APRAXIA DYSARTHRIA

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PART 1

The diagnosis of apraxic dysarthria still puzzles many speech therapists, and some even doubt its existence despite the fact that it is firmly established in the literature, and at least one standard textbook on speech disorders devotes a whole chapter to the condition.

This paper attempts to explain the condition, how it comes about, its clinical manifestations, and possible methods of treatment. Consider the following case:

John I. is aged seven years. He was born after a normal full term pregnancy, but labour was prolonged and forceps were applied towards the end of the second stage. He appeared to be a healthy infant and developed normally. He sat at five months, crawled at eight months, and walked at thirteen months. He said his first words at fourteen months which were "mamma" and "nanna". Thereafter, his vocabulary slowly increased, and he began to use two to three word sentences only, at the age of three years. From an early age it was noticed that his speech was not clear and this has persisted until now. In all other respects he is quite normal. His parents and siblings understand his speech, but strangers have great difficulty in doing so. He started going to school one year ago and there his speech disorder has become a source of embarrassment. The children tease him and ridicule his "baby talk". As a result, he has become sullen and withdrawn. He can be persuaded to go to school with great difficulty. He has had speech therapy since the age of five years, with little benefit. The family history background is quite normal. There is no suggestion of any emotional disturbance. The mother, father and two siblings are right-handed, and all speak normally.

Speech Examination: This revealed that child's speech is at times almost unintelligible. He has little trouble with vowel sounds or short words, but long words and consonants cause him great difficulty. He regularly mispronounces the "k", "t", "w", "s", "d", and "g" sounds. There are many substitutions and omissions, especially of final sounds. When persuaded to speak very slowly, speech becomes much clearer. He can repeat individual sounds and words quite well. There was no evidence of dysphasia and intelligence as tested on the South African Individual Scale was above average.

Physical Examination: This revealed no significant abnormalities, except that the reflexes in the right half of the body were slightly brisker than those on the left, and the right lower limb was slightly underdeveloped. He is right-handed. There was no evidence of any dysfunction in the lips, tongue, palate or larynx. His reading, writing, and spelling were within normal limits.

Into which diagnostic category can this child be placed? He shows evidence of a very mild left cerebral (dominant) hemisphere lesion, but this has not produced any physical defect in the function of the lips, tongue, palate and larynx. However, when he speaks a serious disorder

appears. His higher speech centres are intact and there is no evidence of any weakness, ataxia, or sensory loss in the lower organs of speech, and no structural defect or hearing loss. Psychogenic factors have been excluded. Where then does the defect lie? He has been previously diagnosed as suffering from dyslalia, but has not responded to suitable therapy. The latter diagnosis thus appears to be wrong, for the following reasons:

1. There is evidence of slight brain damage (the possible cause being a forceps delivery).
2. The condition is far too severe and persistent.
3. Therapy for dyslalia has been unsuccessful.
4. There is no apparent cause for dyslalia.

It is thus suggested that the correct diagnosis is apraxic dysarthria.

A consideration of Apraxic Dysarthria

When the volume of sound and symbolic content of speech is normal, but the articulation and enunciation of the individual words and phrases are distorted, a patient is said to be suffering from dysarthria. Apraxia implies that the motor disorder is due to a lesion in the highest motor centres of the cerebral cortex where voluntary movements are initiated, planned and synthesised, so that the correct result is obtained in the peripheral motor organ. The correct organisation of movements is called praxis, and a disturbance of this function is termed apraxia. It has nothing to do with the strength of muscle contraction, co-ordination or sensation.

There are many definitions of apraxia, but most comprehensive is that given by Worster-Drought:

> "The inability, as the result of an organic brain lesion, to execute familiar, purposive, more or less automatic movements, when there is neither motor paralysis, sensory disturbance, ataxia, or any intellectual impairment."

It is as if the patient retains the will to perform the act, and retains the neurological apparatus capable of performing the movement, but between the two there is a gap which he is unable to bridge. The patient knows what he wants to do but cannot do it. The essential feature of the condition is that voluntary movements are affected so that the same movement which cannot be carried out to command, or voluntarily, may still be carried out reflexly. e.g. A patient may not be able to protrude his tongue when asked to do so, but a moment later he may carry out this movement to lick his dry lips. Similarly, a devout Christian may not be able to make the sign of the cross on request, but will do so reflexly on entering a church.

The movements of speech are carried out in an almost automatic manner, and must be considered as the most highly skilled motor patterns that the body possesses. How these and other skilled movements, for example those of the hands, are carried out is still unknown but a study of apraxia does shed some light on the subject.

Electrical stimulation of the motor cortex in the frontal lobe or the motor centres in the extra-pyramidal system produces crude movements.

A lesion in the motor cortex will produce loss of movements or weakness for both voluntary and reflex movements. This is not apraxia. The lesion causing apraxia must be situated at a higher level, or in a higher motor centre. This higher level or centre is not a small discrete structure or isolated portion of the brain, but involves probably most of the cortex of both hemispheres. Apraxia may be produced by lesions in either hemisphere and in almost any region. The most likely site for such a lesion is, however, anterior to the motor cortex in the dominant hemisphere.

How are skilled movements organised in the cerebral cortex? If we desire to initiate or execute a movement, then this must be translated into some method whereby the motor cortex is stimulated. This transition from a psychological state to physical action in the motor cortex was always assumed to be achieved by means of transcortical fibres. In other words, messages were conducted from areas of the brain said to be concerned only with psychological mechanisms to regions where they could be translated into physical action. We now know that this is probably not so, since these transcortical fibres can be severed with little effect on voluntary movements. Another mechanism therefore has to be postulated, and we now believe that these stimuli descend to lower co-ordinating centres in the brainstem, e.g. the reticular formation, and then ascend again to stimulate the motor cortex. The acquisition or learning of a skilled movement in childhood starts as an almost unconscious process by first bringing into play all the sensory faculties, so as to see, feel and even "hear" the movement. These sensory experiences are then used to try and reproduce a similar motor phenomenon. By constant repetition kinetic engrams or "memories of movement" are stored in the neuronal circuits. These engrams then become part of our permanent motor repertoire and we can recall them whenever necessary. If we store faulty engrams, or cannot properly recall them, then we will have imperfect movements. It is these engrams which in turn stimulate the motor cortex.

The process of carrying out a skilled movement can be summarised as follows:

1. The idea of the movement is formulated spontaneously, or in response to an external command.
2. Correct psychic planning of the action.
3. Correct mobilisation of the kinetic engrams.
4. Stimulation of the motor cortex.

If the first step is defective, this leads to a paucity or absence of voluntary movements. Perhaps the catatonic form of schizophrenia is representative of this type of disorder. A disturbance in the second and third steps leads to apraxia. The following discussion on the various types of apraxia is based on the classification proposed by Liepmann.

Ideational Apraxia. If there is incorrect psychic planning of the action, then the patient is said to suffer from ideational apraxia. The basic defect is a disorder of the conception of the required movement. Patients know and understand each individual part of the movement, but mistakes

occur. They cannot reproduce them in the correct sequence and at the proper time. The usually quoted example of this condition is the patient who cannot light a cigarette. He commits surprising errors without being aware of them. He will take out a match, place it in his mouth, and rub the cigarette against the matchbox. This type of apraxia will only affect a complex movement. The patient can still carry out simple movements when requested to do so, e.g. a symbolic gesture. The kinetic engrams are still intact, but they are mobilised incorrectly.

Idiokinetic Apraxia. The patient knows what he should do and how he should do it, but cannot mobilise the necessary apparatus of action. The engrams are there, but he cannot voluntarily set them in motion. Then suddenly he will do so in a reflex or automatic action. Our earlier example of the person who could not make the sign of the cross falls into this group. Another example is that of the patient who cannot wave goodbye on request, but will do so when the doctor leaves the ward, or he cannot point on demand to a specific object, but can do so if he suddenly develops the need for it. This type of apraxia mainly affects symbolic gestures, and it is mainly the intentional or volitional use of the movement which is disturbed.

Motor Apraxia. In this type of apraxia it is postulated that there is a fault in the engrams themselves. They are either absent or faulty. If a patient, through some disease process, loses his motor engrams, then he will behave as if he is carrying out that movement for the first time. He may lose his engrams for only one specific function. e.g. An expert pianist will sit down at the piano and be quite unable to play (instrumental amnesia). A patient may lose the ability to write (agraphia), or play some game at which he was previously skilled. If the engrams are there, but they are faulty, the movements are not lost, but they are performed incorrectly. Bucco-linguo-facial apraxia belongs to this group. If the engrams for movement of the lips, tongue and palate are lost completely, then the patient becomes anarthric. If they are faulty, then a dysarthria will result.

From the above discussion it can now be postulated that an apraxic dysarthria may arise in one of two ways:

1. The kinetic engrams may be at fault.
2. They may be incorrectly or incompletely mobilised.

In this way the inability to reproduce certain sounds and the omissions and substitutions which form the basic symptoms of an apraxic dysarthria may be explained. Translated into speech terms consonants, diphthongs and vowels are affected, in this order. This may be explained on the basis of the relative complexity of the sounds involved, and therefore on the complexity of the motor engrams involved. The more complex engrams would be affected first, and therefore consonants would be disturbed first. In very severe cases all three may be affected.

The literature on the subject of apraxia is vast, and not all authors agree on the same classification. Many special types of apraxia have been described, of which one is of some clinical importance.

Dressing Apraxia. A highly specific disorder in which the patient loses the ability to dress himself. Basically, this is due to a disturbance of body image and a failure to pay due attention to both the left and right halves of the body and garment concerned.

Expressive aphasia could, according to our definition of apraxia, be classified as such a type of disorder. Indeed, some authors follow this practice. This is incorrect, since the term apraxia refers only to motor functions, and aphasia to the higher psychological or symbolic functions of speech. The distinction, however, is probably a very fine one, and the same process may well be involved in both types of disorder. It is certainly no accident that dysarthria is seen so frequently in aphasic patients.

Part 2

By definition the disorder of apraxia dysarthria is due to an organic brain lesion and the patients who come for therapy do not suffer from any of the progressive diseases which lead to total incapacity or death. In practice the exact cause is of no great importance as the patient is usually seen long after he has sustained his brain injury and the neurologist and therapists are asked to diagnose and treat the residual disabilities.

The pathological processes which may cause an apraxic dysarthria vary widely. In children the commonest causes are developmental disorders, birth trauma, infectious diseases, and head injuries sustained during infancy. In adults caused factors are cerebro-vascular accidents and head injuries.

All these disorders may cause widespread damage so that the apraxic dysarthria is associated with other obvious neurological disabilities. Such cases do not generally cause any diagnostic difficulties. Even if the true nature of speech disorder is not recognised, the organic factor will be recognised. The only difficult group of cases are those patients where the speech defect appears as an isolated phenomenon set against a background of an otherwise apparently normally functioning nervous system. The clinical picture is nearly always the same.

The patient, usually a child, is brought by the parents because his speech is not clear and it has become a social embarrassment. There has frequently been a delay in the development of speech, but this is seldom severe. The mother reports that the child understands speech very well and that within the immediate family circle he can make himself understood quite adequately. Strangers, however, have great difficulty in understanding the child. His vocabulary may be very good. Simple words are often pronounced quite clearly but multi-syllable words cause most difficulty. When the child talks quickly the condition becomes more marked. Sometimes a word is pronounced clearly and a few sentences later the same word is unintelligible. As may be expected consonants are far more severely affected than vowels or diphthongs. The consonants are misplaced, transposed or omitted. Substitutions are frequent and often the final sound of a word is left out. The following are typical examples:

1. *Sang* for *swing.*
2. *otomo* for *tomato.*
3. *sutter* for *supper.*
4. *enil* for *pencil.*
5. *yell* for *yellow.*
6. *poto* for *potato.*
7. *bobo* for *bottle.*

It is often surprising how the older child with this type of disorder does not recognise or hear his own bad speech—as if he had an auditory imperception for his own speech. He will hopelessly mispronounce a word he has just heard and appear satisfied with his effort. Yet it will be easy to establish that his hearing and interpretation of sounds are normal.

Such speech defects are normal in young infants when they are learning to talk. Parents will often imitate them and use the same mispronunciation when talking to the child. When the child is young this type of speech is considered "cute". When such speech persists and becomes a firmly established pattern, then anxiety results and help is sought.

Firstly an attempt should be made to establish the organic nature of the disorder. This is done by searching for evidence of a possible neurological cause which may have arisen in the birth process or early development of the child. In some cases even although there does not appear to be any obvious neurological evidence of brain damage, the history may suggest possible brain damage, e.g. delayed milestones, unexplained convulsions or behaviour disorder.

Speech Examination. This consists of an examination of the peripheral organs of speech to establish that there is normal function for all voluntary and involuntary movements, other than speech.

1. Establish that the tongue and lips function normally for smiling, kissing, "pulling faces", licking the lips, protrusion and diadokinesis of the tongue.

2. Test palate by examining the child's ability to swallow and by examining palatal elevation on stimulation or saying "ah".

3. Assess co-ordination of respiration and phonation by watching and listening.

The findings here will of course depend entirely on the site and degree of brain damage. From this point of view we may divide our patients into two groups:

1. *Those with very slight brain damage.* There may be no abnormal physical signs, although this is rare. A very careful search will nearly always reveal some abnormality, e.g., excessive clumsiness, slight wasting on one side with a change in reflexes, involuntary movements which are very slight, unestablished laterality.

2. *Those with obvious signs of brain damage,* e.g. the adult who has recently had a cerebro-vascular accident resulting in a hemiplegia with a unilateral facial weakness and no dysphasia. Speech may be grossly dysarthric and obviously the facial weakness alone cannot be responsible. This group can also include the child who has a spastic quadriparesis. The cranial nerves appear to function adequately on formal examination, yet speech is very indistinct. With these patients it is sometimes difficult to

decide whether the defect in neuro-muscular control of the lips, tongue and palate is responsible for the dysarthria or whether the lesion is situated at a higher level causing an apraxia.

If a patient is old enough, a search should be made for disorders of perception, reading and writing as these often co-exist with an apraxic condition. Where indicated a detailed assessment of intellectual functions should be carried out.

Finally, if clinical methods have failed to provide evidence of an organic lesion then one may have to resort to special investigations, such as an EEG examination. This may be more helpful and will sometimes reveal evidence of cerebral dysfunction when all other methods have failed.

Differential Diagnosis

It is evident that apraxic dysarthria must be distinguished from dyslalia and developmental dysarthria. The latter condition can be excluded by careful local examination when the cause of the dysarthria will immediately become apparent.

Differentiating dyslalia from apraxic dysarthria gives rise to greater difficulty. Most cases of apraxic dysarthria initially seem to be diagnosed as dyslalia and in this respect a question of terminology arises. Dyslalia has really almost the same meaning as dysarthria. Speech therapists have applied the term dyslalia to a group of children who articulate incorrectly due to one of the following five causes:

1. Persistence of faulty habits of articulation.
2. Imitation of faulty patterns of articulation.
3. The influence of defects of vision on articulation.
4. Mental defect.
5. Environmental and psychogenic factors.

In fact, only the first of these five groups give rise to diagnostic difficulty. The other four can be readily excluded with a careful history and examination. If the dysarthria is severe and persistent, then it is hardly likely to be due to faulty habits. If the dysarthria is mild then difficulty may arise. The most important aid in making the correct diagnosis is the finding of an organic neurological disturbance. Once this has been established then dyslalia or any other functional disturbance can be excluded.

Summary

The nature of apraxic dysarthria is defined and a short description of its manifestations has been given. The relationship between apraxia and apraxic dysarthria is discussed. Clinical manifestations and the diagnosis of the disorder are described in detail.

Opsomming

Die aard van apraktiese-disartrie word gedefineer en 'n kort beskrywing van die manifestasie van die toestand word gegee. Die verhouding tussen apraksie en apraktiese-disartrie word bespreek. Diagnose en kliniese manifestasie van die afwyking word meer volledig beskryf.

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