A healthy body and mind are contingent on a balanced diet. Though the qualitative nature of a balanced diet may vary from individual to individual depending upon their age and personal requirements but recently this emphasis has been shifted to balancing of energy rather than only balancing of the dietary components.

The latest nutritional breakthroughs and researches on brain biochemistry suggest that not only what one eats is important but the time at which it is eaten, makes a tremendous difference. Thus, a diet should be designed to keep one alert, active and high on energy during the day while promoting sound sleep at night.

Therefore, breakfast should consist of low carbohydrate and high protein foods. The reason for increased protein requirement early in the morning is that after a night's sleep, blood sugar level drops and ironically, it takes protein, not sugar - to built them up again. Though refined sugars and carbohydrates
can compensate the lowered blood sugar levels but these dump too much sugar in the system too quickly. Consequently, the blood sugar levels shoot up immediately, thereby, resulting in the release of extra insulin from the pancreas which results in a sudden drop in the blood sugar levels. On the other hand, proteins are converted into sugars at a slower rate and the body requirements can be met for a longer time.

Another factor, which advocates high protein intake in the morning, is that proteins contain an amino acid called tyrosine, which is the precursor of three important neurotransmitters: dopamine (DA), norepinephrine (NE), and epinephrine (E) each of which is associated with wakefulness. Further, research has indicated that laboratory animals fed a single meal composed of 40% proteins had higher levels of brain catecholamines (Wurtman & Fernstorm, 1983). Thus, it appears that catecholamine containing brain neurons are normally under specific dietary control.
On the other hand, for promoting sound sleep at night, dinner should consist of high carbohydrates and low protein foods. The reason for recommending a high carbohydrates diet is that it help to increase the levels of tryptophan, which is the precursor of serotonin, a neurotransmitter that is involved in regulation of sleep. In fact, tryptophan is delivered to the blood by proteins. However, proteins result in the increase of about five other amino acids which compete with tryptophan for transportation from blood capillaries to brain. High carbohydrate intake results in increased insulin levels which reduces the blood levels of competing amino acids without disturbing tryptophan. Thus, the reduced competition results in increased tryptophan supply to the brain.

Further support for the fact that increase in the levels of neurotransmitters can influence cognitive functioning is available from investigations in which it has been undoubtedly established that learning also results in changes in the levels of a number of neurotransmitters and is, in turn, influenced by changes in these neurotrans-
mitter systems. The brain NE concentrations have been found to be sensitive to training procedures and correlated with later retention performance (Gold & van Buskirk, 1978). Training with an intense footshock, which results in good retention performance, causes a decrease of about 80% in the brain NE concentrations, when measured ten minutes after learning (Welsh & Gold, 1984). Further, decrease in NE levels to about 60% of controls occur after some amnestic treatments and is also correlated with poor retention on later test trials.

DA agonists injected into the hippocampus immediately after training have been found to improve retention performance whereas administration of DA antagonists impair retention (Grecksch & Matthies, 1982). Thus, changes in concentrations of neurotransmitters appear to have a window value for predicting later retention performance.

However, peripheral endogenous changes, which are the physiological response of the organism to environmental stressors, have also been found to have a mnemonic effect. It has been
observed that in a stressful situation, a number of hormones are secreted endogenously. Since, a learning situation is also a stressful situation, it may also be possible that these hormones may affect learning. The main hormones which are released in a stressful situation are adrenocorticotropic hormone (ACTH), €, vasopressin (VP) and corticosteroids. Also, the secretion of one hormone, in turn, affects the secretion of other hormones.

The memory modulatory effect of these hormones have been demonstrated by a number of investigators. Pre/post training peripheral administration of these hormones has a dose and time dependent effect on memory. An inverted "U" shaped relationship has been found between doses of €, ACTH and later retention performance, i.e. memory enhancement is seen with moderate doses and amnesia at lower or higher doses (Gold & van Buskirk, 1975, 76; Gold, van Buskirk & Haycock, 1977; Crine, 1982; Yadava & Muhar, 1985).

These findings indicate that there is an optimal level of circulating endogenous substances above/below which performance is poor. This view
gains support from the fact that the amnesia produced by post training administration of ACTH, E or VP can be counteracted by administration of either the same or other substance prior to testing (Izquierdo & Dias, 1983). Thus, it appears that the endogenous state present at the time of training and testing acts as an endogenous cue for retrieval.

From the above discussion, it is clear that various peripheral hormones play an important role in learning and memory. However, the exact mechanism through which these hormones affect the central nervous system (CNS) functioning underlying memory is not clear as these hormones do not readily cross the blood-brain barrier (B.B.B.) (Dunn & Gispen, 1977; Cornford, Brown, Crane & Oldendorf, 1978) rather they cause a transient change in the central neurotransmitters (Gold & van Buskirk, 1978). Further, the doses of E, ACTH and VP which are effective intraperitoneally (ip) when administered after training or prior to testing fail to produce any effect when given intracerebroventricularly (icv) (de Almedia, Kapozinski & Izquierdo, 1983).
Thus, it is possible that these hormones may act on the peripheral adrenergic system to enhance memory. This view gains support from studies in which peripheral injections of adrenergic receptor antagonists were found to attenuate the mnemoactive effect of several amnestic treatments while their central administration was ineffective (Gold & Sternberg 1978; Sternberg & Gold, 1980). However, it has been found that brethylium—a peripheral sympathetic noradrenergic blocker does not have any effect on acquisition or retention of a passive avoidance task nor does it attenuate the amnesia produced by frontal cortex stimulation (Sternberg, Gold and McGaugh, 1982).

Thus, it appears that the memory modulating effect of these hormones is not mediated directly by their action on the peripheral or central neurotransmitter systems. However, a major physiological action of these hormones is the release of hepatic glucose from the liver during the stressful situation. Further, unlike the stress related hormones, glucose is actively and readily transported into the brain (Lund-Anderson, 1979; Oldendorf, 1971;
Paradridge & Oldendorf, 1975). This ready access is important because the CNS stores very little glucose but relies entirely on blood glucose for energy. (Lowry, 1975). Thus, it is possible that glucose may serve as the interacting agent between peripheral hormones and central processes underlying memory (Gorbman, Dickhoff, Vigna, Clark & Ralph, 1983).

Glucose is the principal sugar in the blood and serves as the major metabolic fuel to the tissues. The minimum glucose requirement of the body during a fast is approximately 125 - 150 g/day while an additional approximately 30 - 40 g/day is required by the peripheral nerves, red and white blood cells. The blood receives glucose from two main sources. One of these is the diet, but it is available only for a few hours after ingestion of a meal. Liver is considered to be the second main source of glucose input. Large amounts of glucose input between the meal comes from the liver.

Although, the body tissues can utilize other glucose metabolic substrates, when direct supply of glucose is unavailable, but the brain
relies totally on the fresh and continuous supply of blood glucose and oxygen as it cannot store glucose. Glucose is also a precursor of the substrate for pyruvate-dehydrogenase in the brain, which is critical in the production of cellular energy. Thus, it appears that circulating glucose levels may regulate the efficacy of neural processes underlying central processes of information through activation of this or other enzymes.

Further, glucose is also a key precursor of acetyl-CoA in the CNS which is necessary for acetylcholine (ACh) synthesis and ACh synthesis is sensitive to relatively small changes in plasma glucose levels (Gibson & Blass, 1978). Treatments which affect central cholinergic systems also affect memory processing (Gold & Zornetzer, 1983) and deficits in cholinergic functions may contribute to age-related deficits in memory (Coyle, Price & Delong, 1983).

Since memory does have a substrate in the brain and the brain more than the muscles, requires glucose and it has to have it now—a steady second to second flow, it implies that low
blood sugar levels could result in mental problems. In fact, fatigue, nervousness, indecisiveness, poor concentration have also been linked to low blood sugar levels.

Experimental evidence also indicates that impairments in blood sugar regulation (hypo or hyper glycemia) results in poor memory (Stone, Wenk, Olton & Gold, 1990). Furthermore, in human beings also, low blood sugar levels have been found to be correlated with poor memory (Holmes, Hayford, Gonzalez & Weydert, 1983; Holmes, Koepke & Thompson, 1986).

All these symptoms are generally associated with aging. Furthermore, aging is also accompanied by elevated blood sugar levels and decreased glucose utilization (Fink, Kolterman, Griffin & Olefsky, 1983; Heikinheimo, 1972; Kent, 1976; Khul, Metter, Riege & Hawkins, 1984; Levin, 1982; Lipson, 1986; O'Sullivan, 1974; Tobin, 1984).

In a number of investigations it has been found that primary and immediate memory for visual material declines with age (Halvand, Linn, Hunt & Goodwin, 1983; Poon, 1985). Diabetics have also been found
to be poor on neurophysiological measures, including verbal I.Q., visuospatial tasks, attention and reaction time (Holmes, 1986; Perlmutter, Hakami, Hodgsc. Harrington, Ginsberg, Katz, Singer & Nathan, 1984; Ryan, Vega, Drash & Longstreet, 1984).

Thus, glucose metabolism appears to play an important role in regulation of learning and memory. Its mnemoactive effect has been found to be analogues to that of epinephrine (i.e. dose, time and state dependent). However, research in this area has been limited to the animal level, that is, the albino rats. Its verification at the human level is rather limited. Since, glucose does appear to have a mnemoactive effect and the blood glucose levels are influenced by diet, the present study has been carried out to study the effect of glucose and dietary components in memory at the human level.

With this background we may now pass on the next chapter dealing with the review of pertinent literature.