INTRODUCTION

Headache is a common symptom. Though frequently a trivial disorder, it is also at times a symptom of grave significance (Walton, 1986). Headache is one of the most common complaints in medical practice. In general, it is mild or occasional, but when severe or frequent, it constitutes a significant burden for both the individual and society resulting in loss of productivity, limitation of activity and deterioration of the quality of life (Deleu et al., 2001).

Headache is the seventh leading presenting complaint for ambulatory care encounters in the United States, which accounts for 18.3 million, or 43.2 per 1000, outpatient visits per year (Ries, 1984). About seventy percent of the patients visiting a doctor complain of headache as one of their problems. Nearly ninety percent of people have headache at least once in a year (Ramaiah, 1996). According to Rasmussen et al. (1991), only 4% of the total population had never had a headache. Nearly everyone experiences headaches at some time or the other, but mostly these are mild, transient and do not interfere with normal every day activities and only occasionally (less than 10%) require a medical advice (Clifford Rose, 1988).

Headache is a pain in the head which can range from a slight ache that makes life a bit dreary but does not seriously affect concentration, to a pain so disabling that the thought process is severely disturbed and the sufferer is completely prostrate (Trickett, 2000). It is convenient to divide headaches into primary (chronic) and secondary categories. The primary (chronic) headache disorders are those, in which head pain represents the clinical expression of physiological reactivity in the absence of persistent identifiable organic pathology. Muscle contraction headache (tension headache), migraine, and cluster headache are...
examples. Secondary headaches are those in which head pain occurs as a consequence of identifiable pathology. Toxic and metabolic, structural, infectious, traumatic, and vascular diseases are examples (Saper, 1983). According to Ramaiah (1996), the common type of headaches include tension, migraine, cluster, carcinogenic, after injury, vascular, and effort induced. In rare cases, the headaches may be due to serious diseases such as bleeding in the space around the brain, brain fever, increased pressure on the brain, severe hypertension, and inflammation of some arteries of the brain. Pearce (1994) stated that over 95% patients seen in a general practitioner’s surgery or hospital clinic, however, have tension headache, migraine, or atypical headache without a structural lesion.

The human body operates with a rigorous logic; when it flares up and produces a headache, it is giving warning that its control mechanisms are or have been overloaded. It cannot tolerate the stress it has been subjected to. The enforced idleness of headache is a way of restoring equilibrium to the system; the pain is a way of alerting the conscious mind that mistake in input or direction has been made, for which it might be responsible (Gould, 1973). A headache is the head saying that you are subjecting it to conditions it cannot tolerate (Trickett, 2000).

Individuals with recurrent headache disorders report more psychological symptoms than individuals who do not suffer from recurrent headache disorders (Crisp et al., 1977; Ziegler et al., 1978; Andrasik et al., 1982, 1988; Merikangas et al., 1990; Mitsikostas and Thomas 1999; Zwart et al., 2003; Harter et al., 2003; Karakurum et al., 2004; Bag et al., 2005).

Migraine is one of the most painful and frustrating benign disorders known to medicine. It is considered medically "benign" because it is almost never life threatening or direct cause of some one’s death. But to a person who experiences migraine, it is pernicious, evil, soul
stealing, life robbing, expensive, exhausting, and humiliating (Constantine and Scott, 1994).

Migraine has been a problem and challenge for patients and physicians for so many centuries and yet despite voluminous publications, we still lack a satisfactory definition of migraine. The problem is because attacks of migraine vary markedly among individuals and even in a given person, particular symptoms may occur in one attack but not in another. Over the years various attempts have been made, both by individuals and groups to establish definition of migraine with reasonably high consensus.

According to Lance (1969), “The term migraine is of French origin and comes from the Greek word ‘hemicrania’ like the old English word ‘megrim’. The classical concept of migraine is that of paroxysmal disturbances of cerebral function associated with unilateral headaches and vomiting. The definition of migraine has widened in recent years to include bilateral headaches.”

Sacks, author of “Migraine, Evolution of Common Disorders” views migraine as a safety valve by which repressed feelings are expressed. He believes that attack serves for recuperation, self-punishment or retreat from an unpleasant situation (as quoted by Gould, 1973).

According to Friedman of Montefiore Clinic, “Migraine is psychophysiological and is triggered by body reaction to emotional stress” (as quoted by Gould, 1973).

Saper (1978) views migraine as “a genetically determined physiologic predisposition that is influenced by a wide variety of emotional, biological, and constitutional factors, acting independently or in conjunction with each other and additional unidentified elements to precipitate an attack.”

Blau (1983) opines that “Migraine headache is an episodic headache accompanied by visual or gastrointestinal disturbances, or both; the attacks last for four hours with total freedom between episodes.
Before the headache, visual symptoms occur as an aura and during it photophobia may be present, alimentary symptoms consist of nausea and vomiting. If there are no visual but only gastrointestinal disturbances then vomiting must feature in some attacks.

Saper (1983) defines migraine as “a familial paroxysmal disorder characterized by recurring attacks of wide ranging symptoms, most notable headache. The headache varies in intensity, associated symptomatology, frequency, and duration. Gastrointestinal distress and visual disturbances, though not necessarily present, are most common associated complaints.”

According to Walton (1986), “Migraine is a paroxysmal disorder characterized in its fully developed form by visual/or sensory phenomena in an aura, associated with or followed by unilateral headache and vomiting. While this definition is satisfactory for classical migraine, there are many patients who never experience an aura and in whom the headache is always bilateral; the single most characteristic and constant feature is that migraine is a paroxysmal disorder, i.e., the headache occurs in attacks, separated by intervals of freedom.”

According to International Headache Society (1988), “Migraine is a chronic condition characterized by 4 to 72 hours recurrent attacks of throbbing head pain that may be accompanied by nausea, vomiting, and sensitivity to light and noise.”

Pryse-Phillips et al. (1992) define migraine, “as a chronic condition characterized by episodic attacks of headache commonly associated with various combinations of nausea, vomiting, photophobia, phonophobia, and aura.”

According to The Concise Oxford Dictionary, “Migraine is recurrent throbbing headache that usually affects one side of head, often accompanied by nausea and disturbances of vision” (as quoted by Walji and Kingston, 1994).
Lance (1993) views migraine, “as essentially an episodic headache, usually accompanied by nausea and photophobia, which may be preceded by focal neurological symptoms (aura). The aura may be experienced without any ensuing headache; such attacks have in past been called migraine equivalents.”

The Webster Dictionary (2000) defines a migraine “as a condition that is marked by recurrent severe headaches often with nausea and vomiting.”

So, it can be concluded that migraine is a paroxysmal disorder characterized by severe headache (usually unilateral but sometimes bilateral), nausea, photophobia, and phonophobia.

**PREVALENCE**

Migraine is a common neurological disorder. The 1 year prevalence of migraine in United States of America is 17.2% in females and 6.0% in males. Prevalence is highest between the ages of 30 and 44 (Lipton et al., 2002). It affects 11% of the adult population (Scher et al., 1999). A projection to the US population suggests that 8.7 million females and 2.6 million males suffer from migraine headache with moderate to severe disability. Of these, 3.4 million females and 1.1 million males experience one or more attacks per month. 17.6% of females and 6.7% of males were found to have one or more migraine headaches per year (Stewart et al., 1992). In a study of sample aged between 25-64 years with clinically confirmed migraine, meeting the IHS diagnostic criteria, the age-adjusted annual incidence of migraine was estimated to be 370 per 100,000 person-years, 580 per 100,000 person-years in females and 160 per 100,000 person-years in males (Rasmussen, 1995a). Waters and Connor (1975) calculated much higher prevalence of migraine. In three separate
epidemiological surveys, the prevalence in the preceding year was found to be between 23% and 29% in women and 15% and 20% in men.

According to American Migraine study, a total of 71% of men and 59% of women determined to have migraine from self-reported symptom data, never received a diagnoses from the physician (Stewart and Lipton, 1993). Results of a survey indicated that the majority of people with migraine in the United States do not report that they have been diagnosed by a physician (Lipton et al., 1992).

Over the years number of studies have been conducted to know the prevalence of migraine with aura and without aura. Based on a study of 1000 people using the International Headache Society's Criteria, the over all life time prevalence of classic migraine was 5%, with a female to male ratio of 2:1. The over all life time prevalence of common migraine was 8% with a female to male ratio of 7:1 (Rasmussen and Olesen, 1992).

Rasmussen et al. (1992) conducted a cross sectional epidemiological survey of general population. They found that in the previous year, 6% had migraine without aura, 4% had migraine with aura. In women migraine without aura was twice as prevalent as migraine with aura, whereas in men opposite trend emerged.

Russell et al. (1996) stated that life time prevalence of migraine without aura was 14.7% with M: F ratio of 1:2.2 that of migraine with aura was 7.9% with an M: F ratio of 1:1.5.

Terwindt et al. (1997) found that among migraineurs, 71.3% had migraine without aura and 18.4% had only migraine with aura. A further 10.3% had more than one type of migraine: 69.4% had migraine with or without aura, 16.3% had migraine without aura and aura without migraine, 4.1% had migraine with aura and aura without migraine and 10.2% had all three diagnoses.

Number of studies have been conducted to know the prevalence of migraine in different age groups. Abu-Arefeh and Russell (1994) found that the estimated prevalence rate of migraine in school children was
10.6%. The estimated prevalence rates of migraine without aura and migraine with aura were 7.8% and 2.8% respectively.

Rondon et al. (2001) conducted a study to determine the prevalence of migraine among primary and secondary school students in Merida, Venezuela. Among the students, 84.4% reported having headaches and 16.8% reported experiencing migraines.

Bille (1997) conducted a 40-year follow-up of school children with migraine. The prevalence of migraine was 1.4% at 7 years of age and 5.3% at 15 years of age. From the age of 11 there was a gradual increase of migraine headache and predominance among girls. A subgroup of 73 children with pronounced migraine and average onset of 6 years were followed during a period of 40 years. The results showed that 23% of the children were migraine-free before the age of 25, boys significantly more often than girls.

Wang et al. (1997) investigated the prevalence of headaches in a Chinese elderly population. One-year prevalence of migraine was 3.0% and tension type headache was 35%.

Prevalence of migraine varies across cultures and continents. In the United States, migraine prevalence is highest in Caucasians, followed by African Americans and Asian Americans. In women, migraine prevalence was significantly higher in Caucasians (20.4%) than in African Americans (16.2%) or Asian Americans (9.2%). A similar pattern was observed among men (8.6%, 7.2%, 4.2%) (Stewart et al., 1996).

In America, 17.6% of females and 5.7% of males were found to have one or more migraine headaches per year (Stewart et al., 1992). An epidemiologic surgery on headache was carried out in the Republic of San Marino, which is smallest independent state in the world. Frequency of migraine in the previous year was 9.3% for men and 18% for women (D’Alessandro et al., 1988). Life time prevalence of migraine in Canada was 23% in females and 10% in males (Pryse-Phillips et al., 1992). Life time prevalence of migraine in Germany was 22% for males 32% for
females (Gobel et al., 1994). According to Cull et al. (1992), life time prevalence of migraine in UK was 4.3% for males and 11.0% for females. One-year prevalence of migraine in Ethiopia was 1.7% for males and 4.2% for females (Tekle-Haimanot et al., 1995). According to Wang et al. (1997), one-year prevalence of migraine in Hong Kong was 0.6% for males and 1.5% for females. Life time prevalence in Saudi Arabia is 2.6% (Abduljabbar et al., 1996). According to Alders et al. (1996), one-year prevalence of migraine in Malaysians was 6.7% for men and 11.3% for women. One-year prevalence of migraine in Japan was 3.6% for males and 12.9% for females (Sakai and Igarashi, 1997). Wang (2003) stated that the prevalence of migraine has been quite consistent in Asia, ranging from 8.4% to 12.7%. The consistency of the prevalence of migraine headache among the Asian countries is interesting in a region where the cultural background and development are so diverse. These IHS migraine surveys show that migraine is a significant disease in Asia and that its prevalence rate is close to but in the low range of those reported in Western countries.

Though there are no estimates of the prevalence of migraine in India, the figures would be equal to or more than the global average, say experts. “High temperatures and light levels for more than eight months of the year, and the heavy noise pollution, the Indian habit of not having breakfast, frequent fasting, and eating rich, spicy and fermented food are common triggers” says Ravishankar (as quoted by Sharma, 2003).

These data suggest that although cultural and environmental differences may contribute to international differences in migraine prevalence, race related differences in genetic vulnerability to migraine may also be important (Breslau and Rasmussen, 2001). It can be concluded that prevalence of migraine varies with age, gender, race, socioeconomic status, and geographic location.
HISTORY

Headache, an ancient and much studied complaint, has a rich historical literature that illustrates the evolution of our knowledge from the magical to the molecular level (Rapoport and Edmeads, 2000).

Disease played a punitive part in the early religious systems. Pain was a punishment, a visitation of demons, and migraine won especially vivid example of divine retribution. Its early victims were believed to have been entered by demons, which were cutting a vicious swath through the body, causing the multiplicity of symptoms. The ancient Arab tribes even had a name for migraine demon. They called it Palga, and had special prayers for its exorcism (Gould, 1973). Five thousand years ago, Mesopotamian physicians viewed headache as a disease entity rather than a symptom and attributed it to an evil spirit named ‘Tiu’ (Rapoport and Edmeads, 2000). An Egyptian papyrus of 2500 BCE describes bandaging a clay effigy of a sacred crocodile to the head of the sufferer and praying (Edmeads, 1990). When it failed, recourse consisted of releasing the evil spirit through a hole gauged in the skull by a trephine. Trephination is a remarkably ancient procedure. Neolithic skulls dating from 7000 BCE show trepan holes (Gould, 1973; Rapoport and Edmeads, 2000). It is known that trephination was performed as a treatment for migraine as late as the mid 17th century (Willis, 1685).

Hippocrates, the founder of art of medicine, once wondered if there wasn’t something supernatural which caused disease. His speculation represented a giant step in the progress of science (Gould, 1973). To Hippocrates (circa 400 BCE), neurologists owe a double debt. Not only did he recognize the syndrome called migraine, but he ascribed it to an imbalance of natural forces within the body, rather than to influence of supernatural being (Rapoport and Edmeads, 2000). Hippocrates was the first one to describe several well known features of migraine, viz., visual aura, and the onset of headache after the aura, the headache becoming
generalized and its relief by vomiting (Rose, 1995). It was in the Graeco-Roman period that more knowledge was added, and the first accurate description of migraine has usually been ascribed to Arataeus of Cappadocia (AD 30-90). He reported three different types of headaches, viz. cephalagia, which was mild infrequent, lasted several days, and may well have been tension (muscle contraction) headache, cephalaea, which, lasted longer, was more severe, difficult to treat and probably due to a structural cause, and heterocrania which was one sided headache with blackness before the eyes, nausea, vomiting, photophobia and osmophobia, is migraine (Rose, 1995). Aretaeus of Cappadocia was the first one to note that photophobia and phonophobia of migraine (Rose, 1995).

In the second century AD, Galen wrote of “a painful disorder affecting approximately one half of the head either the right or left side, and which extends along the length of the longitudinal suture (Critchley, 1967). Galen refined Aretaeus’s observation by changing the name of the disease to hemicrania, or one sided headache (Friedman and Merritt, 1959; Critchley, 1967; Gould, 1973; Saper, 1983; Walton, 1986; Walji and Kingston, 1994). The Greek word, hemicrania was changed by the Romans to the Latin, hemicranium, and then corrupted into low Latin as hemicrania, then emigranea, migranea and migrana. In English, it appeared as mygraine (1398), myegrym (1460), migrien (1579), and megrim (1713), the last mentioned being used by Liveing (1873). Migraine appeared in English (from French) in 1777 and is now the standard term (Rose, 1995).

Galen believed that this headache and its accompaniments were caused by the ascent of vapor from the liver to the head, which explained the conjoining of vomiting and headache (Rapoport and Edmeads, 2000). Galen referred to migraine as a disease of the “black bile” and claimed it was basically a disorder of digestive tract (Gould, 1973). In the 17th century Thomas Willis combined keen clinical observation and
meticulous anatomic dissection to revolutionize thinking about megrim (Rapoport and Edmeads, 2000). Willis left the most famous case history of a migraine sufferer ever recorded. It described his attempts in 1674 to cure Lady Anne Conway, a woman who had been victimized by shattering attacks for twenty years (Gould, 1973).

In 1873, an important book was published by English physician, Edward Liveing. Like Wills, Liveing was a master diagnostician. He was the first physician to notice that women were more vulnerable to the disease than men. He also linked changes in personality to the incipient stage of the attacks (Gould, 1973).

The work of 19th century psychiatrists, which culminated with the theories of Sigmund Freud, provided still another causative factor. Freud established, in his theory of personality development a very close connection between the emotional history of the patient and certain physiological disturbances. He himself suffered from migraine and had no qualms about diagnosing it as a psychogenic ailment, connected in some way with unresolved sexual conflict (Gould, 1973). Wolff and his associates had elucidated the pathophysiology i.e., the vascular origin of migraine (Friedman and Merritt, 1959).

THEORIES OF MIGRAINE

Over the years number of theories have been put forward by neurologists to explain the cause of migraine. They can be broadly classified into four categories: vascular theory, neural theory, neurovascular theory (a combination of neural and vascular) and neurochemical transmission / depletion theory.

Vascular Theory of Migraine: Clinicians who support the vascular theory believe that a migraine headache results directly from the expansion and contraction of blood vessels, both inside and outside the brain (Kandel and Sudderth, 1998). An initial vasoconstriction of certain blood vessels supplying the cerebrum and vessels in the retina may produce visual, motor, sensory and possibly other pre-headache phenomena prior to the onset of the headache. This non-painful prodromal period is followed by a painful phase during which dilation and distention of cranial arteries occur (Friedman and Merritt, 1959). Graham and Wolff (1938) demonstrated an increase in pulse amplitude of scalp arteries measured at the onset of a migraine that paralleled the severity of headache. Dilation alone however, could not explain the pain of migraine, since simple dilation as when blushing or following exercise, during periods of warming is not painful (Saper, 1983). A factor that may be important to the production of pain is the appearance during headache of a substance, “neurokinin”, in that extravascular space at the site of pain (Chapman et al., 1960). Neurokinin, a polypeptide substance produces pain and is responsible for the development of a “sterile inflammation” around the blood vessels (Ostfeld et al., 1957; Chapman et al., 1960). Additional evidence and anecdotal references do support the “pulsing temples”, referring to the blood vessels over the forehead. These do actually appear to pulsate in a number of patients during a migraine episode (Kandel and Sudderth, 1998).
**Neural Theory:** In 1873, Edward Liveing theorized that migraine was caused by nerve storms, or discharge originating in the brain (Wilkinson and MacGregor, 1999). Supporters of this theory believe that migraine is actually a dysfunction of the nervous system and an unstable threshold in the brain. It is believed that migraine results from an irregular or abnormal nervous system outflow, a burst of electric energy, which can kindle or trigger a variety area in the brain. This subsequently produces a variety of symptoms, sort of like a microscopic lightning strike inside the brain. As a result, many diverse symptoms may occur, such as severe headache associated with nausea and vomiting (Kandel and Sudderth, 1998).

**Neuro Vascular Theory:** Supporters of this theory take a combination approach, believing that during attacks, migraineurs have both abnormal and unstable blood vessels combined with nervous system irritability. It appears that much of the skull or facial pain that patients experience during migraine is carried along the distribution of the trigeminovascular pathways (the trigeminal nerve is located near the cheek bone). A possible defect in the chemical discharge along this pathway appears to be what causes the problem (Kandel and Sudderth, 1998).

**Neuro Chemical Transmission /Depletion Theory:** Serotonin, 5 - Hydroxytryptamine is one of several biogenic amines (Saper, 1983). Serotonin is one of the many chemical neurotransmitters involved in intercellular communication (Kandel and Sudderth, 1998). Research has strongly implicated the role of serotonin in migraine headaches. Measurable changes in the concentration of this chemical occur during a migraine attack as it is released from its storage sites within the body (Wilkinson and MacGregor, 1999). Researchers have recorded decreased blood level of serotonin during migraine episodes, and urine concentrations of serotonin breakdown products seem to increase. It is clear that there are serotonin changes in the body during a migraine headache (Kandel and Sudderth, 1998).
TYPES OF MIGRAINE

The two major types of migraine are migraine without aura and migraine with aura. The latter is further subdivided into migraine with typical aura, migraine with prolonged aura, familial hemiplegic migraine, basilar migraine, migraine aura without headache, and migraine with acute onset aura.

1 MIGRAINE WITHOUT AURA: It is an idiopathic, recurring headache disorder manifesting in attacks lasting 4-72 hours. Typical characteristics of headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity, and association with nausea, photo-and phonophobia (IHS, 1988). It is synonymous with common migraine and refers to paroxysmal headaches without the aura (Pearce, 1994). Common migraine is not preceded by a well defined aura, though prodromal symptoms of several days duration may occur prior to an attack, such as fluid retention, or mood alteration including lethargy, fatigue, irritability, and exhilaration (Atkinson and Appenzeller, 1984).

2 MIGRAINE WITH AURA: It is an idiopathic, recurring disorder manifesting with attacks of neurological symptoms unequivocally localizable to cerebral cortex or brain stem, usually gradually developed over 5-20 minutes and usually lasting less than 60 minutes. Headache, nausea and/or photophobia usually follow neurological aura symptoms directly or after a free interval of less than an hour. The headache usually lasts 4-72 hours, but may be completely absent (IHS, 1988). It is synonymous with classic or classical migraine (IHS, 1988). Clifford Rose (1988) states that classical migraine consists of a warning called the aura, 10-60 minutes before the headache, and is usually visual, characteristically a homonymous, spreading, scintillating scotoma (fortification spectra, teichopsia), but may be sensory, either pins or needles (positive phenomena) or numbness (negative), in the hand and
epsilon lateral cheek, but sometimes in the tongue and circumorally. The least common aura is motor weakness except in the rare hemiplegic migraine.

a) Family Hemiplegic Migraine: Hemiplegic migraine is a rare type of migraine, characterized by weakness of the limbs down one side of the body, which may last for several days even after the headache has gone (Walji and Kingston, 1994). This form of migraine may be inherited, usually as a dominant characteristic, when it is known as familial hemiplegic migraine (Whitty, 1953; Bradshaw and Parsons, 1965). Frequently such cases have a strong family history of similar disorder, and headaches do not seem to occur apart from these attacks (Jensen et al., 1981; O’Hare et al., 1981). IHS (1988) described familial hemiplegic migraine as migraine with aura including hemiparesis and where at least one first degree relative has identical attacks.

b) Basilar Migraine: It is a migraine with aura symptoms clearly originating from the brain stem or from both occipital lobes. To be diagnosed as basilar migraine, the patient must have two or more aura symptoms of the following types: visual symptoms in both the temporal and nasal fields of both eyes, dysarthria, vertigo, tinnitus, decreased hearing, double vision, ataxia, bilateral paresthesias, bilateral pareses, decreased level of consciousness. Basilar attacks are mostly seen in young adults (IHS, 1988). Previously used term for basilar migraine was basilar artery migraine. Basilar artery migraine was the name that Bickerstaff (1961) gave to that form of classical migraine where the focal neurological features, e.g., vertigo, ataxia, diplopia, could be localized to the distribution of basilar artery. Basilar artery migraine may be associated with a feeling of faintness and according to Lance and Anthony (1966), 7% have loss of consciousness. According to IHS (1988), originally the term basilar artery migraine was used, but since spasm of the basilar artery may not be the mechanism of the attacks, the term basilar migraine should be preferred.
c) **Migraine With Typical Aura:** It is migraine with an aura consisting of homonymous visual disturbances, hemisensory symptoms, hemiparesis or dysphasia or combination thereof, gradual development, duration under one hour and complete reversibility characterize the aura which is associated with headache (IHS, 1988).

d) **Migraine With Prolonged Aura:** It is migraine with one or more aura symptoms lasting more than 60 minutes and less than a week. Rare patients have only this form. The majority who experience prolonged aura have it rarely and intermingled with much more frequent attacks of typical aura (IHS, 1988).

e) **Migraine Aura Without Headache:** It is migraine aura unaccompanied by headache. It is common for migraine with aura that headache occasionally is absent. As patients get older, headache may disappear completely even if aura continues. It is less common to have always suffered exclusively from migraine aura without headache (IHS, 1988).

f) **Migraine With Acute Onset Aura:** Migraine with aura develops fully in less than 5 minutes (IHS, 1988)

**OPHTHALMOPLEGIC MIGRAINE:** The recurrence of double vision with migraine, associated with signs of paresis of the extraocular muscles, has been termed ophthalmoplegic migraine (Walsch and O’ Doherty, 1960; Bickerstaff, 1964). There are repeated attacks of headache associated with paresis of one or more ocular cranial nerves in the absence of demonstrable intracranial lesion. Patients must have at least 2 attacks of headache overlapping with paresis of one or more of cranial nerves III, IV and V (IHS, 1988).

**RETNIAL MIGRAINE:** There are repeated attacks of monocular scotoma or blindness lasting less than an hour and associated with headache. To be diagnosed as having retinal migraine, the patient must have at least 2
attacks with fully reversible monocular scotoma or blindness lasting less than 60 minutes and confirmed by examination during attack or (after proper instruction) by patient’s drawing of monocular field defect during an attack. Headache must follow usual symptoms with a free interval of less than 60 minutes, but may precede them (IHS, 1988).

CHILDHOOD PERIODIC SYNDROMES THAT MAY BE PRECURSORS TO OR ASSOCIATED WITH MIGRAINE (previously used term was migraine equivalents).

i. **Benign Paroxysmal Vertigo of Childhood:** This probable heterogeneous disorder is characterized by brief attacks of vertigo in otherwise healthy children (IHS, 1988).

ii. **Alternating Hemiplegia of Childhood:** Infantile attacks of hemiplegia involve each side alternately. It is associated with other paroxysmal phenomena and mental impairment (IHS, 1988).

COMPLICATIONS OF MIGRAINE

i. **Status migrainosus:** Is attack of migraine with headache phase lasting more than 72 hours despite treatment. Headache free interval of less than 4 hours (sleep not included) may occur (IHS, 1988).

ii. **Migrainous Infarction:** Previously used term was complicated migraine. There is one or more migrainuos aura symptoms not fully reversible within 7 days/and or, associated with neuroimaging confirmation of ischemic infarction (IHS, 1988).

MIGRAINOUS DISORDER NOT FULFILLING ABOVE CRITERIA

Headache attacks which are believed to be a form of migraine, but which do not quite meet the operational diagnostic criteria for any of the forms of migraine (IHS, 1988).
SYMPTOMS / PHASES / STAGES

The migraine attack consists of four phases: the prodromal state, the aura, the headache, and recovery. Many other symptoms like nausea, vomiting, photophobia, and phonophobia are usually associated with headaches.

PRODROME: Sometimes migraine sufferer gets a kind of advance warning of an impending migraine, a sort of “yellow light” alert, which is called prodrome (Kandel and Sudderth, 1998). The term prodrome has roots in Greek word prodromos which means ‘coming before’ (Trickett, 2000). According to Waelkens (1985), premonitory symptoms can be grouped into:

1. Autonomic disturbances affecting the intestinal tract, fluid balance, anxiety, yawning and sleepiness as well as vasomotor symptoms.
2. Altered behavior provoking intro- or extraversion, hypo- or hyperactivity (physically or mentally) as well as mood changes.
3. In addition, muscle pains can precede the onset of headache particularly neck tightness.

Saper (1983) reported that mood disturbances, appetite change with specific food craving, or changes in thirst occurring the evening before the attack are common in migraine sufferers. Drummond and Lance (1984) interviewed 530 patients with recurrent headache, ranging from typical migraine to tension type headache, out of these, 160 reported premonitory symptoms. Mood swings such as irritability or depression were pointed out by 86 patients and a feeling of elation by 54. Of the 47 who mentioned hunger, 34 craved for sweet food such as cake, biscuits, and chocolate (which was specifically mentioned by 12 patients). Drowsiness and yawning were symptoms in 38 patients and thirst in 15.
Incidence of premonitory symptoms varies. Blau (1980) observed these symptoms in 1/3 of his 50 patients, Isler (1986) noticed an incidence of 65% and Ramaiah (1996) stated that nearly 50% of people who suffer from migraine complain of these disturbances. Duration of premonitory symptoms ranges from 1-24 hr. (an average of 10 hr.) and elapses before the neurological aura in those with classical migraine or before the onset of headache in common migraine (Blau, 1980). In a recent study, Kelman (2004b) found that the most common symptoms were tiredness, mood change, and gastrointestinal symptoms; all three of these symptoms were present together in 17% of the patients with prodrome. The duration of prodrome was less than 1 hour in 45.1%, 1-2 hours in 13.6%, 2-4 hours in 15.0%, 4-12 hours in 13.1%, and greater than 12 hours in 13.2%. Most people do not at first recognize the prodromal state because the symptoms are indefinite. Sometimes these symptoms are recognized by other members of family and only later by the sufferer (Wilkinson and Mac Gregor, 1999).

**AURA:** Migraine sufferers may also experience a range of symptoms, classified under an experience known as the “aura”. The aura is a brief episode of symptoms that are related to focal area of dysfunction in the brain (Kandel and Sudderth, 1998). The neurological disturbances occurring as part of the aura of classic migraine usually last only 15 to 30 minutes and are seen in approximately 15 to 20% of the patients with migraine (Saper, 1983). Sometimes aura may last 10 to 60 minutes (Clifford Rose, 1988; Wilkinson and Mac Gregor, 1999). Most commonly, these neurological phenomena precede the headache usually by a period of 30 minutes or less and fade with onset of head pain, but they may persist into the headache period, sometimes for a matter of hours (Zeigler, 1991). Russell and Olesen (1996) in an investigation found that headache followed the aura in 92.6% of the cases, occurred simultaneously in 4.7% of the cases and aura followed headache in 2.7%
of the cases. Jensen et al. (1986) did a systematic prospective recording of aura symptoms of 50 patients. They found that visual aura was present in 94% of the patients; somato-sensory aura symptoms in 40% of the patients; motor disturbances in 18% of the patients and speech difficulties in 20% of the patients. Russell and Olesen (1996) in an investigation found that in migraine aura patients, visual aura was the most frequent symptom (99%), followed by sensory (31%), aphasic (18%), and motor (6%) aura. Only visual aura frequently occurred in isolation whereas sensory, motor, and aphasic auras were nearly always experienced in association with visual aura.

**Visual aura:** Visual aura is the most common type of aura. A remarkable variety of visual disturbances may be experienced during the aura (Wilkinson and MacGregor, 1999). Quieroz et al. (1996) studied visual aura among 100 patients suffering from migraine with aura. They stated that the most common aura were small bright dots (42%), flashes of light (39%), blind spots (32%), and foggy vision (27%). A fortification spectra was reported by only 20% of the patients. Although most (65%) patients had a combination of phenomena, the majority (72%) had only one uniform constellation of manifestations.

Fortification spectra represents glittering zigzag patterns experienced by many migraine patients, and were so named because they were reminiscent of the fortification constructed around embattled sites (Saper, 1983). According to Schiller (1975), the term fortification was first used by Fothergill in his 1778 account of 'sick headache'. He noted that during a migraine aura objects appear surrounded with luminous angles like those of fortifications. Airy (1870) coined the term teichiopsia (a translation of seeing fortification). Lashley (1941) suffered from visual auras without headache, and mapped his auras very accurately.

‘Photopsia’ which consists of circles, triangles, squares or other geometric patterns, usually white or multicolored are seen by
migraineurs (Kandel and Sudderth, 1998). About 25% of migraine aura patients report flashes of color and non color images (Dalessio, 1980). Migraine sufferer may complain of bright stars in front of the eyes, sometimes with one star brighter than the rest, starting from lower corner of the visual field and passing rapidly across it (Wilkinson and MacGregor, 1999).

The Alice in wonderland phenomenon refers to the bizarre alterations in shape, color, size, and body image that may occur during some migraine attacks (Saper, 1983). Lewis Carroll suffered from migraine and it has been suggested that some of the inspiration for illusion of vision and body image in ‘Alice in Wonderland’ may be origin in migraine aura (Lance, 1969; Gould, 1973).

Sometimes during an aura there is some accompanying impairment of vision. This may be a central scotoma or a homonymous visual field defect that can progress to a dense heminaopsia. Rarely, temporary cortical blindness or impairment of vision in one eye due to retinal artery dysfunction can occur (Pearce, 1968; Carroll, 1971). Other eye signs include half vision in one eye or both eyes, blurring of vision, when everything in sight generally appears completely out of focus, or even complete blindness for varying length of time (Leyton, 1955). The presence of visual auras has been reported in a woman with both eyes removed (Peatfield and Rose, 1981).

**Olfactory Aura:** Occasionally hallucinatory odors may precede headache (Ziegler, 1991; Kandel and Sudderth, 1998). Crosley and Dhamoon (1983) reported a case of child and her mother who had olfactory hallucinations preceding their headache. Child’s headache was preceded by a smell of gas, burning cookies or wood chips. Mother’s headache was usually preceded by smell of the inside of a pencil sharpener. Fuller and Guiloff (1987) reported the cases of three patients who had olfactory hallucination related to their migraine. They described their
hallucination as repulsive smell, unusual unidentifiable smell, peanut butter smell or grand father's cigar smell.

**Auditory Aura:** Other hallucinatory experiences among migraine sufferers are auditory misperceptions such as ringing or crackling noise in the ears (Kandel and Sudderth, 1998).

**Motor and Sensory Aura:** Unilateral or bilateral weakness and sensory deficits can accompany migraine attacks (Saper, 1983). Unilateral parathesis in association with aphasia and sometimes weakness may occur in an estimated 4% of the patients (Dalsgaard-Nielsen, 1948). Several patients have encountered recurring events of transient headaches and aphasia and accompanying right-sided weakness (Saper, 1983). In some patients there may be parenthesis or weakness of the face or extremities on the side opposite to the headache. In addition, there may be aphasia, vertigo or confusion (Friedman and Merritt, 1959). Perioral and tongue tingling with unilateral or bilateral hand symptoms are also seen among migraine sufferers (Saper, 1983). Unilateral paresthesia usually in one hand, arm and side of the face is the most frequent among migraine sufferers. There is characteristically a ‘march’ or progression of the paresthesia from hand proximally and then to face (Ziegler, 1991). Numbness of one side of the face, usually the mouth, and also in the hand and arm on the same side of body, weakness or clumsiness in the arms and legs, often occur as part of an aura (Kandel and Sudderth, 1998).

**HEADACHE:** Migraine headache commonly starts as a dull headache which rapidly becomes more severe and assumed a throbbing or pulsating quality (Lance, 1969; Saper, 1983; Atkinson and Appenzeller, 1984). Pain may be described as “the pressure of an iron ring”, “being hit on the head by a hammer”, “a needle through my eye” etc. (Friedman and Merritt, 1959). Headache tends to start in the morning, on waking or soon after and generally increases in intensity, taking an hour or two to
reach its maximum severity, and remain at plateau for several hours before slowly diminishing, or sometimes rapidly after remitting, or after a sleep, particularly if it is deep sleep (Clifford Rose, 1988). Migraine headaches may last 4-72 hours (IHS, 1988; Ramaiah, 1996; Ellis, 1997; Wilkinson and MacGregor, 1999; Trickett, 2000). Patients with migraine often report unilateral onset of the headaches with subsequent progression to bilateral (Friedman and Merritt, 1959). Headache may even switch sides (Leyton, 1955; Kandel and Sudderth; 1998). de Queiroz et al. (1998) conducted a detailed study of clinical characteristics of migraine without aura. The headache was side-locked in 19% of the patients. It was exclusively bilateral in 9% of the patients. The majority (86.2%) of the patients, who described headache in only one site, located them, in fronto temporal area. Neck pain was associated with migraine attacks in 70.5% and face pain in 73.5%. A pounding quality was noted by 81% of the patients.

**NAUSEA:** Nausea is the most frequently associated symptom with headache in migraine (Friedman and Merritt, 1959; Blau, 1983; Ellis, 1997; Kandel and Sudderth, 1998). Other symptoms along with nausea are vomiting, diarrhea, and constipation (Trickett, 2000), anorexia, abdominal cramping or simply a queasy feeling are often present (Saper, 1983). Nausea occurs in about 95% of attacks, vomiting in about 25% and diarrhea in 20% attacks (Wilkinson and Mac Gregor, 1999). de Queiroz et al. (1998) in their study found that nausea occurred in 91% of patients and vomiting in 50% of patients. Nausea often comes on within an hour of onset of the headache and may proceed to vomiting, which often provides relief, to the extent that a sufferer may well self-induce vomiting by stimulating the back of the throat. Vomiting tends to more likely in the more severe attacks (Clifford Rose, 1988). Leyton (1955) stated that with vomiting, headache subsides. In some cases the vomiting is so severe that the sufferer cannot, take tablets. In that case
patient may require inhaler or injection (Wilkinson and MacGregor, 1999).

PHOTOPHOBIA AND PHONOPHOBIA: Photophobia and phonophobia are also associated with migraine headaches (Atkinson and Appenzler, 1984; Ellis, 1997; Kandel and Sudderth, 1998). During migraine attack the sufferer has acute sensitivity to light, smell, and sound (Kandel and Sudderth, 1998; Trickett, 2000). About 80% of the patients find light unpleasant during headache. de Queiroz et al. (1998) conducted a detailed study of clinical characteristics of migraine without aura and found that photophobia and phonophobia were present in 77% of the patients. Kallela et al. (1999) stated that there remain trends for more photophobia in migraine with aura and more nausea in migraine without aura.

RESOLUTION AND RECOVERY: In the resolution stage, pain subsides and the body functions return to normal. Resolution can be either a gradual process as during sleep, or sudden, as after vomiting (Ramaiah, 1996). Blau (1982a) conducted a study on the different ways by which migraine sufferers resolve their headaches. Out of 50 subjects, 18 subjects found decreased imperceptibly during the day “it just drifts away”, eight subjects noted a rapid decline of pain estimated between ¼ and 1 hour; in four of these vomiting preceded pain relief. Twenty eight subjects noted relief after a night’s rest and 14 subjects ended attack by going to bed during day light hours. Many attacks in children often last only 2 to 6 hours and in adults attacks end gradually after 24 to 48 hours (Pearce, 1994). Termination of attack is gradual and often accompanied by extreme fatigue or weakness (Friedman and Merritt, 1959; Ramaiah, 1996; Kandel and Sudderth, 1998). Blau (1991) found that most common symptoms that remained after migraine were physical and mental tiredness, subdued or depressed mood, impaired
concentration, reduced physical activity and yawning. However weak or clumsy limbs, head tenderness, neck ache or stiffness, impaired sight and altered fluid balance were less frequent. These number of symptoms ranged from 2 to 11 (average 6) per patient lasting for a near of 18 hours, usually the whole of the next day. Only minority of migraine sufferers feel ‘cleansed’ and in high sprits after the migraine attack (Trickett, 2000).

ETIOLOGY

Precipitants or trigger factors can provoke a migraine attack in a susceptible person. Virtually anything in the external environment and many things in the internal milieu may provoke migraine in susceptible individuals (Soloman, 1994).

Stress, food, allergies, neuroendocrine imbalances, and nutritional deficiencies may all contribute to migraine attack (Sinclair, 1999). Spierings et al. (2001) conducted a study and found that most common precipitating factors acknowledged by migraine and tension headache groups of patients were stress/tension, not eating on time, fatigue, and lack of sleep. Weather, smell, smoke, and light were the precipitating factors that differentiated migraine from tension headache subjects. Turner et al. (1995) conducted an investigation of migraine trigger factors in non clinical population. The most frequently reported triggers for females with migraine were missing meal (58.9%), weather changes (54.4%), menstruation (53.6%), post-crisis let down (52.7%) and fatigue (51.8%). The most frequently reported trigger for males were fatigue (58.8%), sleep (56.3%), post-crisis let down (41.2%), and weather change (37.5%). Leviton (1984) stressed on multi factorial model and holds that the risk of migraine is the sum of all the potential risk factors. Exposure to A and exposure to B usually have an additive effect.
FOOD: Lack of food, insufficient food, food allergies, or intolerance to a particular food item can precipitate migraine attack in susceptible person. Fasting and missing or delaying a meal can result in a migraine in susceptible people (Saper, 1983; Ramaiah, 1996). According to Robbins (1994), missing a meal is a triggering mechanism in up to 40% of those suffering from migraine. Martin and Seneviratne (1997) stated that hunger and negative affect can precipitate headaches in individuals who suffer from both migraine and tension type headache.

Intake of certain food can cause migraine attack in a susceptible person. Twenty percent of the migraine sufferer's link certain food to their migraine (Clifford Rose, 1988; Wilkinson and Mac Gregor, 1999). The most common food items are chocolate, cheese, and citrus fruits, commonly referred to as the three C'S (Wilkinson and Mac Gregor, 1999). Savi et al. (2002) found that approximately 1/3 of the patients reported susceptibility to certain foods. The foods more commonly reported as headache triggers were alcoholic drinks, chocolate, and cheese.

Tyramine is a potent trigger of migraine in selected individuals particularly if ingested in large amounts (Kandel and Sudderth, 1998). Tyramine is a naturally occurring sympathomimetic amine that acts directly on the blood vessels and causes the release of nonepinehrine (Ryan and Ryan, 1978). Food rich in tyramine such as cheese, beer, red wine, chocolate, beef, liver, canned meats, soya sauce, eggs, broad beans, spinach, oranges, figs, plums, bananas, and tomatoes are suspected as migraine precipitants (Ramaiah, 1996).

Chocolate is among one of the common precipitants of migraine headaches (Saper, 1978; Saper, 1983; Clifford Rose, 1988; Ramaiah, 1996; Wilkinson and Mac Gregor, 1999). It contains vasoactive amines (Hanington and Harper, 1968) and is frequently incriminated as a cause of headache (Hanington, 1969; Hanington et al. 1970).

MSG (Mono Sodium Glutamate) popularly associated the Chinese food can cause migraine in susceptible persons (Saper, 1983; Ramaiah,
1996; Kandel and Sudderth, 1998). Many migraineurs avoid food products containing sodium nitrite (e.g. preserved meat and fish, hot-dogs etc) since they can trigger migraine (Kandel and Sudderth, 1998). Citrus fruits can be potent trigger for migraine in susceptible individuals (Saper, 1983; Clifford Rose, 1988; Ramaiah, 1996).

Excessive intake of caffeine can trigger migraine headache (Saper, 1978; Ziegler, 1991). The role of caffeine in triggering migraine is probably not related to the immediate ingestion of this compound but rather to its withdrawal (Kandel and Sudderth, 1998). People who drink lot of tea, coffee, or cola drinks develop headache on sudden withdrawal of caffeine which causes distension of the blood vessels and therefore headache (Ramaiah, 1996).

Alcoholic beverages are one of the most frequently cited trigger of migraine (Friedman and Merritt, 1959; Amery and Vandenberg, 1987; Walji and Kingston, 1994; Ramaiah, 1996; Wilkinson and Macgregor, 1999). Peatfield (1995) conducted a study and found that 18.4% of migraine patients reported sensitivity to all alcoholic drinks, while another 11.8% were sensitive to red wine but not to white wine, 28% migraines patients reported that beer could precipitate headaches.

Vitamin B deficiency, fried and fatty food and indigestion can some times precipitate a migraine attack (Selby and Lance 1960; Gould, 1973).

**STRESS:** Mental or physical stress can become main precipitating factor of migraine in susceptible person. Stress and strain, fatigue, worry and other trials of life do precipitate attacks of periodic headache in those individuals who are susceptible to it (Leyton, 1955). According to Selby and Lance (1960), “migraine sufferers report more minor life stresses than controls and appear to react more to them.” A high frequency of stress was found in migraine sufferers. Interpersonal stress was more prevalent in females, where as occupational stress was more prevalent in
males (Puca et al., 1992). Kroner-Herwig et al. (1993) found no support to assumption of higher sensitivity to stress in migraineurs.

More often, it is a period of relaxation after stress which may trigger off a migraine attack, exemplified by those patients who experience headache regularly on weekends when looking forward to a respite from the week’s problems (Lance, 1969). It may be due to sleeping longer, since the attacks may not occur if patients getting up at their usual time (Wilkinson, 1986). According to a study conducted on the women with weekend headache, work habits, family life, and leisure were such as to suggest possible increase in stress and frustration on weekends, which might have made them perceive the headaches occurring on Saturdays and Sundays as more severe (Torelli et al., 1999).

Even pleasant things can be stressful for migraine sufferer, for example promotion at work or a heavy, but interesting assignment (Wilkinson and MacGregor, 1999). Traveling by any vehicle can lead to migraine or physical stress due to jogging or exercise can precipitate migraine (Blau, 1982b; Ramaiah, 1996). According to Ziegler (1991), head trauma and violent exertion initiates a migraine in some. Migraine can be precipitated by relatively mild head trauma sustained by a soccer player (Atkinson and Appenzeller, 1984).

There is substantial consensus among clinicians that stressful experiences precede migraine attacks (Wolff, 1937; Henryk-Gutt and Rees, 1973; Ziegler, 1979) and that migraineurs as a group may be stress prone individuals (Dalessio, 1980). Levor et al. (1986) stated that migraine is an end point of multi day (4 day) cycle of psychosocial instability, manifested in an elevated awareness of stress during that period, and a decline of physical activity. Kohler and Haimerl (1990) carried out a 6-month longitudinal study to examine whether migraine attacks were preceded by or occurred on stressful days. They found that increased stress was found on the day before an attack and on the migraine attack day itself. Certain findings attach more weight to
vulnerability factors or coping abilities of migraine sufferer, compared to social stress they experience (Howorth, 1965; Jensen, 1988). In general, headache group have higher level of arousal and are more responsive to stress (Cohen et al., 1983). Schmitz and Otto (1984) analyzed the case of 30 year male with migraine. They found that variety of stress can lead to migraine and frequency of subject’s migraine attacks depends on his coping behavior. Subject's migraine attacks were observed more frequently with changing level of stress.

Chronic headache patients report a significantly higher frequency and density of daily hassles, but not of major life events, than controls (Benedittis and Lorrenzetti, 1992). Chronic headache patients might have a higher tendency, based on pre-existing vulnerability, ongoing suffering from headache and negative emotional-cognitive appraisal to report more untoward events than they would when they were feeling well (Paykel et al., 1971) possibly because they perceive minor life events as more threatening or as a consequence of their headache problem (Benedittis and Lorrenzetti, 1992).

Onset of migraine can be due to one or more stressful life events with which the person is not able to cope up. Henryk-Gutt and Rees (1973) reported that over half of the subjects had suffered their first migraine during a period of emotional stress. In another study, chronic primary headache patients reported significantly more stressful life events with negative impact on their life style, in a year prior to headache onset, compared with headache-free controls (Benedittis et al., 1990). High intensity stress during mid adolescence increases the likelihood of migraine diagnoses (Waldie, 2001).

**HORMONES:** The normal female life cycle is associated with a number of hormonal milestones: menarche, pregnancy, contraceptive use, menopause and the use of replacement of sex hormones. All these events and interventions alter the level and cycling of sex hormones and may
cause a change in the prevalence or intensity of migraine (Greene and Dalton, 1953; Somerville, 1972; Kudrow, 1976; Silberstein and Merriam, 1999; Silberstein, 1999). The link between migraine and menstruation was first recorded by Hippocrates (Dalton, 1973; Wilkinson and MacGregor, 1999). Menstrually related migraine begins at menarche in 33% of women (Nattero, 1982). The primary trigger for migraine occurring during menses may be the withdrawal of estrogen rather than maintenance of sustained high or low estrogen levels (Somerville, 1971; Somerville, 1972; Silberstein, 1999). Nattero (1982) found that 55% of female migraineurs reported a relationship between migraine and their menstrual cycle particularly during the time of menstruation, ovulation or both. Solbach et al. (1984) reported that 70% of female migraineurs indicated that their headaches were associated with changes in their menstrual cycle. Amery and Vandenbergh (1987) surveyed over 200 women migraineurs and found that 52% considered their menstrual cycle to be related to their migraines making it one of most frequently cited precipitants of headache in this sample. Rasmussen (1993) conducted a study and found that among women with migraine and age of onset before the age of 51, 24% stated menstruation as a precipitating factor and 67% of those had their migraine attacks 1 or 2 days before onset of menstruation, 28% during the menstruation and only 5% during mid-cycle. Of those who had migraine attacks related to menstruation, 70% had more than half of all their attacks in relation to menses. In a recent study Kelman (2004a) reported that only 3.4% of women had headaches exclusively with menses. Development of migraine at menarche and menstrually related migraine attacks are observed principally in migraine without aura (Granella et al., 2000; Massiou, 2000). Attacks of migraine without aura but not migraine with aura are more likely to occur 2 days before onset of menses and first during the 2 days of menses (Stewart et al., 2000; Silberstein, 2001).
Pregnancy has a variable effect on migraine. Although the first trimester frequently witnesses an intensification of existing migraine (Callahan, 1968; Epstein, et al., 1975; Saper, 1978; Silberstein, 1993) relief of migraine occurs during 2nd and 3rd trimesters (Silberstein, 1993). According to Saper (1983), 70% of women experience an improvement during the second and thirds trimester. Following delivery however, headache may return within hours to days. Scharff et al. (1997) studied headache during pregnancy and in the postpartum. Contrary to previous retrospective study reports, migraine sufferers demonstrated an increase in headache in the third trimester. Rasmussen (1993) found that among women who had ever been pregnant, 48% stated unchanged migraine during pregnancy, 48% stated disappearance or substantial improvement of migraine and only 4% stated that migraine got worse. Headache pattern in women changes in relation to fluctuation of oestrogen level. Increased oestrogen level in early pregnancy offers a protective effect against headache, particularly for women with migraine. However some women continue to experience troublesome headaches throughout pregnancy (Marcus, 2002). According to a study conducted by Granella et al. (2000), in both forms of migraine, pregnancy had a favorable effect, however, lower percentage of migraine with aura than migraine without aura showed improvement or remission. Migraine can develop for first time during pregnancy in few cases (Callaghan, 1968; Bending, 1982; Wright and Patel, 1986).

Oral contraceptives can induce, change or alleviate headaches (Massiou, 2000). Use of oral contraceptives increases the frequency and severity of migraine attacks (Whitty et al., 1966; Kudrow, 1976; Dennerstein et al., 1978). According to Massiou and MacGregor (2000), the influence of oral contraception on the course of migraine is highly variable. Headaches that worsen in frequency or severity have been reported in 18-50% of cases, with most attacks occurring during the drug-free interval of the cycle. Migraine improvement has been reported
in 3-35% of women and there has been no change in 39-65% of cases. Kelman (2004a) reported that birth control pills triggered headaches in 64% of the study group. Use of oral contraceptives can worsen migraine with aura more frequently than in migraine without aura patients (Granella et al., 2000). Sometimes a woman with common migraine starts experiencing attacks with an aura when she starts the pill (Wilkinson and MacGregor, 1999).

The influence of menopause on migraine is variable. Migraine may either regress, or worsen at menopause (Whitty and Hockaday, 1968; Greenblatt and Bruneteau, 1974). Many women with migraine, especially those with history of menstrual migraine experience an exacerbation as they approach menopause. During this time, the orderly pattern of estrogen and progesterone secretion is lost. The fluctuating and falling level of estrogen during the perimenopausal years may increase the frequency of migraine (Fettes, 1999). For most women, migraine settles after the menopause. This is possible because the hormonal fluctuations cease and concentration of oestrogen stabilizes at lower levels (Wilkinson and Mac Gregor, 1999). Migraine improved during post menopause in 2/3 of the cases (Neri et al., 1993). A long term study of migraine after menopause was conducted and it was found that of 40 women at menopause, 18 reported no change in their headaches, 5 actually experienced worse headaches, 1 started to have migraine at that point of life and 2 noticed improvement (Whitty and Hockaday, 1968).

**CLIMATIC INFLUENCES:** Change in weather and barometric conditions have been suspected for centuries of provoking headaches as well as other bodily discomforts (Saper, 1978). According to Weather Sensitive Survey conducted by Hoppe et al. (2002), the most frequent symptoms reported by weather sensitive subjects are headaches and migraines. Bener et al. (2000) found hot climate (37.1 %) is one of the most common triggers of migraine. Hot stuffy rooms, cinema halls can precipitate a
migraine attack in susceptible persons (Wilkinson and Mac Gregor, 1999). Dematteis et al. (1994) found significant correlation between geomagnetic activity and migraine attack frequency. According to Arregni et al. (1994), “low level of oxygen most likely influence the prevalence of migraine because migraine is three times higher in residents living permanently at 43,00 m than in residents living at sea level.”

**LIGHTS:** Prolonged exposure to bright light can precipitate migraine in some people (Friedman and Merritt, 1959; Blau, 1982b; Ziegler, 1991; Ramaiah, 1996; Wilkinson and Mac Gregor, 1999). Flickering and dazzling light and irregular illumination or flashes of bright light, prolonged exposure to sunlight can also become precipitating factors of migraine (Saper, 1983; Ziegler, 1991; Kandel and Sudderth, 1998; Avatollahi et al., 2002). Watching movie or television or prolonged focusing on computer, and working down on microscope can too precipitate attack are some individuals (Lance, 1969; Gould, 1973; Kandel and Sudderth 1998).

**NOISE:** Loud noises can trigger migraine attack in susceptible persons (Lance, 1969; Blau, 1982b; Ramaiah, 1996; Wilkinson and Mac Gregor, 1999). According to a study conducted by Bener et al. (2000) among school children; loud noise (41.5%) was one of the most common precipitants of migraine headaches.

**SMELL:** Strong smells and odors can be one of the precipitating factors of migraine headache (Ziegler, 1991; Wilkinson and MacGregor, 1999). According to Spierings et al. (2001), smoke and smell are one of the precipitating factors of migraine. Odors that cause problems in susceptible individuals often emanate from gasoline and cleaning solution, as well as from perfumes, lotions, and deodorants (Kandel and Sudderth, 1998).
SLEEP: Sleep can precipitate exacerbate or relieve migraine headache. Sleep deprivation or loss of sleep can stimulate migraine headache in susceptible individuals (Saper, 1983; Ziegler, 1991; Kandel and Sudderth, 1998; Spierings et al., 2001). Excessive sleep or prolonged sleep can also cause a migraine headache (Saper, 1983; Ziegler, 1991; Kandel and Sudderth, 1998; Wilkinson and Mac Gregor, 1999). Saper (1983) stated that in between naps can also lead a migraine attack. According to a study conducted by Turner et al. (1995), one of the most common triggers for males was sleep.

HEREDITY: The migraine headache has long been considered familial. Some investigators like Liveing (1873), Graham (1937), FitzHugh (1940), Lennox (1941) have presented family occurrences as evidences of its hereditary character. Goodell et al. (1954) collected data showing the occurrence of migraine headache in families for two, three or more generations. It is highly probable that migraine is inherited. When the family history included only parents and siblings, 46% of migrainous patients had a family history of migraine, as compared with 18% of the patients who suffered form typical tension headache, who served as a control group (Lance and Anthony, 1966). If grand parents were included as well 55% of patients had a positive family history (Selby and Lance, 1960).

Migraine is usually inherited through maternal line, 79% being due to transmission through the mother, as compared with 21 % incidence from paternal inheritance (Flateau, 1912). Atkinson and Appenzeller (1984) stated that migraine is familial and 70% of patients have an affected parent. Among women with migraine, the incidence may be as high as 90% and the affected parent is usually the mother. If both parents are affected, offspring has 70% chances of also having migraine. Bener et al. (2000) conducted a study to find genetic and environmental
factors, associated with migraine in school children. The most common migraine symptoms in school children had been aggravated by physical activity (47.2%) and positive family history of migraine (46.5%) and most frequently affected relative was the mother (17.6%). Contrary to this, Water’s (1971) suggested that history should not be included in the definition of migraine and that heredity was much less important in migraine than was usually supposed.

Concordance of “disabling” or “severe” headache was found in two of nine monozygotic twin pairs and in two of 14 dizygotic twin pairs in non-clinic twin population, suggesting that environmental factors are more important than, genetic factors (Ziegler et al., 1975). In another study Lucas (1977) showed concordance in 26% (22/86) of monozygotic twin pairs and in 13% (10/65) of dizygotic twin pairs and concluded that role of genetic factors in migraine are small. Another study found concordance in (12/24) of monozygotic twin pairs and in 10% (6/60) of dizygotic twin pairs and concluded that heredity factors are of significance in the etiology of migraine (Harvald and Hauge, 1956). Russell et al. (1993) carried out a study of patients with migraine with aura and migraine without aura. They found three fold and twofold increase in family risk of migraine without aura and migraine with aura, combined with lack of increased risk of spouses, strongly suggests that migraine without aura and migraine with aura are genetically determined. Russell and Olesen (1995) investigated family occurrence of migraine with and without aura. Different familial patterns indicated that migraine without aura seemed to be caused by a combination of genetic and environmental factors where as migraine with aura was probably determined or exclusive of genetic factors. Vlajinac et al. (2004) studied the hereditary patterns of Belgrade university female students with migraine and nonmigraine primary headache. Migraineurs had significantly more frequently one or more first-degree and/or second-degree relatives with migraine. The results obtained are in line with the
results of genetic epidemiologic studies suggesting that genetic factors play a role in the occurrence of migraine.

So it is evident from the substantial body of literature that migraine etiology involves an interaction between inherited characteristics, which predispose individual to migraine, and exposure to internal factors and/or external environmental factors.