INTRODUCTION
**Historical Account**

"Pigs whose meat is tender have bladders which are like hailstones in the region of thigh, neck and loin. If they are few in number, the meat is leaner; if there are many, the meat becomes soft and filled with serous fluid".

—Aristotle, History of Animals, 384-322 BC

Neurocysticercosis (NCC) is a complex and most common parasitic disease afflicting central nervous system of man. It poses a major health problem in Asia, Africa, Latin America, Mexico and different regions of Europe (Mahajan, 1982). The disease was described in pigs by Aristophanes and Aristotle in the third century BC, but Paranoli is cited as the first to describe in 1550, the presence of round white bladders full of a clear fluid involving the corpus callosum of human brain (Neito, 1982). Human cysticercosis is caused by the infection of larval stage (Cysticercus cellulosae) of tapeworm Taenia solium. Laennec first used the term cysticercus which was derived from Greek words 'Kystic' meaning bladder and 'Kercos' signifying tail (Neito, 1982). Rudolphi in 1809, named the species as 'cellulosae' because of its great affinity for connective tissue.

**Prevalence of the Disease**

The literature available on the prevalence of neurocysticercosis in India and other parts of the Asian subcontinent is very sparse, and whatever existing information available on the prevalence rates is only from autopsy studies. Dixon and Lipecomb (1961) documented one of the largest series of cases in British soldiers and their families in India.
Exceptionally high rates exist in Latin American countries. Flisser et al. (1979; 1980) reported an average of 1.9% of autopsies performed in hospitals of Mexico city and 1% seropositivity in a seroepidemiological survey, carried out in Mexico, provide an evidence of cysticercosis. In Chile and Columbia, 12.5% of all suspected brain tumors have been found to be cases of neurocysticercosis (Barrientos et al., 1967). Canelas (1963) reported 276 cases of neurocysticercosis from Brazil over a period of 15 years. Among African countries, the incidence in Kenya was reported to be as high as 3.2% and in Zimbabwe 0.45% of autopsies carried out (Froyd, 1965 and Gelfand, 1968). In North America, the disease is unusual, however, it has become a significant problem because of increase in immigrant population from underdeveloped and developing countries (endemic areas), (McCormick et al., 1982; Earnest et al., 1987). Important foci exists in the USSR, China, India, Pakistan, the Phillipines and Indonesia (Muller et al., 1987). In India, over a nine years period seroepidemiological study revealed the seropositivity of 17.4 – 29.2% of cerebral cysticercosis in 1038 cases of epilepsy and intracranial space occupying lesions by indirect haemagglutination test (Mahajan et al., 1982). Ramamurthy and Balasubramanian (1970) reported cerebral cysticercosis of 1.5% of all intracranial space occupying lesions. The incidence of 2.2% has been reported from southern part of India in case of epilepsy (Mani et al., 1974).

Life Cycle of Taenia solium and Mode of Infection of Man

Man is the sole definitive host for adult tapeworm Taenia solium and pig is the intermediate host. Following ingestion of viable cysticerci from infected pork (measly pork), the scolex when digested out of the cyst attaches to small intestinal wall. In the intestine of man, it grows into
a adult tapeworm and attains a length of 2-3 meters in 5 - 12 weeks and may live here for upto 25 years. The scolex anchors itself by means of four muscular suckers and a double row of hooklets which are present on the rostellarum. Gravid segments break off from the distal end of the worm may be passed out intact in faeces. Alternatively, gravid segments dis-integrate in the colon with the liberation of thickwalled eggs (31-43 µm in diameter) which are passed out in the faeces. The cycle is completed when eggs are ingested by pigs and eventually develop into cysticerci in various organs especially skeletal muscle.

Human cysticercosis arises when man become an incidental intermediate host. Eggs are ingested in food or water contaminated by human faeces. They begin to hatch in the stomach as the egg wall is dissolved by gastric juices and the process is completed in the duodenum. Oncospheres here emerge, penetrate the intestinal mucosa and enter local lymphatic mesentric vessels. From there, they are transmitted via the blood stream and get lodged in various organs and tissues. The chances of invasion of brain is about 60%, skeletal muscle (5%) and eye 3% (Acha, 1964). Within two to three months, the oncospheres develop into fluid filled bladder worms or cysticerci.

The usual routes of infections are (i) external autoinfection i.e., an individual harbouring an adult worm may get infected via faecal-oral route and (ii) internal autoinfection i.e., resulting from reverse peristalsis, i.e., gravid proglottids would enter duodenum and stomach from the small intestine with the subsequent hatching of eggs.

As cysts mature in various tissues, cysticerci evoke a chronic lymphocytic and granulomatous reaction, the capsule subsequently undergoes
fibrosis and is surrounded by neutrophils, eosinophils and later lymphocytes, plasma cells and giant cells. In the brain, the cyst lies within the wall of neuroglia which later undergoes degenerative change, and appears as a discoloured ring which is walled off from the brain tissue. In muscles and some other soft tissues, the cyst wall may collapse, becoming flattened, and after calcification take on a spindle or oat shaped form. However in the brain, the cysts remain oval or spherical. Dying cysts are surrounded by acute inflammation associated with tissue damage. As calcification of cysts occurs, symptoms usually subside and may disappear completely.

**Clinical Presentation**

The clinical presentation of this disease may be protean as the parasite may produce single or multiple lesions in any part of the brain depending upon the number and location of the cysts. Presentation of disease is usually from two months to 30 years after infection (Wiederholt et al., 1982). The initial symptom is usually appearance of small subcutaneous nodules or intramuscular swellings (Cook, 1988). If neurological involvement does not occur, cysticercosis remains a benign disease. Any part of the central nervous system can be affected and in approximately 50% of the cases, symptoms are seen. The commonest clinical manifestations of neurocysticercosis are seizures, increased intracranial pressure and stroke (Wiederholt and Grisolia, 1982; Grisolia and Wiederholt, 1982 and McCormick et al., 1982). Cysticercosis is classified clinically, broadly into two categories namely symptomatic and asymptomatic (Zenteno Alanis, 1982). The former can be divided into four groups in accordance with the localization of the parasite.
Group I: Disseminated cysticercosis—patients with the parasite distributed in the viscera, skin and muscle

Group II: Ophthalmic cysticercosis—comprising of those affected in the eyes and orbits.

Group III: Neurocysticercosis—parasite loaded in central nervous system.

Group IV: Mixed cysticercosis—cysticerci in more than one of the above locations.

The possibility of diagnosis of neurocysticercosis should be considered whenever patients present with fits, increased intracranial tension and acute or progressive neurologic deficits who had resided in an endemic area. Although the presentation may be a mixed one, clinical neurocysticercosis can be roughly divided into four main groups based on the localization of cysts in the CNS (Cook, 1988).

1. Parenchymal cysts: These are found in the majority of patients and cause fits, acute or progressive focal abnormalities and raised intracranial pressure. Epilepsy may be either focal or general, usually caused by fresh living cysts.

2. Meningeal cysts: These are found in approximately 50% of the affected patients in the basal meninges. There may be intense inflammation with destructive hydrancephalus arterial thrombosis and stroke.

3. Ventricular cysts: These are found in 15% of the affected patients which are common in 4th ventricle and may be free floating or attached. They become symptomatic when they block the aqueduct of sylvius causing raised intracranial pressure which is associated with severe headaches and vomiting.
Spinal-cord cysts: These are found in 3% of cases and cause arachnoiditis with transverse myelitis or signs of a local mass lesion.

Recently, Sotelo et al (1985) proposed a classification of neurocysticercosis into two forms - (i) active and (ii) inactive based on the results of radiological studies and CSF analysis, which is devised for diagnostic and therapeutic purposes. Active form of NCC include arachnoiditis, hydrocephalus, secondary to meningeal inflammation, parenchymal cysts, brain infarction secondary to vasculitis, mass effect due to a large cyst or cyst clumps, intraventricular cysts and spinal cysts.Inactive forms of NCC are parenchymal calcifications and hydrocephalus secondary to meningeal fibrosis.

Other clinical manifestations which have been described in another series of cases include cerebral infarction, ocular involvement, dementia, spinal arachnoiditis, thrombosis of superficial cortical vessels by chronic meningitis and a hemisensory deficit (Grisolia and Wiederholt, 1982). Korsakoff's psychosis, Parkinson's disease, motor neuron disease, a variety of pituitary fossa syndromes and isolated cranial nerve palsies have also been recorded (Grisolia and Wiederholt, 1982).

Idiopathic epilepsy, multiple intracranial space occupying lesions, chronic meningitis and other causes of raised intracranial pressure can be mimicked. Differential diagnosis include tuberculosis, cryptococcosis, neurosyphilis, primary and secondary malignancies affecting the central nervous system (Grisolia and Wiederholt, 1982).

The above mentioned severity and manifestations of neurocysticercosis largely depend on several factors (Sotelo, 1987). These include (a) the number of cysticerci; (b) the host immune response against the parasite...
from immune tolerance to a severe inflammatory reaction; (c) the localization of cyst, eg., in brain parenchyma, ventricles, subarachnoid space or spinal canal and (d) the state of activity of lesions, from cysts and inflammation to granuloma, calcification and residual fibrosis. All of the above factors present multiple combinations making neurocysticercosis a complex disorder (Sotelo et al., 1985).

Diagnosis

The diagnosis of neurocysticercosis is based mainly on clinical symptoms in combination with radiological findings. Definitive diagnosis depends on histological examination of a cysticercal cyst (Cook, 1988). Imaging techniques like computed tomography (CT) and nuclear magnetic resonance (NMR), have vastly improved diagnostic accuracy for neurocysticercosis (Rodriguez – Carbajal, 1982). Routine CSF analysis is of little value diagnostically (Grisolia, 1982; McCormick, 1982). On the other hand, Sotelo (1988) suggests that the precise information on the diagnosis and characteristics of neurocysticercosis could be obtained through a combination of CT and CSF analysis.

Immunodiagnosis of neurocysticercosis has improved in the recent years. A number of immunodiagnostic tests like gel diffusion (Biagi and Tay, 1958), complement fixation test (Neito, 1956; Mahajan et al., 1975), immunoelectrophoresis (Espinoza et al., 1982), indirect haemagglutination test (Powell, 1966; Mahajan et al., 1974; Larralde et al., 1986) and enzyme linked immunosorbent assay (ELISA) (Arambulo, 1978; Diwan et al., 1982; Espinoza et al., 1982; Mohammad et al., 1984; Rosas et al., 1986; Larralde et al., 1986; Coker-vann et al., 1984; Nascimento et al., 1987) have shown varied results using different sources of antigen for the
detection of anticysticercal antibodies in serum and or CSF. Cross-
reactions with hydatidosis, coenurosis cerebralis, and other phylogeneti-
cally related parasites still appears to be troublesome in spite of using
purified or partially purified antigens (Espinoza et al., 1982; Coker-vann
et al., 1984).

Detection of cysticercal antigens in human CSF has also been
attempted as a diagnostic tool in NCC (Estrada and Kuhn, 1985; Telez-
Girron et al., 1987).

Thus it appears that many workers are of different opinion regarding
the use of source antigen in immunoassay, which has yielded varied
results. Currently available methods lack sensitivity and specificity due
to the use of ill defined cysticercal antigens in immunoassays. There has
been no collective study on characterization of specific antigens in
various compartments of the cyst to use in immunoassays. Therefore, there
is a great need for a systematic approach to identify, characterize
specific antigenic components of *Cysticercus cellulosae* and its morpho-
logical parts.

In this study an attempt has been made in this direction. Antigens
prepared from different compartment of the cyst was evaluated and few
simple and sensitive immunological methods were standardized to apply them
in immunodiagnosis of neurocysticercosis.