THE G.P.I. AND THE SPEECH PATHOLOGIST

PRATHIBHA S. SHETTY

'Half a century ago general paralysis of the insane constituted a high proportion of mental hospital admissions and accounted for an appreciable part of the chronic population of such institutions. However, with the development of increasingly effective methods of treatment, cerebral syphillitic infection is becoming relatively rare' (Mayer-Gross *et al.,* 1960).

This statement, however, may not hold good in our country where cerebral syphillitic infection is still at large and cases of general paralysis of the insane (G.P.I.) are not uncommon. As such G.P.I, not only poses a present day problem to us but will also probably remain so for several decades to come.

Definition: 'The chronic brain syndrome associated with syphillitic meningo encephalitis, formerly known as general paresis, general paralysis of the insane, or dementia paralytica is a disorder produced by a progressive syphillitic meningo encephalitis leading to a degeneration of brain parenchyma with an infiltration of interstitial elements. Clinically general paresis is characterized by a comprehensive but variable syndrome of neurological and mental disturbances associated with fairly constant serological changes' (Noyes and Kolb, 1966). Although syphillitic meningo encephalitis is more acceptable, the term G.P.I, is more commonly used and understood elsewhere and in our country and hence it has been retained in this paper.

Pathology: The pathological process of inflammation and degeneration seen in G.P.I, is a chronic syphillitic meningo encephalitis. The degenerative process that begins and is specially marked in the frontal region, progresses to the temporal and parietal regions with the progress of the disease, resulting in a thickened meninges which is adherent to a brain shrunken in size with widened sulci and indistinct convolutions. Other important histopathologial features of paresis are an increase in subarachnoid and ventricular cerebrospinal fluid, a thickened ventricular ependyma; microscopically a diffusion of leucocytes and plasma cells in the pia arachnoid, an increase in number as well as size of microglia cells seen as 'rod cells' and presence of iron pigment in the walls of the cortical vessels and in the cortical microglia.

Clinical picture: G.P.I, usually develops after about five to twenty five years of the primary infection with syphillis. The first symptoms of G.P.I. appear generally when the patient is between thirty and fifty years of age, and occurs three to four times more often in men than in women. During the pre-clinical

Miss Prathibha S. Shetty is an Internee at the A.I.I.S.H.

period when the patient is said to be suffering from asymptomatic paresis, **the forerunner of clinical paresis, spinal fluid** findings characteristic **of** the disease **is the only evidence of the condition.** A similar syndrome caused by congenital **syphillis** may be seen in children and adolescents and is known as juvenile paresis.

The clinical symptoms of G.P.I. make its first appearance in terms of changes in personality and mentation. The very first symptoms are often an exaggeration of previous personality traits. On questioning, the patient will reveal that he has been suffering from giddiness, insomnia, headache, irritability, fatigue, depression, difficulty in concentration, periods of confusion, restlessness, weakened drive and vitality and such other ill defined symptoms, for sometime.

One of the important early symptoms of G.P.I. is a dementia which in the early stage is diffuse involving memory, particularly for recent events, and judgement. As the disease progresses the thought process becomes retarded and laboured. Associations are slow, limited and superficial. The patient has difficulty in grasping situations and in discussions beats around the bush with vague irrelevancies. Professional skill is impaired. Episodic clouding of consciousness may also be seen. Apperception is destroyed and there is a cessation of spontaneous activities. Tests of intelligence indicate mental deterioration. The patient, however, lacks insight into his condition. He often exhibits a contented indifference, an apathy and unconcern which may be mistaken for laziness. Noticeable too is a frequent tendency to drowse. As the disease progresses and reaches its final stage the mental processes of the patient are reduced to the reflex and vegetative level.

Parallel to the intellectual deterioration of the G.P.I. is a breakdown of higher ethical and cultural sentiments and standards. The behaviour of the person often shows a deterioration of tact and judgement and an undermining of moral and ethical control. These behavioural changes are often noted before any serious intellectual changes have occurred and are usually the precursors of intellectual deterioration. As the dementia progresses emotional responses are impoverished to such an extent that news of the death of a dear friend or relative is accepted nonchalantly. More often than not the patient responds with a vacant stupefied equanimity to all such situations. Change of mood of which apathy and depression are frequent is yet another characteristic of G.P.I. While anxiety may be exhibited by some cases in the early stages, some others are euphoric and expansive in the later stages. Delusions are also experienced by general paretics, most of which are grandiose. Some patients are also observed to experience depressive, self accusatory, nihilistic or persecutory delusions.

In the initial stages of paresis the patient usually has numerous somatic complaints such as headache, giddiness etc. He is easily fatigued and is physically weak. He is under weight and the condition is often mistaken for one of neurasthenia. Optic atrophy and pupillary disturbances are also common symptoms of G.P.I. There is a loss of tone in the facial muscles and a progressive incoordination of all voluntary muscles, including those of the face as a consequence

of which speech becomes distorted. The patient's gait becomes slouchy with flabby musculature. With progress of the disease the patient becomes bed-ridden.

Several neurological symptoms are associated with G.P.I. The deep tendon reflexes are, as a rule, exaggerated. Sudden movements in an advanced case often causes fractures due to trophic changes in the bone. The paretic subject is also noticed to develop a sudden rise in temperature, the cause of which is unknown. Most paretics also suffer from convulsions at some time during the course of their illness. The convulsions are most often of the epileptic form but at times are of the apoplectiform type, followed by temporary hemiplegia or localized paralysis. Occasionally a permanent paralysis is left by a hemorrhage.

Serological changes such as positive Wasserman reaction on blood and cerebrospinal fluid indicates neurosyphillis. An increase in cell count, protein and a rise in the globulin content of the cerebrospinal fluid is usually indicative of G.P.I.

Clinically G.P.I. may be confused with neurasthenia, manic-depressive psychosis, chronic alcoholism, presenile dementia, Alzheimer's disease, Pick's disease, multiple sclerosis, cerebral tumour or other such diseases producing cerebral damage. However, when data gathered by mental, neurological and serological tests are put together an unmistakable pattern or syndrome of G.P.I. emerges.

*Speech of the general paretic: D*istorted speech is one of the very first clinical symptoms exhibited by the general paretic. In the early stages the speech shows a gross dysarthria characterized by slurring, drawling and hesitations, which can be explained in terms of the weakness and incoordination of the voluntary muscles of the paretic patient, mentioned earlier. Since these symptoms are quite apparent, they might bring the patient to a speech clinic. Tremor of the facial muscles, first noticed in the lips of the paretic makes articulation, particularly of labials and labio dentals, difficult. A coarse tremor of the tongue may affect the tongue-tip consonants. Consequently, there is an overactivity of all facial muscles when speech is attempted, which further distorts speech. The paretics' articulation is now characterized by omissions, substitutions and repetitions. One important feature to be kept in mind is that at this early stage of the disease the paretic usually has good control over his articulation at the syllable and word level. The speech disturbance is noticeable more often than not only in running speech.

In addition to defective articulation the voice of the paretic may also be affected. If the general paresis involves the laryngeal nerves and muscles resulting in a paralysis of the vocal cords, neuromuscular incoordination or poor muscle tone, the paretic's voice becomes' breathy and/or hoarse. Fading of voice or abrupt changes in pitch and intensity during conversations may also be observed. (If the muscles of deglutition are involved, as they sometimes are, the patient complains of difficulty in swallowing.) Muscular incoordination of the fingers make them tremulous, thus resulting in distorted writing. Letters may be reduplicated

or omitted when writing, and often show diminution in size and spacing making it illegible.

As the disease progresses 'Speech is disturbed not only by dysarthria but also by emotional, volitional and linguistic aberrations' (West 1957). Words are often omitted and the paretic becomes incoherent in thought and speech. His speech now resembles that of the expressive aphasic. However, the two conditions should be differentiated as the underlying pathology in the two are different though the symptoms are alike. Differential diagnosis is also important in view of the very different treatment procedures involved. This is all the more important, because the pathology underlying the speech symptoms of G.P.I. is a degenerative process unlike that of the aphasic, and as the disease progresses speech of the general paretic takes on a psychotic colouring which again is not true of aphasia.

Differential diagnosis between aphasia and G.P.I. becomes a rather ticklish problem in cases of head trauma, for a head trauma can precipitate general paralysis in a patient already suffering from syphillis. A head trauma if of sufficient intensity either activates an asymptomatic process into a clinical condition or aggravates an already present but unrecognized clinical condition of G.P.I. As such it is highly probable that the condition be mistaken for one of true expressive aphasia the head trauma being identified as the cause of aphasia. It is therefore advisable in all cases of head trauma resulting in aphasia or aphasic like symptoms to take a careful case history that should reveal syphilitic contact if any in the past and also to have serological examinations done so as to rule out the possibility of G.P.I.

Aphasia may be super imposed upon an already present general paresis, for as mentioned earlier, during the course of the disease a haemorrhage leaving a permanent paralysis may occur. Here again, a carefully taken history will reveal the G.P.I. symptoms being present earlier to the onset of paralysis and concomitant symptoms.

When the entire clinical picture of which speech is an important element, is obtained, general paralysis of the insane cannot be mistaken for any other than what it is.

The Responsibility of the Speech Pathologist

Considering the fact that speech is not only one of the earliest functions to be involved in G.P.I. but also one which in the early stages draws comparatively greater attention to itself, it is not surprising that the speech pathologist encounters with quite a few cases of G.P.I. in the course of his profession. The speech pathologist's responsibility towards the general paretic can be considered to be two fold: (I) To be able to recognize the condition of general paresis and help the patient avail immediate medical treatment to arrest the underlying degenerative process in untreated cases, and (2) To provide therapy for those cases whose disease has been medically arrested but who are left with residual speech symptoms.

It is essential that a speech pathologist be familiar with the clinical picture of G.P.I. so as to be able to recognize the condition when he is confronted with

one. G.P.I. today is effectively treated medically with a course of penicillin injections. The earlier the treatment the better the prognosis, provided of course that the treatment is adequate. In fact early and effective medical treatment brings about a complete cure. Consequently the speech pathologist can be instrumental in bringing about a complete cure by immediate recognition of the , disease followed by prompt action, for he may often be the very first specialist to be consulted by the patient. On the other hand a speech pathologist who is ignorant of this condition can do great harm by taking up speech therapy for the patient, having mistaken the problem for aphasia or dysarthria and thus wasting precious time while the underlying disease progresses to such an extent that a day may come when the patient suddenly appears in the speech clinic with a full-blown psychosis with very bizzare behaviour. Then there would be little hope left for a total recovery.

The literature in the field of speech pathology is noticeably silent in this area, probably due to the fact that G.P.I. is no longer a pressing problem in the Western countries to whom we owe most if not all of our knowledge of speech pathology. Although there are no statistical figures available it is probably true that the treatment and prevention of syphilis and the consequent reduction in G.P.I. have not been as effective in our country as they have been elsewhere. And hence the speech pathologist in our country should probably be more alert to this condition. We could work with the speech of the paretic whose illness has been medically arrested, but has a residual speech problem, with a few choice methods. A method of choice for the paretic whose residual speech symptoms is of the dysarthric type would be to increase his control, in the clinical setting, over articulation in sentences of varying length, with the syllable or word level as the basal line, for most of these cases have good control over articulation at the word and syllable level. This method was tried at our Institute with case No. 1805 reported elsewhere in this journal. Half an hour therapy sessions were held daily for a period of 15 days at the end of which the case was forced to discontinue the therapy due to lack of leave of absence from his work. Some improvement was noticed. If and when the voice is involved, therapeutic methods used for paralysis of the vocal folds in general, can be tried with these cases also. Paretics with residual symptoms of expressive aphasia could perhaps be worked with on the same therapeutic principles as used with other cases. However more systematic work is needed to find efficient techniques for the arrested G.P.I. cases.

Summary

In view of the fact that literature in speech pathology is lacking in adequate information on G.P.I., one of whose major symptom is a speech problem, an attempt was made to summarize the symptomatology and pathology of this condition. It was thought that such an attempt would familiarise the speech pathologist with the syndrome of general paralysis of the insane. Having defined G.P.I.

a brief discussion of the underlying pathology was presented followed by a description of the clinical picture in all its different aspects—intellectual, emotional, physical, neurological and serological, with special reference to the speech problems. The responsibility of the speech pathologist towards the general paretic was then discussed, followed by brief suggestions for therapy.

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