Sensory Jacksonian seizures

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Sensory Jacksonian seizures were analyzed in 42 patients with regard to anatomical and temporal sequences. The origin of sensory Jacksonian seizures, in contrast to motor Jacksonian seizures, often began at peripheral sites with little cortical representation. The progression of seizure activity across the cerebral cortex followed a course that was neither rectilinear, radiate, nor random; it appeared to proceed in an organized manner to involve functionally coherent units. The patterns analyzed conformed more closely to cortical somatosensory maps reported for the chimpanzee than the sensory sequences presently available for the cortex of man. Complete diagnostic studies are indicated in patients presenting with sensory Jacksonian seizures because of the frequency of related focal pathology.

Key Words • epilepsy • somatosensory cortex • sensory Jacksonian seizure

The progression of a somatic sensory seizure as experienced by the aware patient often can be reported in fine detail. It was felt that collation of the features of these seizure patterns would provide information on cortical localization in man as well as the general concept of propagation of epileptic discharge.

Clinical Material and Methods

Patients with sensory marches are uncommonly encountered. The 42 cases in this series were collected over a period of 20 years by one of us (RAL), who was interested in the problem of cortical localization. Each patient interview was carried out using nondirected questioning during which the sequences of symptom progression were never suggested. This method of interviewing necessarily raises the question of the chronological accuracy of the historical record. However, since the patient’s seizures are strictly a subjective experience lacking objective signs, the nondirected interview diminishes the possible introduction of the interviewer’s preconceptions. Histories from patients considered unreliable were discarded, and several patients who experienced sensory phenomena were ultimately excluded from the series because of a later diagnosis of migraine. In two patients, attacks occurred during interviews, and it was possible to obtain with accuracy the chronology and nuance of symptoms during the seizures.

In 42 patients, 27 males and 15 females, there were 54 seizure patterns; several patients had more than one seizure pattern and one patient had four different sensory marches. The clinical features of the attacks were then mapped and analyzed according to anatomical and temporal sequences.

Illustrative Cases

Case 1

The onset of this patient’s seizure was in the left corner of the mouth, “like when you
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have Novocain." Next, this sensation radiated up the left upper lip to the left ala of the nose and proceeded to involve the left side of the tongue. The sensation then appeared in the tip of the index finger and proceeded down the volar aspect to the central palm (Fig. 1). The attack then subsided, but she could not recall a temporal order of cessation. There was no motor component to the attack nor loss of consciousness.

Case 2

This patient first noted numbness simultaneously in the thumb, index, and middle fingers, which then rapidly spread to involve the entire hand and forearm. In 20 to 30 seconds twitching developed in the first three fingers followed by clonic movements of the arm with abduction of the shoulder (Fig. 2). There was no involvement of the face.

Case 3

This patient reported numbness in the entire left leg from the mid-calf down. He then noted a "pulsing, like when your heart's beating." The sensation ascended the left leg and thigh, and stopped in the lower anterior quadrant of the abdomen. No sensation was noted in the buttock, but the left portion of the scrotum was definitely involved. After the abnormal sensation ascended to the lower abdomen, it "skipped" the rest of the trunk. The patient then "lost control" of the left arm and developed an abnormal feeling in the arm "as though it were wood" (Fig. 3). This feeling was definitely different from the sensation perceived in the leg. There was no orderly progression in the arm such as that noted in the leg; the entire arm was involved simultaneously.

Case 4

This patient noted "numbness in a 6 × 4-inch area just above the right knee," at onset. The sensation then ascended the anterior surface of the thigh and curved posteriorly over the head of the femur onto most of the right buttock. The genitalia and perianal regions were not involved. On one occasion, the march progressed farther, involving the right lateral flank, chest, axilla, anterior and posterior shoulder, and thence to the side of the neck, stopping at the level of the right ear. The right arm was not involved. Associated with the ascent of the abnormal sensation was motor activity involving the abdominal musculature, shoulder, and neck. There was a very close somatic anatomical correlation between the area involved by the sensory seizure and the motor activity. Simultaneous with the ascending activity, numbness would descend the lateral aspect of the leg in a 10-cm wide strip to the lateral malleolus, the dorsum of the foot, and into the great toe. Occasionally, when the foot became involved,
Discussion

In 1827, Bravais reported what is believed to be the first case study of a focal seizure, but it was a motor seizure; the bearing of this phenomenon on the broader question of cortical localization was clarified by Hughlings Jackson in 1870. Since then, there have been numerous studies analyzing focal motor seizures. In contrast, sensory Jacksonian seizures, which consist solely of the patient's subjective observations, have been reported infrequently.

In a typical sensory Jacksonian seizure the subject perceives an abnormal sensation which appears without apparent stimulus at a localizable cutaneous site. The sensation then spreads or "marches" to involve a variable extent of the contiguous sensory surface of the affected body member, then commonly progresses to other members. Some motor activity of an involved part may occur. There is usually no impairment of awareness and the march ordinarily halts and disappears without apparent reason, although it may progress to become a generalized seizure. It is presumed that the march reflects propagation of an epileptic discharge within a cortical area of somatic sensory representation.

The perceived origins of the sensory marches in this series are represented in Fig. 5. If this is transcribed onto a schema for the somatic sensory cortex, the distribution of the loci of origin can be better appreciated (Fig. 6). In this instance a simunculus of the chimpanzee adapted from Woolsey has been used since it probably reflects spatial distribution of cortical representation better than homunculi already described for man.
Ten seizures originated in the head area, 24 in the upper extremity, and 20 in the lower extremity. The origin of the seizures did not appear to favor those peripheral sites which have a relatively greater cortical representation. Consequently, the distribution of apparent sites of onset appears random, with all parts of the body except the tongue and trunk represented. There does, however, appear to be an increased frequency of origin occurring in the distal portions of the extremities.

In marked contrast to motor Jacksonian marches, only two of our patients had onset in the thumb alone. Similarly, only one case exhibited onset in the great toe, which is the favored point of origin for those motor attacks initially involving the lower extremity. Four attacks originated in the hip and/or thigh, sites which are seldom if ever involved initially in motor Jacksonian seizures. The onsets of some seizures were ascribed to body parts with meager cerebrocortical fields; for example the third toe, wrist, and occiput.

Once a somatic site of onset was established, it tended to persist even though the pathological lesion might change, as with an infiltrating neoplasm. With enlargement and more diffuse invasion by the tumor, a change in the character of the seizures might be anticipated. This rarely occurred, however, and the consistency of the loci of seizure origin observed in this series suggested to us long-term facilitation of a cerebrocortical focus.

Analysis of the temporal and topographic features of each patient’s seizure showed that the progression of an attack was neither rectilinear, radiate, nor disorganized. The front of epileptic discharge appeared to follow a path of predilection toward the anatomical limit of a functionally coherent unit, such as a digit, a hand, or an arm. Despite the known proximity of hand representation to that of the face in the sensory cerebral cortex, seizures beginning in the hand generally involved other parts of the upper extremity before proceeding to the face. Furthermore, only 50% of the attacks originating in the hand eventually progressed to the face (Fig. 2). This disparity of spread, despite similar cortical contiguity, suggests enhancement of seizure progression by predilection, possibly with a functional basis rather than simple spread across adjacent cortex. The implication here is that corticocortical connections between anatomically related parts are better...
developed or more facilitated than between cortically adjacent areas which have little functional relationship.

Progression was occasionally bidirectional, but the general tendency was seizure spread toward the base of a member. For example, where the seizure began in the palm of the hand, the march would tend to involve the rest of the upper extremity, but would usually not spread to the fingers. This, however, was not universal, and true bidirectional advance within a member was encountered (Fig. 4).

In some cases a cutaneous area appeared to be “skipped” by the march. For example, in Case 3 sensory symptoms began in the lower extremity, ascended to the lower abdomen, and stopped at the level of the umbilicus. Symptoms were noted next in the entire arm, apparently without involving the intervening trunk. Most of the other incongruities of seizure progression were described when a portion of the trunk was involved. This may possibly be explained by variation in the intensity of involvement, the lack of critical focus of the subject’s attention, rapidity of symptom progression, or more probably, the limited cortical representation of the trunk.

Other phenomena were associated with the sensory march. A motor component was present in 20 patients; 16 of whom had close anatomical and temporal correlation between the motor and sensory components of the attack. In only four instances did motor activity precede the sensory perception. No clue was found as to whether the associated motor march was the result of progression to the motor cortex or activation of the motor components known to exist in the primary somatosensory area. 6

A cortical construct summarizing these sensory marches conformed more closely to Woolsey’s evoked potential map of the chimpanzee cortex 9 than to the cortical sensory sequences described for man. 4, 5 Several discrepancies were observed, illustrated most clearly in reference to the cortical relationship of the hand to the face. In the usual ladder sequence of representation for man, the thumb is related to the brow and the corner of the eye. 4, 5 In Woolsey’s figure, the thumb is juxtaposed to the lower lip and corner of the mouth. Analysis of our cases strongly supports this latter relationship (see Fig. 7).

Table 1 lists the pathology encountered in our patients. In 24 cases, histological diagnoses were made; most of these were either vascular abnormalities or tumors. Five other cases were diagnosed radiologically but were deemed inoperable; one of these patients had a large hemispheric arteriovenous anomaly. Of the 13 remaining patients in this series, five were lost to follow-up and eight were found free of gross pathology despite repeated examinations. This high frequency of diagnosable focal pathology makes mandatory a complete evaluation of patients presenting with sensory Jacksonian seizures.

Acknowledgments

The authors wish to thank Dr. Robert S. Bourke, Division of Neurosurgery, Albany Medical College, for reviewing the manuscript and Dr. Clinton Woolsey, Laboratory of Neurophysiology, University of Wisconsin, for his assistance in case analysis and manuscript review.

References

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This paper was presented in part at the annual meeting of the American Association of Neurological Surgeons, Los Angeles, California, April 8–12, 1973, and at the Richard A. Lende Memorial Symposium, Albany, New York, August 15–16, 1974.

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