prolonged sleep deprivation. *American Journal of Psychiatry*, 1969, *126*, 488-497.

Korner, A. Growth hormone control of biosynthesis of protein and ribonucleic acid. *Recent Progress in Hormone Research*, 1965, *21*, 205-208.

Kreuzer, F.J.A. Physiological adjustments to exercise. In E. Johl, & I.E. Simon (Eds.), *International Research in Sport and Physical Education*. Illinois: Charles C.Thomas, 1964.

Kuhn, D.J., Meltzer, H.Y., Wyatt, R.J. & Snyder, F. Serum enzyme changes during sleep deprivation. *Nature*, 1970, *228*, 768-769.

Lacey, J.H., Crisp, A.H., Kalucy, R.S., Hartmann, M.K. & Chen, C.N. Weight gain and sleeping EEG. *British Journal of Medicine*, 1975, *4*, 556-558.

Lewis, S., Oswald, I. & Dunleavy, D. Chronic femfluramine administration: some cerebral effects. *British Journal of Medicine*, 1971, *3*, 67-70.

Maloletnev, V.I., Telia, Z.A. & Tchatchanashvili, M.G. Changes in the structure of night sleep in man after intensive muscular work. *Sechenov Physiolical Journal of the U.S.S.R.*, 1977, *63*, 11-20.

Margaria, R. *Biomechanics and energetics of Muscular Exercise*. Oxford: Clarendon Press, 1976.

Matsumoto, J., Nishisho, T., Suto, T., Sadahiro, T. & Miyoshi, M. Influence of fatigue on sleep. *Nature*, 1968, *218*, 177-178.

McFadyen, C.M., Oswald, I. & Lewis, S.A. Starvation and human slow wave sleep. *Journal of Applied Physiology*, 1973, *35*, 391-394.

Meddis, R. On the function of sleep. *Animal Behaviour*, 1975, *23*, 676-691.

Morehouse, L.E. & Miller, A.T. *Physiology of Exercise*. Saint Louis: C.V. Mosby Co., 1971.

Moruzzi, G. Sleep and Instinctive Behaviour. *Archives of Italian Biology*, 1969, *108*, 175-216.

Moruzzi, G. The sleep-waking cycle. *Ergebn. Physiology*, 1972, *64*, 1-165.

Moses, J.M., Johnson, L.C., Naitoh, P. & Lubin, A. Sleep stage deprivation and total sleep loss: Effects on sleep behaviour. *Psychophysiology*, 1975, *12*, 141-146.

Naitoh, P., Johnson, L.C. & Lubin, A. Modification of surface negative slow potential (CNV) in the human brain after total sleep loss. *Electroencephalography and Clinical Neurophysiology*, 1971, *30*, 17-20.

Newman, E.A. & Evans, C.R. Human dream processes as analogous to computer programme clearance. *Nature*, 1965, *206*, 534.

Oswald, I. *Sleep*. Penguin Books, 1974.

Oswald, I. Sleep the great restorer. *New Scientist*, 1970, *46*, 170-172.

Oswald, I., Berger, R.J., Jaramillo, R.A., Keddie, K.M.G., Olley, P.C. & Plunkett, G.B. Melancholia and barbiturates: A controlled EEG body and eye movement study of sleep. *British Journal of Psychiatry*, 1963, *109*, 66-78.

Parker, D.C., Rossman, L.G. & Vanderlaan, E.F. Persistance of human growth hormone release during sleep in the fasted and nonisocalorically fed normal subjects. *Metabolism*, 1972, *21*, 241-252.

Parker, D.C., Sassin, J.F., Mace, J.W., Gotlin, R.W. & Rossman, L.J. Human growth hormone release during sleep: electroencephalographic correlation. *Journal of Clinical Endocrinology*, 1969, *29*, 871-873.

Parmalee, A.H. & Stern, E. Development of states in infants. In C.D. Clemente, D.P. Purpara, & F.E. Mayer (Eds.), *Sleep and the Maturing Nervous System*. New York: Academic Press, 1972.

Pasnau, R.O., Naitoh, P., Stier, S. & Kollar, E.J.
The psychological effects of 205 hours of sleep deprivation. *Archives of General Psychiatry*, 1968, *18*, 496-506.

Peschle, C., Rappaport, I.A., Sasso, G.F., Gordon, A.S. & Condorelli,M.
Mechanism of growth hormone (GH) action on Erythropoiesis. *Endocrinology*, 1972, *91*, 511-520.

Rechtschaffen, A., Cornwell, P. & Zimmerman, W.
Brain temperature variations with paradoxical sleep in the cat. Paper presented at 5th Annual Meeting of the Association for the Psychophysiological Study of Sleep, Washington, D.C., 1965.

Rechtschaffen, A. & Kales, A. (Eds.),
A Manual of Standardized Terminology, Techniques, and Scoring System for Sleep Stages of Human Subjects. Washington, D.C. U.S. Government Printing Office, 1968.

Reite, M., Stynes, A.J., Vaughn, L., Pauley, J.D. & Short, R.A.
Sleep in infant monkeys: Normal values and behavioural correlates. *Physiology and Behaviour*, 1976, *163*, 245-251.

Roffwarg, H.P., Muzio, J.N. & Dement, W.C. Ontogenetic development of the human sleep-dream cycle. *Science*,1966, *152*, 604-619.

Rubin, R.T., Poland, R.E. & Tower, B.B. Prolactin-related testosterone secretion in normal adult men. *Journal of Clinical Endocrinology and Metabolism*,1976,*42*, 112-116.

Ryback, R.S. & Lewis, O.F. Effects of Prolonged Bed Rest on EEG Sleep Patterns in young healthy volunteers. *Electroencephalography and Clinical Neurophysiology*, 1971, *31*, 395-399.

Sassin, J.F., Frantz, A.G., Kapen, S. & Weitzman, E.D.
The nocturnal rise of human prolactin is dependent on sleep. *Journal of Clinical Endocrinology and Metabolism*, 1973, *37*, 436-440.

Sassin, J.F., Parker, D.C., Johnson, L.C., Rossman, L.G., Mace, J.W. & Gotlin, R.W.
The effect of SWS deprivation on human growth hormone release in sleep: preliminary study. *Life Science*, 1969a, *8*, 1299-1307.

Sassin, J.F., Parker, D.C., Johnson, L.C., Rossman, L.G., Mace, J.W. & Gotlin, R.W.
Human growth hormone release: Relation to slow wave sleep and sleep-waking cycles. *Science*, 1969b,*165*, 513-515.

Schnure, J.J., Raskin, P. & Lipman, R.L. Growth hormone secretion during sleep: Impairment in glucose tolerance and

nonsuppressibility by hyperglycemia. *Journal of Clinical Endocrinology and Metabolism*, 1971, *33*, 234-241.

Scott, T.D. The effects of continuous, high intensity, white noise on the human sleep cycle. *Psychophysiology*, 1972, *9*, 227-232.

Shapiro, C.M., Griesel, R.D., Bartel, P.R. & Jooste, P.L. Sleep patterns after graded exercise. *Journal of Applied Physiology*, 1975, *39*, 187-190.

Simonson, E. *Physiology of Work Capacity and Fatigue.* Illinois: Charles C. Thomas, 1971.

Snyder, F. Physiology of human sleep. *Clinical Neurosurgery*, 1971, *18*, 503-536.

Snyder, F. Toward an Evolutionary Theory of Dreaming. *American Journal of Psychiatry*, 1966, *123*, 121-136.

Snyder, F., Hobson, J.A., Morrison, D.F. & Goldfrantz, F. Changes in respiration, heart rate and systotic blood pressure in human sleep. *Journal of Applied Physiology*, 1964, *19*, 417-422.

Takahashi, Y., Kipnis, D.M. & Daughaday, W.H. Growth hormone secretion during sleep. *Journal of Clinical Investigation*,1968, *47*, 2079-2090.

Taub, J.M. & Berger, R.J. Effects of acute sleep pattern alteration depend upon sleep duration. *Physiological Psychology*, 1976 , *4*, 412-420.

Thomas, V. *Exercise Physiology.* Great Britain: Granada Pub. Ltd., 1975.

Tinbergen, N. *The Study of Instinct.* Oxford: Oxford University Press, 1951.

Walker, J.M., Floyd, T.C., Fein, G., Cavness, C., Lualhati, R. & Feinberg, I. Effects of exercise on sleep. *Journal of Applied Physiology*, 1978, *44*, 945-951.

Webb, W.B. Selective and partial deprivation of sleep. In W.P. Koella, & P. Levin (Eds.), *Sleep: Physiology, Biochemistry, Psychology, Pharmacology and Clinical Implications.* Basel: Karger, 1973.

Webb, W.B. Sleep behaviour as a biorythm. In W.P. Colquhoun (ed.), *Biological Rhythms and Human Performance.* London: Academic Press, 1971.

Webb, W.B. The Adaptive functions of sleep. In P. Levin, & W.P. Koella (Eds.), *Sleep.* Basel: Karger, 1974.

Webb, W.B. & Agnew, H.W. Effects on performance of high and low energy expenditure during sleep deprivation. *Perceptual and Motor Skills*, 1973, *37*, 511-514.

Webb, W.B. & Agnew, H.W. Sleep : Effects of a restricted regime. *Science*, 1965, *150*, 1745.

Webb, W.B. & Friedmann, J. Activity as a determinant of sleep in rats. *Psychophysiology*, 1969, *6*, 272.

Wells, D.T., Allen, R.P. & Wagman, A.M.I. A single-channel system of recording eye movements. *Psychophysiology*, 1977, *14*, 73-74.

Wilkinson, R.T. Sleep deprivation. In O.G. Edholm, & A.L. Bacharach (Eds.), *The Physiology of Human Survival*. New York: Academic Press, 1965.

Williams, R.L., Agnew, H.W. & Webb, W.B. Sleep patterns in young adults: An EEG study. *Electroencephalography and Clinical Neurophysiology*, 1964, *17*, 376-381.

Williams, R.L., Hammack, J.T., Daly, R.L., Dement, W.C. & Lubin, A. Response to auditory stimulation, sleep loss and the EEG stages of sleep. *Electroencephalography and Clinical Neurophysiology*, 1964, *16*, 269-279.

Williams, R.L., Karacan, I. & Hursch, C. *EEG of Human Sleep: Clinical Applications*. New York: John Wiley and Sons, 1974.

Winer, B.J. *Statistical Principles in Experimental Design*. New York: McGraw-Hill Book Co., 1971.

Zepelin, H. & Rechtschaffen, A. Mammalian sleep, longevity and energy metabolism. *Brain, Behaviour and Evolution*, 1974, *10*, 425-470.

Zir, L.M., Smith, R.A. & Parker, D.C. Human growth hormone release in sleep: Effect of daytime exercise. *Journal of Clinical Endocrinology*, 1971, *32*, 662-665.

Zloty, R.B., Burdick, J.A. & Adamson, J.L. Sleep of distance runners. *Activitas Nervosa Superior*, 1973, *15*, 217-221.

APPENDIX I.

Group and subject information as to sex, age, weight, baseline SWS, pre- and post-exercise VO_{2max} and pre- and post-exercise HR on the Step Test.

Group 1 subject information as to sex, age, weight, baseline SWS, pre- and post-exercise VO_{2max} and pre- and post-exercise H.R. on the Step Test.

Subject	Sex	Age (yrs)	Wt.(kgs)	Baseline SWS (mins)	Pre-Exercise VO_{2max}(l.)	Post-Exercise VO_{2max}(l.)	Pre-Exercise HR on Step Test (bpm.)	Post-Exercise HR on Step Test (bpm.)
A.V.	F	21	56	92.5	1.8	1.8	155	150
W.J.	F	19	60	104.5	1.5	1.5	-	177
C.M.	M	20	64	141.5	2.6	2.3	148	159
S.E.	M	17	78	70.5	1.7	1.7	180	180
D.B.	F	22	51	103.5	1.4	-	176	-
T.T.	M	25	63	95.0	2.3	1.8	167	174
Mean - Males		20.7	68.3	102.33	2.20	1.93	165.0	171.0
Females		20.7	55.7	100.17	1.57	1.65	165.5	163.5
Total		20.7	62.0	101.25	1.88	1.82	165.2	168.0
SD - Males		4.0	8.4	36.06	.46	.32	16.1	10.8
Females		1.5	4.5	6.66	.21	.21	14.8	19.1
Total		2.7	9.2	23.22	.47	.29	13.6	12.9

Group 2 subject information as to sex, age, weight, baseline SWS, pre- and post-exercise VO_{2max} and pre- and post-exercise HR. on the Step Test.

Subject	Sex	Age(yrs)	Wt(Kgs)	Baseline SWS (mins)	Pre-Exercise VO_{2max}(1.)	Post-Exercise VO_{2max}(1.)	Pre-Exercise HR On Step Test(bpm.)	Post-Exercise HR on Step Test (bpm.)
G.D.	F	20	54	71.5	1.5	1.5	180	175
A.B.	F	29	75	79.5	2.1	-	-	-
A.P.	F	22	51	106.0	1.5	1.5	159	174
G.J.	M	25	81	166.0	2.2	2.1	165	165
M.H.	M	16	66	96.5	2.0	2.1	175	173
M.V.	F	21	56	79.0	2.1	2.2	-	-
Mean - Males		20.5	73.5	131.25	2.10	2.10	170.0	169.0
Females		23.0	59.0	84.0	1.80	1.73	169.5	174.5
Total		22.2	63.8	99.75	1.90	1.88	169.8	171.8
S D - Males		6.36	10.6	49.14	.14	0	7.1	5.6
Females		4.08	10.9	15.12	.35	.40	14.9	.7
Total		4.45	12.2	34.86	.32	.35	9.5	4.6

Group 3 subject information as to sex, age, weight, baseline SWS, pre- and post-exercise VO_{2max} and pre- and post-exercise HR on the Step Test.

Subject	Sex	Age(yrs)	Wt(Kgs)	Baseline SWS (mins)	Pre-Exercise VO_{2max}(1.)	Post-Exercise VO_{2max}(1.)	Pre-Exercise HR on Step Test (bpm.)	Post-Exercise HR on Step Test(bpm.)
K.S.	M	26	64	83.5	2.4	2.4	177	163
M.B.	M	19	66	114.0	2.3	2.1	133	148
M.H.	M	27	54	86.0	1.8	3.4	-	-
C.G.	M	17	66	153.5	2.1	2.6	158	160
C.W.	M	25	87	46.0	2.3	2.5	180	166
A.W.	M	21	74	85.0	2.6	2.8	175	167
Mean - Males		22.5	68.5	94.67	2.25	2.63	164.6	160.8
Females								
Total		22.5	68.5	94.67	2.25	2.63	164.6	160.8
S D - Males		4.1	11.1	36.05	.27	.44	19.6	7.7
Females								
Total		4.1	11.1	36.05	.27	.44	19.6	7.7

Group 4 subject information as to sex, age, weight, baseline SWS, pre- and post-exercise VO_{2max} and pre- and post-exercise HR on the Step Test.

Subject	Sex	Age(yrs)	Wt(kgs)	Baseline SWS (mins)	Pre-Exercise VO_{2max}(l.)	Post-Exercise VO_{2max}(l.)	Pre-Exercise HR on Step Test (bpm.)	Post-Exercise HR on Step Test (bpm.)
H.M.	F	23	60	73.0	1.4	1.8	165	157
J.R.	F	23	48	111.0	1.5	1.5	158	158
A.D.	M	21	68	63.5	2.0	2.6	182	-
N.H.	M	17	67	101.0	2.0	-	180	-
S.M.	M	22	65	103.5	2.8	-	159	-
G.B.	F	22	50	103.0	1.7	2.3	150	120
Mean - Males		20.0	66.7	89.33	2.27	2.60	173.7	-
Females		22.6	52.7	95.67	1.53	1.87	157.7	145.0
Total		21.3	59.7	92.50	1.90	2.05	165.7	145.0
S.D. - Males		2.7	1.5	22.41	.46	0.00	12.7	-
Females		.6	6.4	20.03	.15	.40	7.5	21.7
Total		2.3	8.7	19.32	.51	.49	12.8	21.7

APPENDIX 2.

Condition means for sleep variables analyzed.

APPENDIX 2.

Mean Stage 2 levels (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	210.92	214.75	210.67	206.92	210.82
Group 2	211.58	192.42	186.36	193.67	196.01
Group 3	213.08	212.92	193.67	191.33	202.75
Group 4	199.58	214.58	215.25	188.92	204.58
Mean	208.79	208.67	201.49	195.21	

Mean Stage 2 levels as a percent of TST for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	45.93	46.96	46.79	47.55	46.81
Group 2	47.35	43.61	44.62	44.18	44.94
Group 3	49.89	48.34	44.94	43.62	46.70
Group 4	46.60	49.70	49.16	44.98	47.61
Mean	47.44	47.15	46.38	45.08	

APPENDIX 2

Mean Stage 3 levels (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	33.00	35.75	32.67	32.42	33.46
Group 2	37.83	40.25	38.21	49.83	41.53
Group 3	49.33	40.33	44.92	47.00	45.40
Group 4	34.92	37.00	41.92	31.33	36.29
Mean	38.77	38.33	39.43	40.15	

Mean Stage 3 levels as a percent of TST for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	7.45	8.07	7.42	7.55	7.62
Group 2	8.29	9.26	9.22	11.22	9.50
Group 3	11.35	9.31	10.40	10.72	10.45
Group 4	8.33	8.64	9.61	7.29	8.47
Mean	8.86	8.82	9.16	9.20	

APPENDIX 2.

Mean Stage 4 levels (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	68.25	61.00	72.83	61.67	65.94
Group 2	61.92	65.83	59.88	58.08	61.43
Group 3	45.33	57.50	54.50	54.00	52.83
Group 4	57.58	60.58	53.58	64.25	59.00
Mean	58.27	61.23	60.20	59.50	

Mean levels of Stage 4 as a percent of TST for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	14.96	13.62	16.11	14.08	14.69
Group 2	13.62	15.07	14.00	13.16	13.96
Group 3	10.11	12.91	12.24	12.21	11.87
Group 4	13.77	14.28	12.50	15.92	14.12
Mean	13.12	13.97	13.71	13.84	

APPENDIX 2

Mean levels of SWS (Stage 3 + 4) as a percent of TST for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	22.35	21.70	23.53	21.63	22.30
Group 2	21.92	24.33	23.22	24.38	23.46
Group 3	21.46	22.22	22.63	22.12	22.11
Group 4	22.10	22.91	22.12	23.21	22.59
Mean	21.96	22.79	22.88	22.84	

Mean Stage 2 + 3 + 4 levels (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	312.17	311.50	316.08	300.92	310.17
Group 2	311.25	294.00	284.74	301.58	297.89
Group 3	307.75	310.75	293.08	292.42	301.00
Group 4	292.08	311.92	310.75	284.50	299.81
Mean	305.81	307.04	301.16	294.86	

APPENDIX 2.

Mean SWS latency (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	10.33	13.08	10.92	11.83	11.54
Group 2	13.58	8.33	12.74	8.41	10.77
Group 3	12.17	9.75	8.83	9.75	10.13
Group 4	16.67	13.00	10.75	13.50	13.48
Mean	13.19	11.04	10.81	10.87	

Mean sleep onset latency (Mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	30.75	20.25	16.33	32.75	25.02
Group 2	19.33	22.42	26.09	16.92	21.19
Group 3	24.92	20.75	21.83	10.25	19.44
Group 4	35.50	22.00	18.17	24.09	24.94
Mean	27.63	21.36	20.61	21.00	

APPENDIX 2.

Mean time awake during sleep period time (SPT)(mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	7.83	9.67	6.75	4.25	7.13
Group 2	12.25	5.67	7.70	10.08	8.93
Group 3	2.83	13.83	1.00	5.25	5.73
Group 4	8.83	11.42	1.42	15.67	9.34
Mean	7.94	10.15	4.22	8.81	

Mean time awake during SPT as a percent of SPT for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	1.57	2.14	1.44	.91	1.52
Group 2	2.86	1.37	1.77	2.33	2.08
Group 3	.65	3.08	.20	1.17	1.28
Group 4	2.18	2.45	.32	3.71	2.17
Mean	1.82	2.26	.93	2.03	

APPENDIX 2.

Mean total time awake (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	34.83	24.58	18.42	37.83	28.92
Group 2	27.67	27.17	38.18	22.92	28.99
Group 3	24.25	30.58	18.42	11.17	21.11
Group 4	37.83	26.17	11.92	30.00	26.48
Mean	31.15	27.13	21.74	25.48	

Mean total time awake as a percent of time in bed (TiB) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	7.09	5.22	3.88	8.26	6.11
Group 2	5.85	5.82	8.16	5.04	6.22
Group 3	5.02	6.22	4.02	2.44	4.43
Group 4	7.91	5.58	2.61	6.64	5.69
Mean	6.47	5.71	4.67	5.60	

APPENDIX 2.

Mean number of awakenings for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	5.50	6.33	4.50	4.00	5.08
Group 2	5.33	4.83	6.74	5.67	5.64
Group 3	2.50	6.00	1.00	2.50	3.00
Group 4	3.50	5.17	2.33	3.83	3.71
Mean	4.21	5.58	3.64	4.00	

Mean level of Stage 1 (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	38.58	41.17	32.83	36.25	37.21
Group 2	40.75	36.42	44.84	43.17	41.30
Group 3	35.67	49.42	39.17	37.33	40.40
Group 4	41.92	41.50	40.50	44.58	42.13
Mean	39.23	42.13	39.34	40.33	

APPENDIX 2.

Mean Stage 1 levels as a percent of TST for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	8.38	8.94	7.25	8.33	8.23
Group 2	9.15	8.39	10.89	10.04	9.62
Group 3	8.02	11.15	8.69	8.33	9.05
Group 4	9.86	9.51	9.04	11.99	10.10
Mean	8.85	9.50	8.97	9.67	

Mean levels of Movement Time (MT)(mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	5.75	9.08	7.25	6.33	7.10
Group 2	5.33	5.67	3.73	5.58	5.08
Group 3	5.08	6.17	5.75	7.25	6.06
Group 4	5.33	3.92	5.58	4.00	4.71
Mean	5.37	6.21	5.58	5.79	

APPENDIX 2.

Mean levels of MT as a percent of TST for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	1.25	1.98	1.60	1.44	1.57
Group 2	1.19	1.25	.90	1.28	1.16
Group 3	1.15	1.38	1.28	1.60	1.35
Group 4	1.25	.90	1.29	.86	1.08
Mean	1.21	1.38	1.27	1.30	

Mean levels of time awake + MT + Stage 1 as a percent of SPT for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	9.90	11.70	9.18	10.62	10.35
Group 2	10.69	9.40	11.52	11.84	10.86
Group 3	8.62	14.40	9.18	9.93	10.53
Group 4	11.18	10.85	8.99	11.84	10.72
Mean	10.10	11.59	9.72	11.06	

APPENDIX 2.

Mean levels of time awake + MT + Stage 1 as a percent of TiB for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	16.49	15.62	12.33	17.30	15.44
Group 2	15.55	14.81	18.89	15.71	16.24
Group 3	13.60	17.83	13.53	12.12	14.27
Group 4	18.10	15.28	12.65	18.33	16.09
Mean	15.94	15.89	14.35	15.87	

Mean Time in Bed (TiB)(mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	491.50	479.75	468.17	472.17	477.90
Group 2	476.67	466.08	457.38	461.58	465.43
Group 3	457.42	472.42	453.83	451.17	458.71
Group 4	462.42	456.67	447.83	447.00	453.48
Mean	472.00	468.73	456.80	457.98	

APPENDIX 2.

Mean Sleep Period Time (SPT)(mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	453.67	459.17	451.83	427.67	448.09
Group 2	451.65	437.08	418.58	442.00	437.33
Group 3	430.58	451.67	432.08	440.92	438.81
Group 4	442.08	434.67	427.75	419.50	431.00
Mean	444.50	445.65	432.56	432.52	

Mean Total Sleep Time (TST)(mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	456.67	455.17	449.75	434.33	448.98
Group 2	449.00	438.92	419.20	438.67	436.45
Group 3	433.17	441.83	435.42	440.00	437.61
Group 4	424.58	430.50	435.92	417.00	427.00
Mean	440.86	441.61	435.07	432.50	

APPENDIX 2.

Mean levels of Stage REM (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	100.17	93.42	93.50	90.75	94.46
Group 2	91.67	98.33	86.18	88.33	91.13
Group 3	84.67	75.50	97.42	103.00	90.15
Group 4	85.25	73.17	79.08	83.92	80.36
Mean	90.44	85.11	89.05	91.50	

Mean levels of Stage REM as a percent of TST for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	22.04	20.42	20.84	21.06	21.09
Group 2	20.39	22.42	20.37	20.13	20.83
Group 3	19.48	16.90	22.46	23.53	20.59
Group 4	20.20	16.98	18.39	18.96	18.63
Mean	20.53	19.18	20.52	20.92	

APPENDIX 2.

Mean REM latencies (mins) for Groups on each exercise day.

	EXERCISE DAY				
	Baseline	E1	E3	E5	Mean
Group 1	102.08	100.75	101.92	88.20	98.24
Group 2	96.33	86.17	73.44	100.30	89.06
Group 3	79.08	97.42	125.83	99.20	100.38
Group 4	98.33	152.42	104.33	94.90	112.50
Mean	93.96	109.19	101.38	95.65	