OBSERVATIONS IN HYPOGLYCAEMIA: V. DISORDERS OF SPEECH.

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During the hypoglycaemia produced by insulin for the treatment of psychoses, disorders of speech occur as symptoms of the dissolution and restitution of cerebral function. Although transitory, they can be observed accurately, as the hypoglycaemic condition is repeated daily for weeks in the same patient. They have in fact been studied and commented on by several workers since the method was initiated by Sakel in 1934; we shall refer later to some of the findings and views in the literature. In this paper an attempt will be made to describe and classify the various speech disorders, and to relate their characteristic features to other hypoglycaemic symptoms occurring at the same time. Attention will be drawn to certain speech-like symptoms which hitherto have been scarcely noticed. The possible influence of the underlying psychosis will also have to be considered.

Like the motor phenomena in hypoglycaemia, the nature and duration of speech anomalies vary a good deal from patient to patient, but the individual patient has his pattern of symptoms, which is reproduced in every detail in each treatment.

1. Loss of Speech and Isolated Dysphasias.

In some cases no abnormal speech or sound is produced during the whole hypoglycaemic condition. The patients quietly lapse from sleep into deep coma, the transitional period being relatively short. On waking up, consciousness is regained quite quickly and the patient seems in full control of his speech.

In a second group, speech function is dissociated from consciousness. Motor speech seems earlier affected by hypoglycaemia, and returns later to normal function than both consciousness and other motor behaviour. The patient appears dumb at a time when his eye movements still follow the observer; while he can carry out on request movements of his limbs, face and even tongue, he is, he reports afterwards, unable to speak. Occasionally we saw this speechlessness remain for some time after all other signs of hypoglycaemia had disappeared. That the patient was fully conscious is proved by his clear recollection of this condition. If speechlessness occurred in a setting of general immobility, it was probably part of the syndrome of "akinetic mutism" discussed by one of us in a former paper (Mayer-Gross, 1943), and related to a subcortical mechanism. To differentiate clinically this picture from catatonic mutism often seemed impossible. As most of our patients were schizophrenics a predisposition to catatonic reactions related to the underlying illness could not be excluded, but it was also observed in cases that never showed catatonic features in their psychosis either before or afterwards.

That remaining speechlessness can be of the nature of a motor aphasia of cortical origin was proved in one of our cases, a Norwegian, who remained mute after waking up from coma. He got out of bed, dressed himself, moved about freely and understood what was said to him. After half an hour he began to answer in Norwegian, seemingly unaware that nobody could understand him. Only after another hour did he regain his control of English, which he ordinarily spoke very well. In one of a group of Polish soldiers treated together a similar observation was even more characteristic and better understood—thanks to the help of his compatriots. During the stage of waking from coma the patient began to shout and sing in Polish, repeating the same phrases and swear words, some of them uttered in a distorted paraphasic form. He then spoke to the nurses in his mother

tongue as though expecting to be understood. He did not make use of his small vocabulary of English for some time. Both cases thus demonstrated the earlier recovery of the mother tongue in bilingual aphasics. One might speculate whether the same mechanism was operating when incoherent schizophrenic language reappeared during the awakening of those patients in whom it had formed part of the original psychotic picture. When they were entirely awake the " word salad " disappeared. It was a transient revival of psychotic symptoms similar to the return of hallucinations or paranoid episodes so frequently observed in the post-comatose stage.

Periods of auditory (sensory) aphasia are much more difficult to verify, since the clouding of consciousness prevailing in hypoglycaemia interferes with perception in general. Many self-descriptions are suggestive of a suspension of speech perception at a time when consciousness is apparently restored and sounds are clearly perceived. One of Sakel's patients describes a condition reminiscent of word-deafness: " I saw you perfectly clearly and heard you talk and recognized your voice, but it was impossible for me to understand what you wanted of me or what you were asking for . . . later, even though I understood you, I could not find words to answer your questions sensibly. Then I realized that I was not telling you the things I wanted to say . . . "

Jargon paraphasia, as seen in sensory aphasics with a permanent cerebral lesion, seems to be rare in hypoglycaemia. The majority of paraphasic productions were similar to those in normal dreams, and therefore were probably part of the disorders due to clouding of consciousness.

2. Speech Disorders Accompanying Disturbances of Consciousness.

(a) *Dysarthria and Kindred Symptoms.*

Speech anomalies coincident with and dependent upon clouding of consciousness are much more common than disorders outlasting the general hypoglycaemic condition. If they are not manifest in spontaneous utterances by the patient, they can be easily elicited. This is more easily done during the waking stage than when the patient is going into coma. During the latter, if the patient answers at all, he seems to control his speech relatively well. Speech definitely lags behind, and is slow in recovering during the much shorter stage of awakening. The main symptoms are those seen in other states of reduced clearness of consciousness, dysarthria, slurring, paraphasic condensation of words, clang associations, iteration and echolalia.

Dysarthria is the most common. If one addresses the patient at the right moment he answers with the faulty articulation of the so-called bulbar type, slurring and omitting consonants and syllables. Many patients also perseverate, repeating the same word or phrase whatever the question. The similarity of the behaviour with that in alcoholic intoxication is often striking because of the accompanying elated mood. Like the drunkard, some patients seem to get playful enjoyment from the testing of their disability. Spontaneous speech in this stage is of the same type.

In cases of relatively quick recovery, e.g. after intravenous administration of glucose, echolalia is a very frequent symptom. For about five minutes the patient seems forced to imitate every word and sound in the room without dysarthria or other distortion, but also without proper apperception and understanding—as far as can be judged from his imitation of foreign words. The moment he is fully conscious the symptom disappears without an intermediary stage of dysarthria or perseveration. From its observation in hypoglycaemia its mechanism seems to differ somewhat from that of the other speech disturbances mentioned above. Perhaps it is produced at another level of lowered consciousness.

If proof were needed that the speech disorders described in the last paragraphs are in fact produced by a general reduction of cortical function or, in psychological terms, by clouding of consciousness, it would be provided by identical observations in clouded states of various origin—epilepsy, head injury, and drug intoxications of many kinds. Hemphill (1940) for instance has noted them after convulsions induced for therapeutic purposes. Detailed studies by Bonhoeffer (1898), Symonds (1937), Stengel (1937) and others have shown that apperceptive and cognitive functions including thinking are reduced parallel with speech to a lower level. To

illustrate this with an example in hypoglycaemia: A patient questioned in the waking stage takes in one word, e.g. " sister," and repeats for a few minutes " good sister " without grasping the whole question. Then he grasps another detail of the situation, say the blue sky seen through the window, and now perseverates the words " lovely day." All mental processes seem equally restricted, the lack of verbal expression corresponding to the narrowing of the perceptive and cognitive field. If pressure of speech is present, scarcity of thought produces reiteration; the same effect may be due to a desire to say something he cannot formulate in words. In short, it is often very difficult for the observer to decide to what extent the patient's failure in language is caused by disorder of speech, of cognition or of thinking.

The character of the disturbance caused by clouding of consciousness is well illustrated in the following verbal report of the waking-up behaviour of one of our patients, a former fighter pilot. The fragmentary emotional reactions in this condition are also shown:

Patient sits up in bed shedding tears. " I never cry—I never cry—I just had some incendiary—incendiary." Doctor touches his legs in sitting down on the bed. " Am I disturbing you?—I just put my feet behind there—I just crashed behind there—there is a fellow over there, pardon me, sergeant, fly through that window, straight through the glass, sergeant, surgeon, drudgeon—good morning, sister." Smiles at nurse and begins to sing. " It doesn't matter, my God—quite, quite, bite, bit, bit, bit—listen, listen, I am delected—" (delighted?). " Why should I be delighted? Doctor, director, what for? Why doctor, doctor!" (please, wake up!). " Sir, how many letters are in that?—in that letter—Oh God, you should be sorry for me. I am very sorry for you because I am inspirational for you—do I say, see, say—I say things—I want things—a drink, a drunk—well let's have some fun." A few minutes later the patient was completely awake, clear and controlled, and gave rational answers.

The speech disorders connected with the dimming of consciousness in hypoglycaemia showed no relation to the underlying illness of our patients. They were mostly suffering from schizophrenia, a disease in which disturbances of consciousness are rare. Semistuporous catatonics, however, sometimes show echolalia. There was no instance of echolalia being a symptom of the psychosis among the many patients who had echolalia when hypoglycaemic.

(b) *Articulate Ejaculations Associated with Myoclonisms.*

More specific for hypoglycaemia and closely connected with its other motor symptoms are certain automatic speech phenomena. They are found only in the hyperkinetic stage, but it should be pointed out that hyperkinesis in hypoglycaemia is not always associated with production of speech and sounds. Two main groups of movements stand out among the varied motor symptoms: the so-called myoclonisms and the movements of the " extrapyramidal," choreo-athetotic type. Myoclonic twitchings are short rhythmic jerks of limbs or of the whole body. They are obviously of cortical origin, being identical with the motor reactions observed when certain parts of the cortex are stimulated by the electric current during an operation. Beginning in the arms, sometimes in the face, the " march of spasms " spreads over all the extremities until the whole patient is shaken by regular jerks. One side of the body may be affected first, so that for instance only the right facial muscles and the right arm may twitch at the start. The movements may continue for a considerable time; but more frequently an interval of rest lasting a few minutes is followed by another wave of twitches, and so on. The symptom is reminiscent of what has been described as epilepsia partialis continua (Kojevnikov, 1895). Myoclonic twitchings are often the forerunners of a generalized epileptiform fit.

The movements are frequently accompanied by verbal ejaculations of an articulate character. Speech and jerks are produced in the same rhythm, the words becoming louder with the increasing myoclonisms, both apparently the result of the same cortical discharge. As a rule the ejaculations appear independent of the anatomical distribution of the twitchings, but on a few occasions we observed them together with twitchings of the right face and right arm only. Many times the articulations, increasing in strength with the myoclonic movements, culminated in a scream which marked the beginning of an epileptiform seizure.

There was a great variety of words and phrases associated with myoclonism, most of them being of the nature of the "emotional language" of H. Jackson. Exclamations like "My God," "Oh God," "Christ," "Hell," "Oh shucks," "damn" and all the obscene swearwords were common; also "yes—yes—," "no—no—"; and short phrases like "I love you," "good work," etc. One patient regularly repeated the word "white" as soon as he started twitching, another the word "dial," both being unable to account for it when questioned afterwards. The reason for the choice of word or phrase in the special case remained obscure, as did the reason why speech production was present in one patient and absent in another though both showed the same motor phenomena. It was never observed that a word heard or spoken by the patient in the initial stage of twitching was picked up and reiterated. In each case the same word or phrase reappeared every day signalling the myoclonic stage of the patient's hypoglycaemic condition.

If there were no culmination in an epileptiform fit, myoclonic twitchings and ejaculations either disappeared with deepening of the coma, or with the patient awakening through the adreno-sympathetic counteraction caused by muscular exertion. In the latter case he quickly regained his normal speech. During this lucid interval we tried repeatedly to get information regarding his experience, ideas or feelings in the stage of twitching and verbal ejaculations, but with no result. Although some patients seemed uneasy or even embarrassed when questioned about it, they neither remembered what they had uttered nor what they had experienced at the time.

(c) *Primitive Utterances Associated with Choreo-athetotic Movements.*

Movements of the choreo-athetotic group, including torsion movements, are very common in the precomatose stage of hypoglycaemia. They closely resemble the hyperkinetic symptoms in lesions of the basal ganglia. The accompanying utterances are of a much more primitive character than those related to myoclonic twitchings. As they are entirely inarticulate it is doubtful whether they can be described as speech; but they must attract our attention as precursors of speech development because of their similarity to the phonations of the infant in its first year. Some could be described simply as sighing, moaning or groaning, while in other cases their resemblance to the babbling, cooing and gurgling noises of the baby was most striking. They consisted of the typical inarticulate lallation, like ba-ba- or la-la, or in the vowel "ah" only, sounded in regularly varying pitch. Equally frequent were cries and yells, also best described by comparison with the cries of the hungry suckling. These sounds had not the rhythmic jerky character of the articulate words associated with myoclonisms even when they were repeated. The picture of regression was in many instances completed by rocking movements of the head and body, by kicking, pushing, and throwing about of the arms and legs characteristic of the corresponding early stage of life. Here, however, it was seen in a grown-up person deprived of cortical function by hypoglycaemia.

Other sounds produced in this stage of hyperkinesis cannot be adequately described by human comparisons. They suggested voices of animals, crying monkeys, howling, roaring, barking, quacking or croaking. The visitor to an insulin ward in which were several patients in this stage was reminded inevitably of the noise heard in a menagerie at feeding time. Some sounds defy description and could only be represented by sound recording instruments—a method holding out great promise for the analysis of speech disorder in general. Animal sounds if associated with "climbing movements," as described by one of the authors (M.-G.), are suggestive of regression to another low level of integration.

The similarity of these sounds to those of infants or animals was accentuated by their peculiar emotional appeal to the listener; they seemed to express instinctive feelings mainly of an unpleasant kind. Furthermore, while myoclonic phonations could not be influenced by cutaneous stimuli, the "choreo-athetotic" cries became louder and more urgent in response to pin-pricks or other painful stimulation.

In studying these inarticulate sounds it was noticed that it was rare to have only one noisy patient in the insulin ward at one time; his noise seemed to provoke noisy behaviour in other patients, especially in those who were in the same stage of hyperkinesis. One recalled the imitation of crying among babies in a crèche, or the contagiousness of yelling, barking, etc., among a herd of animals. But while

infants awaken one another by their cries, and animals respond to the soundings of their kind with lucid sensorium, the observations in hypoglycaemia seem to point to a possible " subconscious," subcortical intercommunication in man. We have certainly seen cases which had hitherto been quiet in their hyperkinetic stage emitting sounds in the same stage from the day on which another patient started ejaculations of the " subcortical " type. Should this observation be confirmed it might explain the changes between the prevailing quiet and restfulness of some days and the noisiness of the same patients on other days at approximately the same hour. A Swiss worker commenting on these changes suggested that meteorological factors might be responsible.

Naturally, little could be ascertained about the patient's own experiences in the stage of primitive sounds; but whereas the articulate ejaculations associated in the myoclonisms seemed entirely forgotten, patients waking from this stage frequently apologized for their noisy behaviour. One female patient was seriously distressed about having disturbed the others by her screeching. She wanted to discontinue her treatment. This preserved memory of the primitive sounds may be due to cortical functions recovering before the subcortical. Consciousness thus looks on as a spectator, and registers the subcortical symptoms still present at the moment of waking up; but symptoms of irritation of the cortex itself, like the sounds associated with the myoclonisms, cannot be recalled.

Discussion.

The inhibition of cell oxidation caused by hypoglycaemia affects first and foremost the cerebral cortex. Parallel with the impairment of clearness of consciousness, indicating, for all we know, a general cortical involvement, speech is disturbed because it is one of the highest cortical functions. In cases without manifest abnormal speech production the loss of speech is an important symptom of depressed cortical function. There is no reason to assume that certain areas of the cortex are more sensitive to the reduced glucose level and therefore earlier affected. To explain cases of aphasia supervening in the waking state as described in the first part of this paper no recourse to localization is needed. The observations can be explained by the principle that the most differentiated of the disturbed functions returns later to normality. The same principle applies to the cases in which dysarthria and kindred symptoms continue for a short time while the patient regains his full consciousness.

The concept of consciousness has been for a long time a bugbear of certain psychological schools. Lately it has been pilloried again as a " crude metaphor," a " misleading one-dimensional abstraction." Paterson (1944) discussing confusional states after closed head injury has pointed out that " circumscribed and localizable functions " are disturbed in these cases, and maintains that he can dispense with the concept of a general disturbance of consciousness. However that may be, the clinical picture of hypoglycaemia cannot be adequately described without recourse to a unitary concept signifying degrees of deviation from the waking state. For lack of better terms, " dimming," " clouding " and " loss of consciousness " have to be used.

It is not an entirely satisfactory concept even so. Paterson's criticism of the quantitative one-dimensional idea of consciousness and its disturbances can be understood in the light of observations like the following: There are in hypoglycaemia at least four types of clouded consciousness, differing from each other not only in degree but also in kind, if their association with different speech phenomena is used as a distinguishing criterion. The clouded condition in which dysarthria and kindred symptoms are present seems unlike that in which echolalia appears. The latter seems to be nearer the waking state. We find a third mode of disturbed consciousness associated with myoclonic twitchings and rhythmic articulation, both probably symptoms of cortical irritation. The clouding here is certainly deeper than in the stages of dysarthria and echolalia, but it seems doubtful if it can be differentiated by degrees of lesser or greater depth from the fourth type associated with " extra-pyramidal " hyperkinesis and primitive sounds. If, however, memory is used as a gauge for measuring the degree of unconsciousness, the stage associated with echolalia and myoclonisms must be considered more

severe than that in which dysarthria and primitive sounds appear, since only the latter leave some traces behind.

The paradox of these observations and the problem of consciousness in general cannot be solved at present. If we knew more about the functions of different cortical layers, or if recording of electrical potentials could register more refined differences of function, we might perhaps be able to arrive at a better understanding of the underlying mechanisms and of the meaning of discriminations which at the moment are purely descriptive.

The view that hypoglycaemic metabolic changes extend equally over the whole cortex is not shared by Angyal (1937). His theory of speech disorders and other motor symptoms is worth discussing, because it seems to be the only attempt at linking the various symptoms in hypoglycaemia with the underlying schizophrenic illness of the patients under treatment. According to Kleist's (1925) much debated localization of schizophrenic symptoms, it is the parietal area of the cortex which is affected in schizophrenics showing disturbances of thinking and speaking; while catatonic features, mannerisms, etc., are supposed to be due to affection of the frontal region. Angyal, following Kleist's theory, assumes that whichever region is mainly diseased will show most reaction in hypoglycaemia. He thus distinguishes two speech syndromes in hypoglycaemia, the "paralogical and sensory-amnesic" and the "ontogenetic-aphasic." The former corresponds to the group here described as "dysarthria and kindred phenomena." From the author's own detailed record of these speech productions there is no doubt that they are not specific for schizophrenia, but identical with the dysphasia in conditions of clouded consciousness. Angyal's ontogenetic syndrome, however, consists mainly of the regressive and primitive utterances associated with extrapyramidal movements. The presumed correlations with certain types of schizophrenic illness seem to us purely hypothetical, and were not confirmed by our observations. We frequently observed in the same case primitive utterances during coma, and dysarthria, etc., in the waking stage.

Angyal's localization of the "ontogenetic-aphasic syndrome" in the frontal region is closely related to his theory of the oral movements discussed in a former paper (Mayer-Gross, 1941). Mouth movements being so common a symptom of hypoglycaemia, it is not surprising to find them sometimes associated with primitive utterances. In the ordinary sequence of events, however, they make their appearance in an early stage of hypoglycaemia without any sound or speech production. Much later, when the disturbance of consciousness deepens, primitive utterances appear together with a general hyperkinesis of the choreo-athetotic type which sometimes also extends to the oral region. Because of their association with symptoms of the basal ganglia and their similarity with sounds of the infant before complete myelinization of the cortex, we are bound to interpret the primitive utterances as release phenomena after cortical inhibition, and not as signs of irritation of certain cortical areas.

If this interpretation is accepted it can throw some new light on the cerebral functions underlying normal speech development. The instinctive ability to produce primitive sound signals, which can appear in adults if cortical function is depressed, may well have played a paramount role in the development of the human language. Liepmann's frequently quoted simile that speech has grown as a parasite on the feeding function of the mouth region will have to be revised if there is a primary urge to utter sounds after decortication. Surely this subcortical instinctive ability may well be the basic function from which human language starts its development—speaking metaphorically, the wild stock on which language is grafted.

As to the articulate ejaculations associated with myoclonisms, there is little doubt that both symptoms are due to a cortical discharge mechanism. The observation that they may culminate in the scream ushering in a major epileptic fit makes it doubtful whether the traditional explanation of this epileptic cry meets all the facts. It is usually considered to result from a forced expiration initiating the tonic phase of the fit, i.e. as of purely mechanical origin. Kinnier Wilson (1940) maintained that forced inspiration was responsible for the initial cry. There are several reasons for calling these explanations in question. It makes it difficult, for instance, to account for the absence of the cry in a great number of cases which otherwise show all the consistent regular features of the

major epileptic attack. Furthermore, since therapeutic application of epileptiform seizures has made observation easier, it has been confirmed that epileptics occasionally scream well in advance of the general tonic contraction of the muscles, especially those controlling respiration. In electrically induced convulsions this sequence of events is very common. The observations of hypoglycaemia provide another argument against the purely mechanical origin of the epileptic cry, and in favour of its causation by a cortical irritation. Why the epileptic discharge includes the motor speech area of the cortex in some cases only remains unknown.

Summary.

I. The paper gives a description of disorders of speech caused by impairment of cerebral function in hygoglycaemia. Besides total loss of speech in deep coma, the following disturbances could be observed mainly during the stages of going into and waking from coma:

(1) Isolated dysphasias of the motor and probably of the auditory type.

(2) Dysarthria and kindred symptoms closely related to clouding of consciousness. They are identical with those observed in clouded states of other aetiology. Echolalia, one of these symptoms, is frequent in hypoglycaemia.

(3) Articulate rhythmic ejaculations occurring together with myoclonic twitchings of cortical origin.

(4) Primitive utterances, similar to the lallations or cries of the human infant or to the sounds produced by animals, voiced in the stage of choreo-athetotic hyperkinesis. They are considered to be of subcortical origin released by the inhibition of the cortex.

II. The cerebral mechanism of these disorders is discussed with reference to—

(1) Speech as the most highly differentiated function of the cortex.

(2) Consciousness and its disturbance as a concept signifying deviation from the waking state.

(3) Attempts at localization based on the underlying schizophrenic symptoms (Angyal).

(4) The significance of subcortical sounds for the theory of speech development.

(5) The origin of the epileptic cry.

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