CHAPTER I

INTRODUCTION
Stuttering is defined in the International Classification of Diseases (World Health Organisation, 1991) as "Speech that is characterized by frequent repetition or prolongation of sounds or syllables or words; or, alternatively by frequent hesitations or pauses that disrupt the rhythmic flow of speech."

This disorder of communication has been extensively researched with respect to its etiology, onset, nature and treatment. Theories concerning the etiology of stuttering broadly fall into three groups, namely, stuttering as a neurotic response, stuttering as learned behaviour and stuttering as a physiological deficit (Andrews et al, 1983).

The search for a physiological basis for stuttering is understandable considering that stuttering has often been reported following brain damage. Such a disorder has been termed neurogenic or acquired stuttering. In contrast to stuttering which begins in early childhood, usually between the ages of 2 and 5 and in the absence of any obvious cause, acquired stuttering can begin in a previously fluent speaker following a lesion in the brain.

Acquired stuttering can occur following right sided as well as left sided brain damage (Lebrun et al, 1987b). It has been reported in neurological conditions like Parkinson's disease (Lebrun et al, 1987a) where the extrapyramidal system
is involved. Lebrun et al (1983) have also reported acquired stuttering in upper motor neuron disease and suspected that the supplementary motor area or precentral gyri were involved. Thus, acquired stuttering can occur in diverse neurological conditions and because of various different brain sites being involved. This makes it difficult to localize the area responsible for stuttering which occurs in the absence of neurological conditions. McClean (1990) suggests that this may indicate that the neural source and process of stuttering may be heterogenous.

Orton (1927) and Travis (1931) were among the earliest investigators who regarded stuttering as a physiological deficit. They theorized that stuttering results from "incomplete" cerebral dominance, specifically, due to incomplete lateralization of language.

Cerebral dominance refers to "the superior capacity of each side of the brain to acquire certain skills" (Geshwind and Galaburda, 1985a). Existing data suggests that in 96% of right-handers and 66% of left-handers, language is lateralized to the left hemisphere (Bryden and Steenhuis, 1991). Anomalous, mixed or incomplete dominance has been reported not only in left-handers but also other individuals whose pattern of dominance varies from that of the majority.

Early studies on cerebral dominance and stuttering were restricted to examining the relationship between handedness
and stuttering. These included studies of shift of handedness and its relationship to stuttering. The relationship between handedness and stuttering however, was not proven and consequently, interest declined in the theory of incomplete cerebral dominance.

At about this time, psychoanalytic theories of stuttering (Fenichel, 1945) gained prominence. This was followed by the learning theories such as the approach-avoidance model of Sheehan (1953) and the two-factor model of Brutten and Shoemaker (1967).

Interest in cerebral dominance was reawakened following the finding of Jones (1966) who found evidence of incomplete dominance in stutterers. Jones used the WADA test in 4 stutterers prior to surgery. The WADA test consists of an intracarotid injection of sodium amytal. When the language dominant hemisphere is exposed to the short-acting barbiturate, the patient becomes transiently aphasic. Jones noted that all four stutterers became aphasic irrespective of whether the left or the right carotid artery was injected. He concluded that both hemispheres of stutterers were significantly contributing to language.

The provocative findings of Jones (1966) led researchers to review the concept of incomplete cerebral dominance in stuttering. Modern imaging techniques have been used to
examine cerebral dysfunction in stutterers. Wood et al. (1980) measured regional cerebral blood flow (rCBF) in two stutterers during periods of differing stuttering severity. Both stutterers showed higher Broca's area rCBF in the right as compared to the left hemisphere during stuttering. Both showed higher Wernicke's area rCBF in the left compared to the right hemisphere during stuttering. However, during reading aloud without stuttering, the rCBF in the Broca's areas were reversed.

Strub et al. (1987) studying a sibling pair of stutterers using computerised tomography (CT) of the brain reported atypical CT scan asymmetries in both subjects, especially in the posterior (temporo-occipital) areas of the brain. This was related to the use of different processing strategies by the subjects as shown by the neuropsychological and dichotic listening tests.

Rastatter and Harr (1988) showed that plasma glutamine levels of five stuttering subjects was significantly higher as compared to normals. This was interpreted as reflecting a possible increase in left hemispheric GABA (gamma amino butyric acid)ergic activity.

Newer imaging techniques like Positron Emission Tomography (PET) now permit the functional visualization of the intact brain. This opens up new vistas for research in
an attempt to delineate brain structures underlying stuttering.

Possible hemispheric processing differences between stutterers and nonstutterers have been explored using modern investigative techniques. These include dichotic listening tasks, tachistoscopic procedures, electrophysiological studies (electroencephalographic techniques and the averaged evoked response) as well as dual task studies. These techniques have been popular in research as they are safe and noninvasive procedures. The studies, by and large, reveal hemispheric processing differences between stutterers and nonstutterers and various hypotheses have been put forward to explain these differences. Further evidence is required, however, for the emergence of a neuropsychological theory which can explain the etiology of stuttering.

Possibly, a combination of information processing techniques and imaging methods might provide a more complete understanding of the brain mechanisms underlying stuttering.