Cortical dysarthria and dysprosody of speech

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The nature and indeed the existence of a syndrome of cortical dysarthria has been the subject of considerable argument in the past 50 years: some of it acrimonious, and much of it dissolving into semantic discussion. The matter has been concisely reviewed by Critchley (1952) in considering articulatory defects in aphasia. There is no doubt that whatever the descriptive term used occasional cases occur in which a known and limited cortical lesion produces a change in speech which is largely confined to articulation and presents as dysarthria with little or no dysphasic element.

The case reported here showed just such a speech defect, and in addition a change in 'accentation' and melody (dysprosody) of speech, with alteration in the use of gesture which gave the impression of someone talking a foreign language. The cause of these symptoms appeared to be an intracerebral haemorrhage from a small angioma in the territory of the left middle cerebral artery. Angiography and operative treatment of the clot allowed an unusually precise localization of the lesion. The anatomical information and unusual clinical features are thought to justify a detailed report.

CASE REPORT

A 27-year-old healthy married woman noticed the sudden onset of numbness in the right hand while out walking about 4 p.m. one afternoon. An hour or so later she also noticed that her right face seemed weak and she drooled from that corner of her mouth. Others said that her speech was slurred, though it was intelligible and she used correct words. By 7 p.m., when she was admitted to hospital, she found herself unable to utter any words, even attempts at phonation producing no sounds. The numbness of the hand had decreased but voluntary movement was impaired. The facial weakness was also better and she no longer dribbled.

Subsequent enquiry revealed a past history of migraine from the age of 12 to about 23 years. Attacks were ushered in with flashing lights and stars in either field of vision, followed by headache, normally bifrontal and throbbing. Occasionally it was lateralized and had then occurred on both right and left sides. She also had frequent hay-fever and associated with this would get occasional transient headaches always localized to the left parietal region. She had had no other illness apart from the usual childhood ailments.

She was an only child. There was no family history of migraine, but her father had had severe hay-fever. He had died at the age of 35 of peritonitis. Her mother was alive and well. The patient had been married for six years and had no children. She had been educated entirely in England as had her parents and husband. She had learnt elementary French at school and had worked as a secretary until her marriage. She spoke no foreign language. She was right-handed and there was no family history of left-handedness.

On examination about four hours after the onset of her symptoms she was alert and fully cooperative but very frightened. She would make no attempt to speak or phonate but indicated answers to questions by head movements. There was no facial weakness but voluntary movements were impaired on the right. She could not protrude her tongue nor elevate the palate to order. There was some reduction of sensory appreciation both to light touch and pin-prick in the right arm. Although there was no apparent weakness in this limb in spontaneous movements, she was unable to carry out movements on request nor could she imitate them. When seen three hours later she could speak and used words correctly but speech was slow, hesitant, and slurred. She could now move the palate and swallow to order, but lip and tongue movements were incorrectly executed and she could not protrude the tongue or make named sounds on request.

Some 36 hours later she had no objective sensory abnormality at all. Spontaneous movements of arms, face, and tongue were normal, but there was still slight dyspraxia to requested or imitated movements of lips and tongue. Her rate of spontaneous speech was also now normal. However, she spoke with a foreign accent which several independent witnesses regarded as German. Her speech was also emphasized by gesture of the hands which was unusual for her. There was some dysarthric slurring of words, 'rs' being especially liable to mispronunciation, occasional misuse of syllables, and very occasional hesitation and apparent searching for the right word. Attempts at such standard phrases as 'West Registrar Street' and 'ancient and honourable institution' were definitely dysarthric. Nevertheless speech was free, appropriate, and completely intelligible. Its most striking characteristic was its apparently foreign accentation, an impression which was strengthened by the dysarthria. In naming a series of objects she made mistakes in about one in ten. They were usually approximations to the correct word, e.g., spring for string, bungle for buckle, watch-trap for watch-strap, etc., and mistakes were realized and attempts at correction made. There was no evidence of any receptive dysphasia as shown by replies to questions and the execution of simple and complex verbal commands. Reading aloud was correctly done and
she repeated the sense of the passage accurately. Writing
to dictation and copying of printed sentences showed oc-
casional mistakes—omission of words or syllables. This
was much less so in spontaneous writing. Simple mental
arithmetic was unimpaired. In written calculations, addi-
tion and subtraction, she made occasional mistakes, but
these were considered dysgraphic rather than dys-
calculic. Spontaneous drawings of a clock-face and a house
and copying of designs were correctly done. In summary,
she had a minimal motor dysphasia with slight tongue-
lip dyspraxia; but more marked abnormalities were a
dysarthria and a most striking change in accentation and
melody of speech. This condition remained substantially
unchanged until exploratory craniotomy 10 days later.

Investigations included normal findings in cerebro-
spinal fluid, skull radiographs, full blood count and sede-
mentation rate, and serology in blood and cerebrospinal
fluid. An electroencephalogram showed a slow wave
abnormality in the left fronto-temporal region. A left
carotid angiogram demonstrated a small angiomatous
malformation arising from an anterior branch of the
middle cerebral artery in the pre-Rolandic area and just
above the Sylvian fissure. It appeared to be superficial
and local vessel displacement suggested an intracerebral
clot (Figs. 1 and 2). These findings were confirmed by
operation, when Mr. J. M. Potter noted 'a little increased
vasularity in the immediate vicinity of the Rolandic
fissure, 1 cm. above the Sylvian fissure. . . . The small area
of cortex bearing the vascular malformation was com-
pletely excised. This measured approximately 1 cm. by
1 cm. by 0.75 cm. Beneath was a clot cavity 8 cm. antero-
posteriorly by 4 cm. by 4 cm. which was evacuated.'

Her post-operative course was eventful. She went home
eight days after the operation and at that time had no
abnormal dysphasia and no dyspraxia. She still spoke with
a foreign intonation and slight dysarthria. Her use of
gesture was still considered excessive for her. When
examined one month later, seven weeks after the onset,
her speech was normal in all respects. Her 'foreign'
accent had disappeared. Writing spontaneously and
dictation was also normal. She regarded herself as com-
pletely normal as did her relatives.

DISCUSSION

The study of aphasia is a fertile field for theoretical
and interpretive argument, in which the contents of
only do not always define their terms. The resulting
fusion is nevertheless more apparent than real. The
evidence is still convincing for two broad types of
speech defect associated with cortical lesions: one
which the trouble is predominantly motor or expres-
sive, the other where comprehension of words and
meaning is chiefly impaired. Each tends to be
associated also with a particular site of lesion:
the first a limited area at the base of the Rolandic
sulcus and the posterior end of the third and second
frontal convolutions; the second involving a rather
less defined area of the temporo-parieto-occipital
cortex. There is little to contradict and much to con-
firm the earlier views postulating Broca's and
Wernicke's types of dysphasia (Hécaen and Ange-
lergues, 1964). Further more detailed analysis cer-
tainly reveals a number of variants of both types, and
instances in which both expressive and receptive loss
are combined. It is to be expected also that if compre-
dension of words is faulty some aspects of motor ex-
pression will be secondarily affected, just as it will
if that part of the body scheme concerned with lip
and tongue is diseased. When, however, full anatomi-
cal information of the site of a lesion is available, and
this is unhappily infrequent, such mixed and vari-
gated cases still support the broad division, and
the last resort it is the evidence of relatively pure
types of dysphasia and their anatomical correlation
which is crucial.

A consideration of the cases of localized brain
wounds causing speech defects reported by Marie and
Foix (1917) and Russell and Espir (1961) lend ample support to this basic classification, even though the anatomical evidence in many of such cases is not as full as we would like. Within the group of mainly motor defects there are a number of cases in which throughout their disability or at some stage of it, the speech defect is best described as dysarthric. Marie and Foix used the term 'anarthric syndromes' for these: and in most severe examples there was initially a complete inability to utter words. Later, however, mispronunciation of words and the inability to master the French equivalent of our own test phrases for dysarthria made 'dysarthric' the more appropriate adjective. Marie (1906) originally insisted on a pure anarthria whose anatomical substrate lay in a 'lenticular zone' of frontal cortex and sub-cortex between the head of the caudate and the posterior end of the lenticular nucleus. This speech defect he considered had no necessary connexion with aphasia. The latter was for him always a comprehension difficulty and he regarded Broca's 'aphemia' as a mixture of anarthria and aphasia. The distinction is one of definition. Despite his previous view, in his joint paper with Foix in 1917 entitled 'Aphasias of war', anarthric syndromes are included. Henschens's (1922) violent objection to Marie's terminology seems to be largely semantic. If dysarthria is defined as due to a fault of bulbar innervation, then dysarthria from a purely cortical lesion must be a contradiction. But dysarthria is primarily a description of a mode of speech. In this sense some of Marie's cases are excellent examples of dysarthria. The same can be said of some of the cases recorded by Russell and Espir (1961) and Bay (1962): and in all three series the anatomical location of lesions is strikingly consistent. All are grouped towards the lower end of the Rolandic fissure, and most are rather more anterior than posterior to it. The term 'cortical dysarthria' for these cases seems descriptively accurate and appropriate. When it is used, it is common to segregate it from dysphasia proper (Bay, 1964). Such a usage implies, as it did for Marie, that dysphasia must by definition entail some lack of comprehension of the meaning of words. This is logical but obscurity may result if the term 'motor dysphasia' is also retained. Henschen (1922) suggested that Marie's use of anarthria was a source of confusion and should be abandoned. In fact it might add more to clarity if the term motor dysphasia were given up in favour of cortical dysarthria or dyspraxic dysarthria. It is the retention of this anomalous use of dysphasia that is confusing.

In the segregation of these two forms of cortical speech defect the recognition by the subject of mistakes in words and their attempted correction should be regarded as a crucial distinction. Anatomically speaking, if the cortical motor apparatus of speech is at fault, wrong words as well as mispronounced words may be expected, but as long as their fault is recognized no comprehension difficulty need be postulated. Thus what appears at first glance to be an amnesic or receptive dysphasia may in fact be a purely motor one. It is the inability to recognize the error that makes it a true defect of comprehension.

In earlier accounts of the motor types of dysphasia an association with dyspraxia of the lips, tongue, and sometimes pharynx and larynx, has been noted. Nathan (1947) reviewed six such cases in great detail and used the term 'apraxic dysarthria' to describe them. He emphasized that 'praxia' was the cortical motor function par excellence and suggested that apraxia was in fact the basic lesion in many cases of motor dysphasia. Critchley (1960) also discussed this aspect of aphasia, but concluded that articulatory dyspraxia was an independent entity, though it might coexist with dysphasia. The detailed protocols of many cases of motor aphasia contain records of labio-lingual dyspraxia even when this is not specifically mentioned. In dyspraxia voluntary and mimetic movement cannot be carried out, while similar movements may be perfectly executed as part of an involuntary motor sequence. Dysarthria due to dyspraxia might therefore be expected to show itself most readily in mimetic and requested speech: and this is in fact its most usual setting. When flow of speech is established, the dysarthria becomes less apparent and during the course of fluent speech it is when a word has to be voluntarily sought for that mispronunciation again occurs.

Another occasional association with motor aphasia is a disordered melody, rhythm, and accentuation of speech. Monrad-Krohn (1947) has termed this 'dysprosody'. It was noted and recorded in dysphasias before it was named. These cases may give the impression of someone speaking a foreign language. In Monrad-Krohn's much quoted example a Norwegian lady born and bred, who had never been out of her native land, was taken by her compatriots to be a German, because of the dysprosody following a stroke. Critchley (1962) gave further example of a similar change in English people, in whom the accent resembled that of a native Welsh-speaking person. Bay (1964) emphasized its constant association with dysarthric defects and implied that it was not uncommon in such a setting, though he appears at times to be describing something less subtle than a foreign accent. In its florid form it appears to be rare.

The patient reported here had some initial lip and tongue dyspraxia and for a short while a complete anarthria. As this recovered she had a dysarthria of speech which seemed to outlast any formally elicited
dyspraxia of lips or tongue. She also had for a time a
difficulty in using the correct word both in naming
objects and more occasionally in spontaneous speech.
There was also evidence of slight dysgraphia, and
this also had been preceded by a more extensive
dyspraxia of hand movements. It is noteworthy that
in each case she was at once aware of her mistakes
and made some attempt to correct them. It seems
reasonable to formulate her motor disability of
speech and writing as basically a dyspraxic lesion.

The other striking change was the dysprosody that
gave her speech a German accent. This was so marked
that other patients in the ward regarded her as a
foreigner. It was present when dysarthria was marked
and the two tended to clear together, though the
foreign accent was still obvious when dysarthria was
minimal. This phenomenon is doubtless capable of
further and more detailed phonetic analysis which
may show it to be part of a spectrum of basically
executive alterations in speech. For the present it
must be noted as an unusual and bizarre aspect of
motor dysphasia. It stands a little aside from the
dyspraxies since it is a constant phenomenon while it
lasts; and there is no equivalent here of a deficiency
of voluntary movement which can be performed
normally in other contexts, as is seen in dyspraxia.

The site of the lesion in the present case was well
established. The angioma from which bleeding
occurred was situated at the base of the Rolandic
area more anterior than posterior. Haemorrhage
appears to have been largely intracerebral and initial
cerebrospinal fluid showed no blood staining or rise
in protein. The clot cavity was subcortical and extended frontally for some 8 cm., thus undermining
part of the third and probably second frontal con-
volutions. It certainly impinged on Marie's 'zone
lenticulaire' and its position is similar to cases
described by Nathan (1947), by Bay (1962), and by
Russell and Espir (1961). It was confined to the
frontal lobe and there was no evidence of any more
posterior extension. This is worth emphasizing
because of the suggestion that dyspraxia may be
associated with lesions of the supramarginal gyrus
(Pussep and Levin, 1923). The final cortical defect
must have been at least the area 1 cm. by 1 cm.
by 0·75 cm. excised at operation. However, since this
was the seat of an angiomatous malformation before
any speech symptoms occurred, and its function was
therefore in part defective, it cannot be regarded as
necessarily subserving motor speech in her case. The
speech defect seems to have been produced by the
subcortical haemorrhage and consequent interrup-
tion of function of overlying cortex. Recovery of
function was finally complete. Rate of recovery was
rapid and is more likely to have been by resumption
of function in temporarily disturbed cortex than by a
take-over of function by corresponding cortex of the
opposing hemisphere. One can only say 'more
likely' until we know more of the time scales of
improvement by local recovery and contralateral
take-over. On present evidence this case lends some
support to Marie's view of cortical dysarthria, dependent on a lesion of the 'zone lenticulaire' and
Nathan's view that such dysarthria is essentially
dyspraxic in nature. It illustrates once more the need
for segregating the motor speech abnormalities
of cortical lesions from those involving comprehension.
Such a segregation should be maintained in the
terminology of the subject and cortical dysarthria
or dyspraxic dysarthria would be suitable terms
to describe many such motor changes. The term motor
dysphasia is best avoided as its past usage renders it
meaning equivocal.

It is of incidental interest to note that this woman
suffered from migraine, but that its aura bore no
anatomical relation to her one established vascular
lesion. The presence of other lesions in the verebro-
basilar circulation is not excluded by her carotid
angiogram.

SUMMARY

A case is reported of intracerebral haemorrhage
from an angioma at the base of the fissure of Rolando
causing transient cortical dysarthria and dysprosody.
The case is taken as evidence in support of the occurre-
ence of a cortical dysarthria, basically dyspraxic in
nature and due to a strictly localized frontal lesion.

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with the operation findings.

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