Confusion and Disturbance of Speech from Stimulation in Vicinity of the Head of the Caudate Nucleus

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The effective treatment of certain disorders of movement by lesions in the thalamus and pallidum has opened a new chapter in human neurophysiology. Since the physiological verification of the anatomical placement of the surgical instrument in the depths of the brain forms an important clinical safeguard, such studies form an integral part of the surgical protocol. From an investigational point of view, this research is hampered by the lack of direct anatomical proof of the sites of stimulation, and indirect measurements derived from various ventricular landmarks must be accepted as a substitute. These in turn are subject to individual anatomical variations which are mentioned below and have been reviewed in greater detail elsewhere.25

The present report concerns observations made with electrodes placed in the mesial segment of the globus pallidus and the inferior portion of the nucleus ventralis lateralis thalami for study of these areas prior to treatment of disorders of involuntary movement. The electrodes were inserted through a burr hole well forward in the frontal region and passed through or close to the head of the caudate nucleus. Since the electrodes were provided with stimulating points at regular intervals along the shaft, the caudate nucleus and its vicinity could be studied by stimulation.

Method

The following 10 patients participated in the present study:

E.B. Male, 58 yrs. Right hemiparkinsonism for 2 yrs.

T.B. Male, 51 yrs. Severe progressive tremor of right hand for 1 yr. Present with voluntary movements but absent at rest.

A.C. Male, 59 yrs. Right hemiparkinsonism for 2 yrs.

J.C. Female, 9 yrs. Progressive dystonia of both legs and the right arm for 2 yrs.


E.S. Female, 52 yrs. Progressive dystonia affecting the trunk and left extremities for 20 yrs.

I.S. Male, 58 yrs. Bilateral parkinsonism for 6 yrs.


V.S. Female, 62 yrs. Right hemiparkinsonism for 2 yrs.

H.T. Female, 53 yrs. Bilateral parkinsonism for 8 yrs.

All of the patients were right-handed by test and history. None had suffered from epilepsy with the exception of E.S. who had a 4-year history of occasional brief periods of confusion and sharp waves were recorded from the right temporal region by electroencephalography.

In order to test the accuracy of the stereotaxic placements physiologically, depth electrodes were placed in targets of possible therapeutic value. After demonstrating the positions of the anterior and posterior commissures as well as the midline of the third ventricle by pneumography, a burr hole was placed just behind the hair line in the frontal region about 2-3 cm. from the midline. One electrode was directed to the medial segment of the pallidum at R or L 15, I 2 and P 6 (according to the coordinate system to be mentioned below) and the other to the basal portion of the nucleus ventralis lateralis. In the latter instance, the coordinates employed varied from time to time but in general fell in the vicinity of R or L 10, S 0 and P 15. The electrodes were retained in position for a period of 7 to 10 days.

The electrodes (Fig. 1) consisted of 22-gauge "Teflon" tubing down the bore of which was threaded .006 inch stainless-steel wire insulated with Kel-F plastic. At sites selected as areas of stimulus the wire was led out a small hole in the side of the tubing, passed around the tubing, then back into the same hole so that the stimulating area was a wire ring about the outside of the "Teflon" tubing. In order to present a smooth outer surface, the "Teflon" tubing was coated with "Tygon" (U. S. Stoneware Co., Akron, Ohio)
which was cleared away from the wire of the ring electrodes. Seven rings were used on the electrode at 2, 4, 12, 14, 22, 32, and 42 mm. from the tip. Since the electrodes were directed to the medial segment of the pallidum and the basal portion of the nucleus ventralis lateralis, most of the responses in the present study were obtained from stimulating between rings 22 to 32 mm. or 32 to 42 mm. from the tip of the electrode. Despite a distance of 10 mm. between the stimulating rings the responses by stimulating between the rings generally were more discrete (and this method was used generally) than if one of the rings was used with the indifferent electrode placed under the buttocks. The resistance between two of the electrodes with an ordinary battery-operated ohmmeter lay in the vicinity of 40,000 to 50,000 ohms.

Stimulation was carried out with unidirectional 2.5 ms. square waves at 60/sec. frequency. None of the stimuli was followed by an overt motor seizure. During the stimulation the wave form and the current and voltage were observed directly with appropriately placed oscilloscopes on the output circuit. The impedance of the patient was estimated for each stimulation by having the stimulating current run continuously through a calibrated variable electrical resistance which was adjusted until the amplitude of the wave shown on the oscilloscopes did not change when the patient was substituted for the mechanical resistance. When the current is given, peak current is implied. The resistance for the particular current employed is given in thousands of ohms (K). It will be noted, in general, that at the levels used in the present study the resistances lay in the vicinity of 3 to 5 K as opposed to the 40–50 K obtained with the low-current meter cited above.

For charting purposes, a coordinate system was used which has been described elsewhere, and the basic zero planes were defined as follows: 1) Sagittal: A vertical plane bisecting the third ventricle and the brain stem. 2) Horizontal: Perpendicular to the sagittal plane and passing through the centers of the anterior and posterior commissures. 3) Transverse: Perpendicular to both the above planes and passing through the center of the anterior commissure. All coordinates are labeled right (R) or left (L), superior (S) or inferior (I), anterior (A) or posterior (P) with respect to these planes. The numbers refer to distance in millimeters. The broad lines shown on Figs. 2, 3 and 4 connect the coordinates of the two points of stimulation used in the particular instance. When the electrode lay oblique to the plane of the sagittal chart (as most of them did) and it was necessary to use two adjacent charts to show both ends of the pair of stimulating electrodes, a tear-drop symbol is used with the pointed end indicating the direction in which the second electrode of the pair lay. In plotting points, loci 8–12 mm. from the midline were recorded on the chart depicting the brain in sagittal section 10 mm. from the midline. In a similar fashion points at 13–17 mm. were drawn on the 15 mm. chart and points at 18 and above were drawn on the 20 mm. chart. The error caused by individual anatomical variation in this region is considerable as measured from the zero planes defined above and 2 standard deviations amount to about 4–5 mm.

**Diagram of sagittal section of brain 10 mm. from the midline upon which electrode coordinates from 8–12 mm. from the midline are plotted.** The heavy vertical axis passes through the lateral projection of the anterior commissure and the heavy horizontal axis passes through the lateral projections of the anterior and posterior commissures. The squares measure 1 cm. Where both electrodes of a stimulating pair are included in the plot, the interval is indicated by a broad bar. When only one of the electrodes is included, it is indicated by a tear-drop symbol and its mate is indicated by a similar symbol pointed in the opposite direction on an adjacent diagram. The patient’s initials may be compared with descriptions in the text.

**Fig. 1.** The flexible depth electrode used in the present study showing the small ring at the end which fits over the beak of the electrode guide shown below and the distal 5 ring electrodes. A millimeter scale is included below.

**Fig. 2.** Diagram of sagittal section of brain 10 mm. from the midline upon which electrode coordinates from 8–12 mm. from the midline are plotted. The heavy vertical axis passes through the lateral projection of the anterior commissure and the heavy horizontal axis passes through the lateral projections of the anterior and posterior commissures. The squares measure 1 cm. Where both electrodes of a stimulating pair are included in the plot, the interval is indicated by a broad bar. When only one of the electrodes is included, it is indicated by a tear-drop symbol and its mate is indicated by a similar symbol pointed in the opposite direction on an adjacent diagram. The patient’s initials may be compared with descriptions in the text.

**Results**

A. **Responses to Stimulation.** Because of the use of implanted electrodes time could be
taken to be certain that the patient was at ease and fully cooperative at the time of stimulation. The sessions of stimulation also could be repeated as often as was necessary to assure the examiner of the validity of the responses. Although the patient was aware that stimulation would be carried out, the use of mock stimuli and rotation of the points of stimulation without the patient's knowledge permitted rapid assessment of the patient's reliability. In the following descriptions the peak current and resistance are given as they were observed for each stimulus. The stimulus usually was applied for 5 to 10 sec. The coordinates of each of the electrode rings used for the stimulus are given according to the coordinate system outlined in Methods and as illustrated in Figs. 2, 3 and 4. Because of the length of the original notes, the following protocol has been abridged for this presentation. In general, the responses to stimulation fell into two categories, namely those limited to the duration of the stimulation and those outlasting the stimulation.

1. Responses Limited to Duration of Stimulation.


During stimulation he repeated "measurements" spontaneously several times. (The word had been used by the examiner shortly before.) Later when asked to count, he stopped 10 sec. after the start of the stimulus. When asked why he did so he replied that his arms felt "tight." During a later stimulus, he repeated spontaneously "sixth column" several times.

He counted for 3 sec. after the onset of stimulation then stopped and put out his tongue. This was repeated 3 times. Later he could not recall having put out his tongue.


Arrest of speech was obtained. Later he explained it by saying, "Like I couldn't count very fast."

Case H.T. 9/14/61 R17 S12 A8 to R19 S20 A14. 12 ma. 5.1 K.
She made a humming noise in her throat with the first few stimuli which she denied later. When stimulated during counting her voice became pitched higher and enunciation was slurred. Occasionally, after several seconds of stimulation, contraction of the lower left face and turning of the head and eyes to the left might appear.

9/18/61 same electrodes as above. 10 ma. 3.9 K.
She stopped counting during the stimuli. No respiratory arrest. She reported after several stimuli, "I have dizziness and a floating feeling. I feel I don't have everything under control. I can't speak properly and I make faces. It's a little frightening."

Case L.St. 11/20/61 R16 S15 P3 to R18 S23 A5. 7 ma. 6.5 K.
Stimulating during counting caused him to stop and he explained later, "My voice fails on me." During the stimuli there was slight deviation of the eyes to the left. Later when he was instructed to keep his eyes deviated to the right, he

FIG. 3. Diagram of sagittal section of brain 15 mm. from the midline upon which electrode coordinates 13–17 mm. from the midline are plotted. Conventions used are given in Fig. 2.

FIG. 4. Diagram of sagittal section of brain 20 mm. from the midline upon which coordinates 18 mm. and more from the midline are plotted. Conventions used are given in Fig. 2.
was able to maintain them in this position during the stimulation. There was no alteration in pupillary diameter.

Case J.C. 12/13/61 L16 S4 P6 to L20 S12 P1. 9 ma. 1.5 K.

On stimulation she reported a “mild” sensation in the right side of the face and down the right arm to the hand. She stopped counting during the stimuli but continued to move and smile. Later she was urged more strongly to continue counting but was unable to do so. She could not explain this except, “Perhaps I wasn’t trying hard enough.”

L20 S12 P1 to L24 S19 A5. 7 ma. 2.9 K.

She could not continue to count during the stimulus and later reported, “A real soft feeling in my cheek” and indicated the right cheek. On later repetition she denied any sensation but was again unable to count. She was able to hold out her tongue during the stimuli and could execute lapping movements despite arrest of regular speech at this level of current. During one arrest of speech she once forced out a number she had already given when urged strongly to continue to count. At the end of the stimulus she asked, “Why did I count backwards?” At another time when urged to continue during an induced arrest of speech, she repeated, “Hush, hush, hush . . . .” No facial contractions or deviation of head or eyes occurred with any stimuli.

Case V.S. 12/17/61 L16 S15 A0 to L19 S22 A7. 8 ma. 7.9 K.

There was transient inhibition of speech during the stimulus but she resumed counting with the stimulator still on.

Repeated at 9 ma. 6 K.

Here there was well defined arrest of speech. She could hold the tongue out motionless during the period of inhibition of speech. There was definitely no motion of face or tongue that would interfere with articulation.

L18 S0 P6 to L16 S15 A0. 10 ma. 4.2 K.

During stimulation she stopped counting and her eyes turned to the right. When asked why she stopped, her response could not be understood. When later asked again, she said, “I thought that was enough.” When the stimulus was repeated, she started to count backwards, then stopped entirely and turned her eyes to the right. When asked why she had stopped she replied, “I thought it was time to stop.” In the presence of additional observers when she was urged especially to continue to count she was able to do so at this level of current with slight hesitation. During these stimuli her eyes turned to the right while she continued to count.

Liminal stimuli on 13/18/61.

It was demonstrated that below the current required for complete arrest of speech, her eyes were turned to the right and her voice was raised in pitch and sounded muffled during the stimulation. During this session stimuli producing turning of the head and eyes to the right and arrest of speech also caused her to cease repetitive movements of the hands or if she was told to hold her tongue protruded she would withdraw it slowly during the stimulation.

Case E.B. 2/20/62 L18 S29 A13 to L20 S29 A20. 5 ma. 2.9 K.

He stopped counting at “8” then resumed at “11” at the end of the stimulus. Later when asked if he had omitted numbers, he recalled omitting “9” and “10.” After he had resumed counting, the stimulus was applied at “5.” He continued until “15” when another observer entered the room. At this point he stopped and closed his eyes tightly. When asked why he had stopped counting, he replied, “Dr. D. came in the room.” A subsequent excuse for stopping was, “I thought I had gone far enough.” (It had been emphasized previously that he was to continue to count until told to stop.)

Same electrodes as above. 10 ma. 2 K.

There were abrupt arrests of speech with each stimulus. Although strictly reminded to continue counting, he continued to excuse his arrests variously, e.g. “I thought I was going too fast.” Later he explained a specific arrest as, “It seemed like a barrier, a natural end.” When given two words, “goldfinch” and “daisy” to recall during an arrest he could recall only the first.

L20 S19 A16 to L21 S26 A24. 15 ma. 2.5 K.

The current was increased from 10 to 14 ma. when arrest of speech was noted. When the arrest occurred he would hurry the next few numbers at the cessation of the stimulus as he resumed counting.

2/21/62 L18 S23 A13 to L20 S29 A20. 5 to 7 ma. 2.9 K.

At 5 ma. arrest of speech appeared only on the third of three trials. At 7 ma. he stopped counting 2–3 sec. after the start of the stimulus. When asked to protrude his tongue and hold it so, he slowly withdrew his tongue during the stimulus and likewise would not continue slowly opening and closing his right hand during the stimulus. He later recalled stopping the latter motion but could not recall why he had done so. Cessation of speech, repetitive movements of the hand and the maintenance of protrusion of tongue were confirmed repeatedly at this session and on 2/23/62.

L20 S19 A16 to L21 S26 A24. 12 ma. 2.2 K.

During stimulation he stopped counting and opening and closing his hand. During the arrest he recalled that the examiner had given him the word “seabells.” The actual word had been “seagulls.”

Case A.C.* 5/2/62 L18 S9 P1 to L21 S12 A6. 5.0 ma. 5.3 K.

* To avoid excessive overlapping, the loci stimulated in A.C. and T.B. have been omitted from Figs. 2, 3 and 4. However, if the reader wishes he may enter them by reference to the conventions mentioned in Method.
On 4 occasions he reversed or confused numbers when counting during the stimulus or reverted to Spanish (the patient was a Puerto Rican). After one mistake he laughed and later explained that his difficulty amused him. There was an associated sensation of being "a little bit dizzy." At 6.0 ma. there was abrupt arrest of speech. He usually was able to recall the arrest of speech but could not explain his difficulty. He repeated correctly the words "goldfish" and "horse" when they were given him during an arrest.

L21 S12 A6 to L25 S17 A13. 7.0 ma. 5.0 K.

With stimulation there was abrupt arrest of speech. He would stop then start counting at the end of the stimulus at the point at which he had been arrested without prompting. He reported that he felt "a little dizzy."

L23 S17 A13 to L29 S32 A31. 10.0 ma. 5.0 K.

When counting during stimulation he might hesitate and on several occasions inverted numbers. "Felt a little dizzy" during the stimuli. (Note that these points lay in the frontal white substance lateral to the region represented in Fig. 4.)

L16 S6 A3 to L20 S10 A11. 7.0 ma. 7.2 K.

On several stimuli he "felt a little dizzy." With each he stopped counting abruptly explaining, "I forgot the numbers." Threshold seemed quite sharp between 6 and 7 ma.

L20 S10 A11 to L23 S16 A20. 10.0 ma. 4.0 K.

Arrest of speech occurred with 4 stimuli. The pupils were observed closely and no change was noted. Several times he screwed up his face in the effort to continue. He denied any sensation during arrest of speech. At the end when asked how many times his speech had been arrested he replied, "Once or twice" whereas it had actually been 4 times. None of the above stimuli was accompanied by any turning of head or eyes.

5/3/62 L18 S9 P1 to L21 S12 A6. 5.0 ma. 4.2 K.

When counting during stimulation near threshold he would repeat the numbers or give them in Spanish prior to complete arrest of speech. He would continue counting at the end of the stimulus. When asked he later explained, "I forgot the numbers." There was apparent rise in threshold with repeated stimuli and later when testing the effect upon both counting and opening and closing the right hand he had arrest of both only at 7.5 ma. with a considerable latency. He could hold out the tongue steadily during stimulation at a level that otherwise would produce arrest of speech. There was no facial movement with any stimulation and he denied any sensation.

L21 S12 A6 to L25 S17 A13. 7.0 ma. 4.8 K.

There was arrest of speech at the start of 3 stimuli but the abruptness of effect was reduced with repeated stimuli. When asked he reported a sensation through his "whole body." When being urged to continue counting during arrests of speech his right-sided tremor appeared during his attempts at speech.

L16 S6 A3 to L20 S10 A11. 8 ma. 4.5 K.

Stimuli in sequence produced: (1) No interference with the posture of the protruded tongue. (2) Arrest of speech after repeating several numbers. At the end he continued counting spontaneously but went back to "1" rather than continuing the series as he had been told to do. (3) No arrest of speech.

L20 S10 A11 to L23 S16 A20. 10 ma. 3.5 K.

On successive stimuli there was: (1) No movement of tongue. (2) Abrupt arrest of speech (repeated 8 times). (3) When tested for both arrest of speech and arrest of movement of hand no effect was seen at this level of stimulation. After resting for 2 min. arrest of speech could be obtained at the above level which did not arrest repetitive movements of opening and closing the right hand. (This observation suggested that the progressive rise in threshold with repeated stimuli was not simply ascribable to damage of tissue from passage of the current.)

No turning of head or eyes was seen with any stimuli.

Case T.B. 5/7/62 L20 S11 P3 to L23 S15 A4. 10 ma. 2.3 K.

When counting during several stimuli he would stutter or mispronounce the numbers. He could not explain this except, "I just flubbed." During the interference with speech he would swallow or move the head or hands about in an impatient fashion attempting to get the words out. He denied any sensation and there was no movement of head or eyes. After several stimuli the response no longer could be obtained. After stimulating at the other sites of electrodes this pair of electrodes was tested again a half hour later. Abrupt arrest of speech again was obtained at 10 ma. 3 times. He denied any subjective sensation and there was no turning of head or eyes.

2. Responses Outlasting Duration of Stimulation.

a. Confusion. Following a series of stimuli inducing arrest of speech a vagueness and mild inappropriateness of behavior was seen in 4 patients (G.G., H.T., V.S., and E.B.). This was not readily apparent to casual observation but could be elicited by close questioning or testing for the interpretation of proverbs or the likes or dissimilarities of abstract concepts (e.g. "law" as opposed to "justice"). No change appeared in simple calculation and no abnormal ideation was noted. The duration of this peculiar state was a matter of several minutes to a quarter of an hour. It should be pointed out that
both V.S. and E.B. had suffered from impairment of recent memory prior to entering the hospital for treatment of their tremor. The defect was apparent to the family but all but completely denied by the patients. Following a ventrolateral thalamotomy (which abolished the tremor completely in V.S. and partially in E.B.) the loss of memory was much intensified although the patient’s responsiveness and activity about the ward were not diminished noticeably. Two examples of confused speech immediately at the end of stimulation follow:

She stopped counting during stimulation. When asked why she did so she replied, “I thought you might whistle.” Later after each of 3 stimuli she said, “Louie took a bite.” (Louie was her husband.) She later explained, “I just thought of him.” She denied any subjective sensation.

16-channel electroencephalographic recording from the scalp and from adjacent depth electrodes failed to show any epileptiform activity after the stimulations.

Case H.T. 9/18/61 R15 S16 P1 to R18 S24 A6. Current 12 ma. 3.2 K.
She stopped counting during the stimuli without facial movement. This was repeated several times. When asked why she had stopped counting she replied variously during this period, e.g. “I thought you didn’t want me to go on.” “I thought I’d gone far enough.” “I thought you were leaving the room.”

Later during a stimulus she turned her head to the left and made a humming noise in her throat. There was rapid blinking of the eyes. Following the stimulus when asked how she felt, she replied, “One of the nurses was saying what they say in court, ‘Y, Z, oh yea, oh yea.’” (No nurse had been present nor could speech from adjoining areas be heard.)

b. Poststimulation confused behavior with amnesia. In all cases the response appeared during stimulation which was stopped as soon as the patient moved. The total duration of the response was less than a minute in all cases.

He continued to lie quietly at the onset of stimulation then suddenly turned his head and eyes to the right. Following this he sat up and tried to remove the plethysmographic capsule from the left hand despite being told not to do so. A few seconds later he became tractable to suggestion but would not respond verbally. Shortly thereafter he recovered completely but had no memory for any part of the episode.

Case E.S. 9/29/60 R10 S13 P2 to R10 S19 A6. 8 ma. 0.5 K.
With stimulation there was tensing of the dystonic left arm and face, then she sat up and looked about in a vague fashion despite being told to remain supine. Shortly afterwards she lay down again on command and smiled. She wrote that she had had a “feeling of relief” and had thought that the whole session was over. No note was made of amnesia. (She communicated regularly in writing because of her dystonic disturbance of speech.)

He lay quietly for the first 3 sec of stimulation, then abruptly sat up turning slightly toward the examiner at his right. At first he was unresponsive, fumbled with his shirt and did not look at the examiner when spoken to. Then, when asked why he had moved, replied, “I wanted to sit up and talk to you. I wanted to tell you something.” Following a vague smile, he added, “Not very much, something less than nothing,” and lay back upon the bed. Within a minute he apparently had recovered fully but had no recollection of any part of the episode.

B. Intensity of Current and Loci of Stimulation. In 2 patients (G.G. and V.S.) 16-channel electroencephalographic examination was carried out during the stimuli that resulted in arrest of speech and/or confusion. The current, since it was delivered through a Grass Isolation Unit, was not monitored but no overt disturbance of the electroencephalographic tracing occurred following the stimulus. Specifically, there was no evidence of epileptiform activation.

In 9 of the 10 patients precise observations were made of the current required to elicit the phenomena mentioned above. The effective current interval lay between 5 and 15 ma. (All reference to current implies peak current.) Using the same technique sensory responses commonly were obtained from the basal region of the nucleus ventralis lateralis (presumably from spread to the nearby sensory nuclei) with threshold currents of 1 to 5 ma. Unfortunately for the present purposes, we do not have any observations of direct stimulation of the sensory relay nuclei which might serve as a further comparison. It is evi-
dent, however, that the magnitude of the current is considerable and hence the local-
ing value of the positions of the electrodes may be reduced even beyond that entailed by the anatomical variation of this region (see Methods). It should be pointed out, however, that confusion was never observed from stimuli well within the thalamus, within the medial segment of the globus pallidus, or in a few instances when the electrode was intro-
duced into the tegmental field. Likewise negative results were obtained from stimuli from the superficial white substance of the frontal lobe. Thus the supposition must rest
that the responses we have observed did arise from stimulation of the head of the caudate nucleus or from the adjacent white matter of the frontal lobe and/or the frontal limb of the internal capsule.

Discussion

The present 10 patients were part of a group of 31 patients suffering from disorders of movement who were studied with depth stimulation prior to therapeutic lesions of the thalamus or pallidum between January 1960 and May 1962. The present results were not anticipated and regular testing of the pa-
tient’s ability to continue speech or motor activity during stimulation was not insti-
tuted until the fall of 1961, when it was realized that interference with these func-
tions might occur. Thus the earlier cases (L.S., E.S., and G.G.) were noted because of unusual positive actions. It is quite evident that simple observation and subsequent in-
quiry as to whether the patient had noted any unusual sensation during the stimulation was inadequate. With only 2 exceptions men-
tioned above, all the patients reported that they had experienced nothing unusual from stimulation of electrodes which would arrest speech or voluntary movement if either were in progress.

To recapitulate briefly, the response to stimulation within or in the vicinity of the head of the caudate nucleus consisted pre-
dominately of arrest of speech or voluntary movement and this usually appeared with the lowest current. In 3 instances (A.C., J.C., and V.S.) arrest of speech appeared at a lower threshold than arrest of movement. The arrest might be overcome to a degree by external circumstances (V.S.) or by strong urging (J.C.). Repeated or prolonged stimuli might lead to reduction in the patient’s ability to recall what had been said to him (recall of words was omitted or the words were returned in a garbled fashion) or speech might be well articulated but not pertinent to the situation. Turning of the head and eyes to the opposite side occurred usually at a higher current than was needed to induce arrest of speech and movement. There was no evidence that arrest of speech was caused by contraction of the oral or pharyngeal musculature. Finally, in 3 in-
stances, excessive stimulation induced an ictal-like episode in which the patient became unresponsive, turned the head and eyes to the side opposite to the stimulation, sat up and fumbled with his clothing or the appara-
tus. In 2 of these there was complete amnesia. In the third case the record was incom-
plete.

Our failure to obtain arrest of speech from the thalamus itself was not felt to be a con-
tradiction of the findings of Guiot et al. 7 As mentioned above our stimuli were delivered well within the thalamus in the vicinity of lateral 10, superior 0 and posterior 15 and from this region along a tract passing anteriorly, superiorly and slightly laterally. On the other hand their stimuli were delivered to the most lateral portions of the thalamus at the junction with or actually within the pos-
terior limb of the internal capsule. Their dia-
grams would indicate that the points of stimulation lay at lateral 17–18, and roughly posterior 10–20 and superior 0–5 by our coordinate system. The responses they re-
ported seem to differ qualitatively from our own. Guiot et al. found both arrest and ac-
celeration of speech and said of the latter, “The phenomenon appears to be conscious, and the patients are subsequently critical of it.” Although they found that some stimuli could produce the phenomena without motor or sensory components, major motor ac-
tivity of the contralateral side suggesting stimulation of capsular fibers was obtained in a number of instances. One may inquir

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whether the arrest of speech might not have resulted either from the patient being distracted by sensations in or motions of his body or by stimulation of fibers in the internal capsule from the facial and oral representations in the cortex. It will be recalled that Penfield and Rasmussen\(^7\) reported arrest of speech from stimulation of the lower portion of the sensory-motor region in either hemisphere.

Among the responses to human cortical stimulation, the response that the author has described resembles most closely that obtained from the supplementary motor area. According to Penfield and Welch\(^8\) the latter pattern includes: 1) Vocalization with movements of the face and jaws. 2) Arrest of voluntary activity (and speech). 3) Muscular synergies of three types: a) posturing, most often with deviation of the head and eyes to the contralateral side with abduction and external rotation of the shoulder and flexion of the elbow, b) repetitive movements of the hand or foot, c) rapid incoordinate movements of the extremities, 4) infrequent sensory responses usually reported as unlocalized body sensation, and 5) occasional autonomic effects including pupillary dilatation, cardioacceleration and respiratory inhibition. The present response appears as a truncated version of this more general response. We have not observed the "vowel cry" or the "interrupted sound"\(^9\) although Case H.T. made a humming noise. The muscular synergies have been limited to contraversive of the head and eyes. The subjective sensations of H.T. and J.C. may be noted in the original descriptions.

Although the marginal gyrus (supplementary motor region) and the caudate head are separated by a considerable distance, it is quite possible that in the internal capsule adjacent to the latter efferent fibers from this cortical area might have been stimulated and have released responses having their cortical representation on the mesial aspect of the hemisphere.\(^1,4,11,20,24\)

Using implanted electrodes in unanesthetized cats, Hess\(^2,16\) was able to trace a portion of the subcortical pathways which carry the impulses causing contraversive of the head and eyes. He obtained the response from the lateral margin of the caudate nucleus (including fibers of the internal capsule) and at lower levels from the lateral border of the anterior nuclei of the thalamus and the medial margin of the internal capsule near the genu. In a similar preparation Forman and Ward\(^5\) reported that stimulation of the caudate nucleus resulted in a turning of the head to the opposite side "in a manner resembling voluntary movement." Stevens et al.\(^22\) recently reported contraversive turning and circling movements after stimulation of the caudate nuclei with cholinergic drugs and other chemicals. Both of these latter groups found that following such stimuli the animals tended to become quiet and somnolent.

Laursen\(^12\) suggested that the fibers responsible for contraversive arise in the caudate nucleus since the response was not lost after degeneration of the internal capsule. It may be noted, however, that his cortical resections apparently were limited to the lateral surface of the hemisphere preserving the mesial aspect and, presumably, its subcortical connections.

Considerable evidence has appeared to suggest that stimulation of the caudate nucleus induces inhibitory effects upon movements already in progress in lightly anesthetized animals.\(^6,14\) In unanesthetized cats, Akert and Andersson\(^1\) found that stimulation of the head of the caudate nucleus induced three major responses: 1) reduction of spontaneous activity so that the animal might sit or lie down but without loss of response to external stimuli, 2) apparent difficulty in perceiving proprioceptive stimuli so that the limbs might be retained in unnatural postures, and 3) autonomic effects including slowing of respiration and miosis. Under narcosis and with stronger stimuli, the respiratory rate and the blood pressure might rise.

Inhibitory effects have been noted by subsequent investigators. Delgado\(^3\) found that stimulation of the caudate nucleus in monkeys caused them to stop ingestion of food and to reject food. In cats, Stevens et al.\(^22\) noted the loss of a conditioned avoidance response in cats during low-frequency stimula-
tion of the caudate. Finally, Spiegel and Szekely described a catatonic-like state in cats following alumina-cream lesions in the head of the caudate nucleus.

In the monkey Rosvold and Delgado found that stimulation of the caudate head did not alter the animal's ability to perform tasks requiring visual discrimination but produced considerable impairment in a delayed alternation test requiring the ability to recall a sequence over an interval of time. An inhibitory effect also was reported in man by Heath and Hodes who noted that prolonged stimulation in the vicinity of the head of the caudate nucleus resulted in the patient going into a "deep sleep" from which it was impossible to arouse him. The effect might outlast the stimulus by several minutes. The use of a schizophrenic subject may have prevented the ready observation of the effect of lesser stimuli.

It has been questioned whether the caudate nucleus is actually the locus from which the inhibitory responses have been elicited. Peacock found in anesthetized animals that inhibition of cortically induced movements could be produced from the internal capsule at thresholds below that producing tonic movement of the opposite limbs but that the two responses tended to blend as the intensity of the stimulus was increased gradually from liminal values. Neither response could be obtained from the striatum or internal capsule in chronic decerebrate preparations. More recently Laursen found that inhibition of a conditioned avoidance reaction was attained at a lower threshold from the rostral limb of the capsule interna than from the caudate nucleus itself.

It will be noted that the responses given by the patients in the present study did not resemble those usually given by patients subject to aphasia caused by epileptic or electrical inactivation of the cortical speech areas. After recovering from such an episode, the patient is well aware that something out of the ordinary had occurred and the response frequently is heard that he could not "think" of the right word and that this concerned him. In the present arrests of speech the patient never mentioned them spontaneously and indeed often seemed to be totally unaware that anything out of the ordinary had occurred. With the exception of one instance with E.B. (who after many arrests of speech and much questioning as to his subject state at these times, remarked about a "barrier") an inconsequential excuse related to external conditions usually was given for their failure to continue to count as had been requested. A similar excuse was given in the case of failure to perform other tasks during stimulation. It will be noted, in addition, that arrest of speech occurred in the right hemisphere of 2 of the patients who by clinical test and history appeared to be definitely right-handed.

Question may be raised as to whether the arrest of speech might have been caused by distracting sensations or motor phenomena. This seems unlikely for although A.C., J.C. and H.T. did report concomitant sensations these were of minor degree and not in themselves particularly distracting. By contrast, when somatic sensation was elicited by stimulation in the thalamus, these patients (and the others in the series) had no difficulty expressing themselves and when the current was maintained near threshold and the patient instructed to continue counting despite the annoyance he always was able to do so. Tonic or clonic motor phenomena were not observed to accompany arrest of speech and in the last patients (T.B., A.C., J.C., V.S., G.G.) particular care was taken to inspect the open mouth and tongue to be certain of this point.

Thus we are left with the impression that what has been produced by stimulation in the vicinity of the head of the caudate nucleus is not indeed aphasia, but a more basic disturbance in which the impulse to speak or to continue another task has been dulled or forgotten.

Summary

The responses described were elicited from the head of the caudate nucleus, the adjacent deep frontal white substance and the frontal limb of the internal capsule. All
10 patients were right-handed and the responses were obtained from the left hemisphere in 7 and from the right hemisphere in 3. With 60/sec. 2.5 ms. unidirectional square pulses, the effective peak currents ranged between 5–15 ma.

Stimulation in the lower range of current would cause the patient to cease counting. In some instances they would stop performing a repetitive act such as opening and closing the hand. When asked why they did so usually some trivial excuse unrelated to the stimulus was given. With greater currents the same response was seen with shorter latency and often the head and eyes were turned slowly toward the contralateral side. If the patient were given a series of words during such a stimulation he might not recall them or would reproduce them in a garbled fashion. In 3 patients an ictal-like state was produced in which the patients became unresponsive and following turning to the opposite side, sat up, stared and fingered clothing or equipment or tried to remove it. There was no later recall of the episode. No stimuli were followed by a generalized seizure. Repeated stimuli at a level only sufficient to induce arrest of speech often were followed by confusion of mild degree.

References