International Journal of Neurology

ISSUE DEDICATED TO:

MOTOR AND PREMOTOR AREAS

FIRST SYMPOSIUM OF THE FULTON SOCIETY

VOLUME 4 NUMBER 1

INTERNATIONAL JOURNAL OF NEUROLOGY

CONTENTS OF VOL. 4 — NUMBER 1:

The Chairman's Address

Dear Fultonians, Friends and Colleagues:

It is with utmost satisfaction that we raise our voice to welcome all of you to this First Symposium of the Fulton Society.

I count it a high privilege to address to you on this occasion, and I want to express my thanks to the outstanding host of speakers and disscussers, who will give unusual brilliancy to this Symposium on Motor and Pre Motor Area, in memory of our dear friend Dr. Fulton.

A true team of scholars, men of science and letters, Professors holding chairs in important centers of clinical and experimental work, have unified their efforts to make this, our First Symposium a golden gate for the many to come.

We want this Symposium be an expression of sympathy to those who have assisted very closely in Dr. Fulton's Scientific endeavors, and first of all to Mrs. Fulton, who for all of us is a dear friend and a mirror of Dr. Fulton's warm friendship to everyone of us. We want to mention specially to, since Dr. Fulton always did, his able Secretaries, who helped make possible the titanic activity Dr. Fulton always carried on. Miss Madelaine Stanton, Miss Margaret Thompson and Miss Mary Wheeler, to all of them our warmest regards and thanks for their devoted work.

The proposal of the Fulton Society has found so many echoing hearts that we could affirm that it was born by itself, like a natural phenomena searching its way to life.

This reunion brings the plans laid in the organizing meeting in Rome in 1961, to fullfilment.

The possibility of unifying the intellectual aristocracy in neurophysiology, neurology, neuropathology, neurosurgery, psychiatry, psychology, history of Medicine and Science, will allow the members of this unique Society, to grasp for the first time by their joint efforts, the meaning and purpose of human life.

And the possibility of establishing novel and untried scientific cooperation gives a wider scope to the inquiring mind and endless horizons to its endeavors.

Through this scientific meeting we honour the memory of a man who has been a magnet to scholars, and a catalyst in the urge which exists in the human being to learn and to know about his nature.

In one of his last Christmas letters, with a dismalness unknown in him, Dr. Fulton addressed a few sentences to us, in which after complaining of ill health he said, "The man is gone, Alas, but the work remains".

And it is in the shrine of science that we will reverence the memory and the deeds of one of its most devote workshippers.

In solitude we create but it is in Societies like this that creation is cross-fertilized, by the interchange of erudition and imagination and it is from this, that the best sort of stimulation may arise.

The warm response to the creation of a Fulton Society was found everywhere, in national and international quarters. The brilliant group of scientists that today honours his name, by bringing here the fruits of their investigations in one of the fields most cherished by Dr. Fulton, the Motor and Premotor Area, are eloquent enough to show that they not only render homage to the outstanding scientist that John was, but to the man who has mastered the art of friendship in all the aspects of human relation. India, Turkey, Korea, Japan, Brazil, Canada, Russia, England, China, Uruguay, Switzerland, Holland, Germany, France, to cite just a few of the different countries in which the Fulton Society has members, shows that friendship like the sunshine spread their warming glow everywhere.

One of the most cherished aim of the Fulton Society, could be expressed in just three words: Fraternity through science. And he who has done so much for it, will always have an undimming light of remembrance which like a votive lamp will make his name be ever present in our midst.

The music of the spheres fills man's heart and mind when the departed ones are remembered.

This is a victory over death, because death fullfils its dissolving mission only when man is forgotten.

The link of the mortal with the everlasting —said Lucrecious— and to think that they can feel together and act one upon the other, is but foolishness". But for us, thinking within the limits of our earthly life, to link the mortal with the everlasting is to link man's name, and man's ideals to the everlasting stream of humanity. Intermingled in its currents in the mind of many generations, dwell the thoughts of the inmortals. Every life is but an unfinished task, the final truth the perfect beauty, is beyond the span of one lifetime.

Life preserves those who have served her. And Dr. Fulton was in love with life. His powerful personality was capable of nurturing the greatest variety of human interest and human purpose embracing History, science, and the humanities, ranging from the nerve cell to the human being as a social entity, nothing was alien to him. This is clearly shown by the great variety of specialists that belong to this scholarly Society.

Science is always in demand. I have not known any country which complains of a surplus of it, and when this is not enough, as in ancient times. they sail for it and import it at any price. Nations of the world price it among their highest possesions. Civilization has drawn its mental vigor and nourishment from this source. The ones who feel themselves as belonging to the kingdom of thought and who wirk for its expansion, to share its blessed ness with every human creature, are very well represented in this international Society in which a fraternal feeling kindles every beating heart.

And this love for knowledge is our force and our richness. As the wisest of the Bible Kings said "Happy is the man that findeth wisdom, and the man that getteth understanding. For the merchandise of it is better than the merchandise of silver and the gain thereof than fine gold. She is more precious than rubies, and all the things that you can desire are not to be compared unto her".

I see a spirit of indomitable force, which through knowledge has come down to us through the centuries and which will surely turn the history of the world for the better.

Wickedness is ignorance, war is ignorance, falseness is ignorance, because through it, we are betraying ourselves by misleading others.

Now more than ever, man holds his destiny in his own hands. In this impredictable field of science, a magnificent story is evolving before our eyes. Humanity is on the verge of breaking the limits that bound man to earth. How it will end, how far he will reach? No one can foresee.

But the present is ours and it confers responsibility on us. The craracter of human Society will be shaped by our resolutions, by our deeds, by our sense of cooperation in the pursuit of man's happiness.

Never before in man's history has science played a greater role than now- We must rejoice on being alive at this cardinal point where the turn of history is so important to our fate, because it is a great opportunity for the man of science to be of service here, on earth.

We hope that the Fulton Society will be a pilot plant for a better world of fraternity through science.

There are many kinds of friendship, but the nobler one is as William James said to Carl Stumpf "A friendship like ours, based on higher mental afinities and sympathy of character, it is the fruit of years and of work".

A friendship of this kind is the legacy of John Fulton to all of us, and can be counted among the priceless things of life. And let me tell you, that wherever you go, it will be difficult not to find a Fultonian with whom you will not be able to share happy moments in a foreign land.

The glacial grasp of loneliness will not take hold of you when you reach or sail to far off shores.

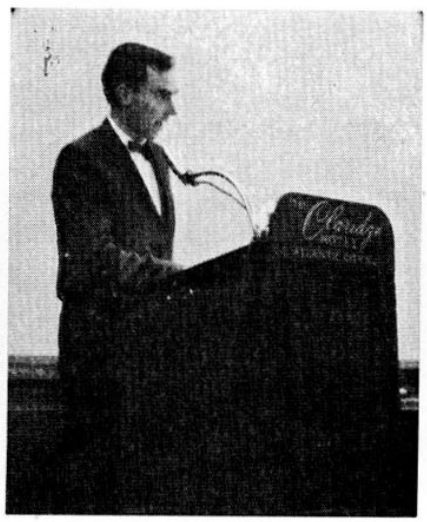
The importance of this is illustrated by the following anecdote: Rupert Brooke in his youthful days was about to sail from Liverpool to New York. When he got on the ship he saw that everyone except himself has somebody on the dock to wave them goodbye when the ship sailed. All of a sudden he felt lonely, he saw a ragged boy in the dock. He went ashore and said to the boy: "Do you want to earn a sixpence?" "Yes", the boy said. "Then wave to me when the ship sails". With a dirty handkerchief this boy waved enthusiastically to his unknown friend until the ship was out of sight".

There is no better example of the yearning need for fellowship and human sympathy that it is in the core of our nature.

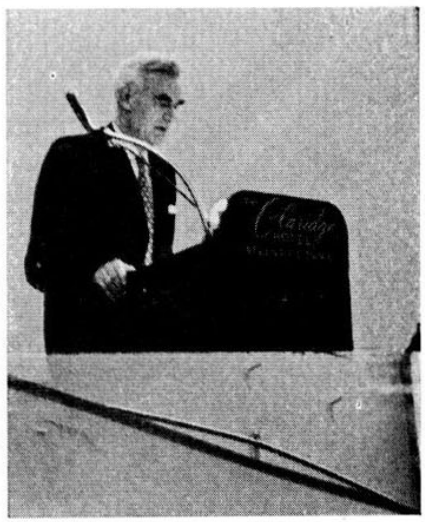
I don't want to delay the time to enjoy the enlightening words of our distinguished co-member and President of the American Neurological Association, Dr. Charles D. Aring, who embodies the nobler traits of the Fulton Society. Neurologist, man of letters and humanist, Dr. Aring will expound about the impact of Dr. Fulton's personality on contemporary Neurology.

VICTOR SORIANO.

Robert Aird



Robert Livingston

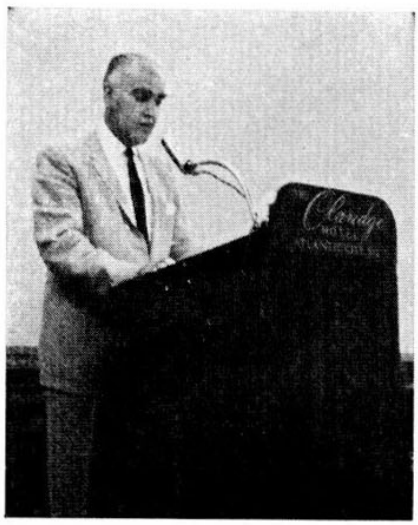


Charles Aring

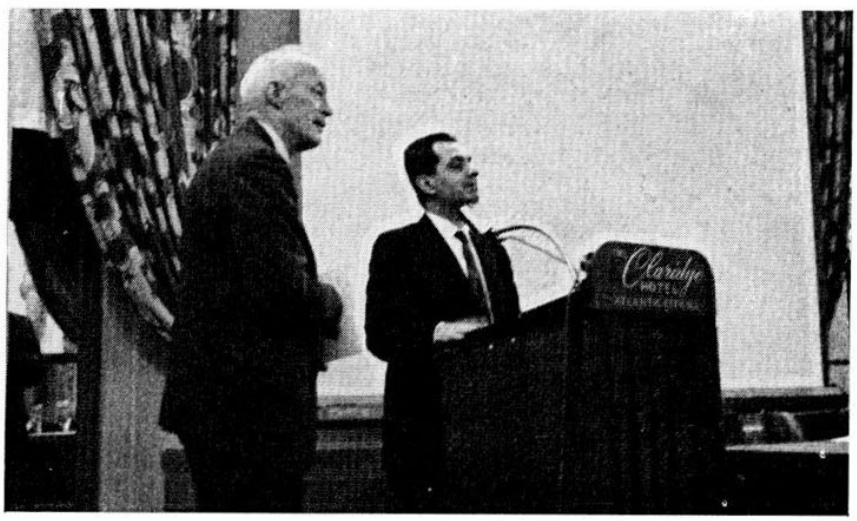
Margaret Kennard

Morris Bender

Maitland Baldwin



A. Earl Walker



D. Denny Brown and Victor Soriano

Acknowledgment: we thank to Dr. Franc D. Ingraham by whose courtesy, we have these pictures of the First Symposium of the Fulton Society.

Impact of John Fulton's personality on the Neurological disciplines

CHARLES ARING
Cincinnati.

When Victor Soriano asked that I open this first meeting of the society with a statement of the impact of John Fulton's personality on the neurological disciplines, it called to mind again John's talent for bringing together disparate and far-flung specialists and integrating them. Consider for a moment the specialties that bear the imprint of this dynamic personality, including neurology and neurosurgery and their ancillary disciplines; as well as psychology, psychiatry, physiology, primatology, endocrinology, and bibliography, including to be sure biography and the history of science and medicine.

Specialization did not imprison him. His versatility allowed him to exert leadership in many areas while simultaneously stimulating innovation. His was the ability to reorient himself in a rapidly changing world, and to function as a generalist as well as a specialist. He was prone to look upon men and women as better than they seemed, and sometimes they lived up to his expectations. In this and many other ways he fostered innovative, versatile and self-renewing scientists, giving them room to breathe.

John Fulton's influence can be illustrated by sketching my own experience at the Yale laboratory, where I arrived with as fine training in clinical neurology as it was then possible to have. I planned to shore up my neurological foundation with physiology and to integrate with it the neuroanatomy and neuropathology already acquired. I reasoned that this orientation would sharpen teaching and clinical skills, and perhaps encourage an investigative bent. The clinician securing his base at Yale was immersed promptly in a world of physiology —neurological as well as general physiology— such as required him to swim if he were not to sink. John Fulton's propensity for surrounding himself with persons of wit, fascination, and good will, allowed for collaborators if they were needed, to assist in a rapidly shifting learning situation.

Books saturated the atmosphere. The unassuming historical approach must have broadened many an uncommitted young person. This never-to-be-forgotten educational experience was leavened by visitors of great moment. They came to New Haven to commune with Fulton and to see his laboratory, coincidentally allowing themselves to be seen by neophytes. It would be unbecoming to omit the social aspects of the learning experience presided over with such unassuming charm by Lucia Fulton. Departure from the laboratory by no means severed connections, since the Fultons continued a lively interest in one's career.

Here was the ideal in educational opportunity, neurological or whatever; the discipline and the equipment, and the philosophy, the friends, and the humanism and warmth surrounding it. By no means the least was Fulton's skill in the management of nascent ideas. The great medical philosopher Wilfred

Trotter has commented on this feature of our culture in these words "...if mankind is to profit freely from the small and sporadic crop of heroically gifted it produces, it will have to cultivate the delicate art of handling ideas." John Fulton was a practitioner of Trotter's serene sanity that is the scientific mind; he gave to every fresh idea its intense moment of cool but imaginative attention before venturing to mark it for rejection or suspense. He knew that youth must be allowed to think for himself, that one of the functions of a fine teacher is to tread ever so gently, though not flabbily, in dealing with developing minds. Education should be gentle and stern, not cold and lax, said Joubert. Highly motivated John Fulton had a healthy respect for the sources of his own energy, enthusiasm and motivation.

I venture that as a humanistic teacher, scientific investigator, and handler of ideas, John Fulton has had as much to do with the neurological sciences as any physiologist of his day. He is in a direct line from his own mentor, Sherrington. It is safe to say that by way of his pupils, he will exert a continuing influence on future neurology.

His procedure of fostering collaboration and integration among highly specialized disciplines and peoples, is apt to be the method of revelation. Specialization is a universal feature of biological functioning, and in these times of intense specialization, the need to coordinate is ever pressing. The neurological tenet of Hughlings Jackson practiced by John Fulton, that increasing differentiation must always be followed by progressive integration, applies to the complex intellectual ventures of modern man.

Will those of us who worked intimately with John Fulton ever forget his magnificent leadership; the friendliness, the liveliness, and the intelligence of it? What a highly civilized department he conducted! And what an amazing person he was! We owe him much. If in our identifications with John, we cannot achieve his versatility, we can try for his flexibility and, in the Fultonian manner, spread the nets widely to bring to our discipline the varying talent. Perhaps a way to amortize our debt to him is to do for others as he did for us.

Comparative Studies on Localization in Precentral and Supplementary Motor Areas

CLINTON N. WOOLSEY
Laboratory of Neurophysiology University of Wisconsin
Madison, Wisconsin.

I accepted with some hesitation an invitation to participate in this first symposium of The Fulton Society, since I am not one of that large number of persons who received part of their scientific training in the Yale Laboratory. My association with Dr. Fulton came later, as a member of the Editorial Board of the Journal of Neurophysiology. Yet, since the early thirties, I have had an interest in the subject of this symposium. The work of recent years in our laboratory on the precentral and supplementary motor areas is directly relevant and, in an indirect way, it can be said to have had its origin, in part, in the work of the Fulton School.

Fulton's concepts of the motor and premotor areas[2], as illustrated in Figure 1, were derived primarily from the physiological and anatomical studies of the Vogts[9].

Reexamination of the details of organization of the frontal motor areas by the method of electrical stimulation brought forth evidence for two complete representations of the somatic musculature in frontal agranular cortex[10], which, however, differed in several significant respects from the results of the Vogts and, in consequence, they did not fit the areal parcellations of the motor and premotor areas, as these were used by Fulton. Figure 2 shows the extents of the precentral and supplementary motor areas in *Macaca mulatta,* with leg, arm, face and epaxial trunk representations indicated in different markings. The general plans of organization, in terms of body parts, are illustrated by the precentral (Ms I) and supplementary (Ms II) simiusculi of Figure 3. Comparison of Figures 1, 2 and 3 will convey the ways in which the motor and premotor areas differ from the precentral and supplementary motor areas. Essentially the precentral motor area, for completeness of representation of body musculature, requires inclusion of Vogt's area 6aα (the caudal half of the premotor area), while the supplementary motor area extents to only a slight degree onto the dorsal aspect of the hemisphere, and so does not include that portion of Fulton's premotor area which corresponds to Vogt's area 6aβ.

Reexamination of the effects of ablations based on the precentral and supplementary motor maps were reported by Travis[6, 7] and by Travis and Woolsey[8] and compared with the earlier findings of other workers, including those from Fulton's laboratory.

The precentral and supplementary motor areas have also been studied in two other animals which greatly interested John Fulton. These are the spider monkey and the chimpanzee. Figure 4 shows the precentral and supplementary motor areas of *Ateles ater,* based on data collected by Coxe *et al.*[1]. The postcentral tactile area (Sm I) is shown in part as it extends around the lower end of the central sulcus. A broken line indicates the bottom of this sulcus. In this animal the precentral motor area is very broad and extends well forward in the

frontal lobe. The breadth of this area, especially of the arm and leg subdivisions, may be related to the elaboration of control over proximal limb musculature for arboreal locomotion. The face area, in contrast,

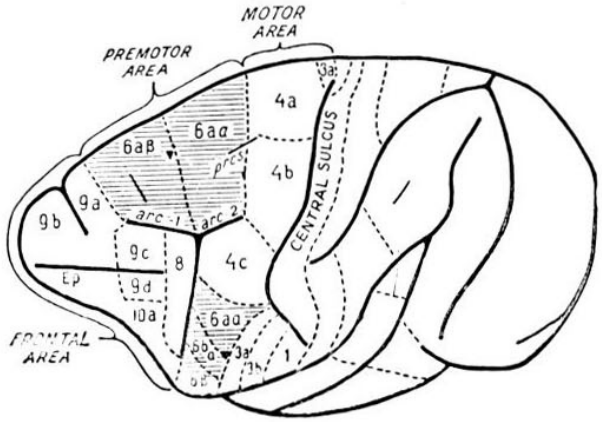


Fig. 1. — *The motor and premotor area of Fulton based on the studies of C. and O. Vogt. (From Fulton, J. F.:* Physiology of the nervous system).

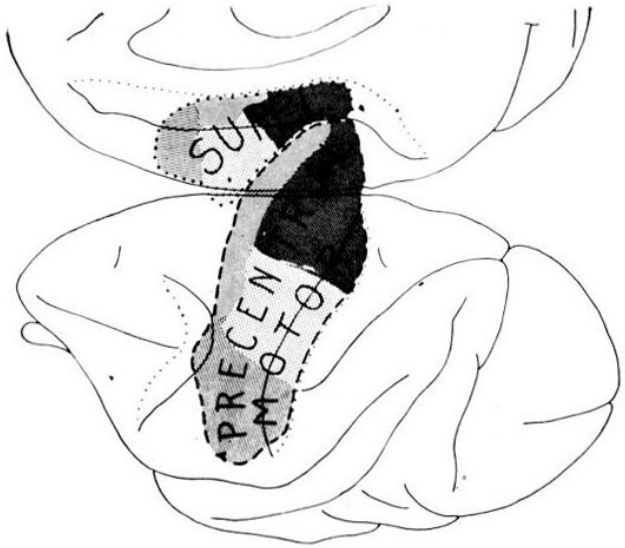
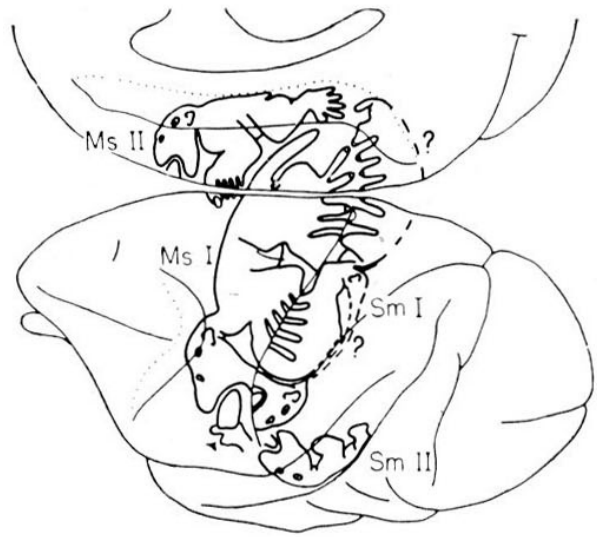


Fig. 2. — *The precentral and supplementary motor areas of* Macaca mulatta, *according to Woolsey* et al. *Fine dotted lines indicate bottoms of central, inferior precentral, and cingulate sulci. In the precentral motor area, face, arm and leg subdivisions from below up; epaxial representation along the rostral border. Subdivisions of supplementary motor area similarly marked with epaxial representation deep in sulcus cingulate.*



Fif. 3. — *Simiusculi showing general plans of organization of precentral (MS I) and supplementary (MS II) motor areas of* Macaca mulatta, *according to Woolsey* et al. *Also shown are the postcentral (Sm I) and the second sensory (Sm II) areas. (Modified from Woolsey, C. N. Organization of somatic sensory and motor areas of the cerebral cortex, in: Harlow, H. F., and Woolsey, C. N.* Biological and biological bases of behavior. *University of Wisconsin Press, Madison, 1958, xx, 476 pp.)*

is much narrower. The large tail area, originally defined by Fulton and Dusser de Barenne[3], and the area for the epaxial musculature of the trunk complete the precentral motor field. The supplementary motor area extends as far forward as the level of the rostrum of the corpus callosum and to the bottom of the sulcus cinguli. The precentral motor area does not reach this sulcus in *Ateles,* and so the hind limb portion of the supplementary motor area is not completely buried within the cingulate sulcus, as it is in *Macaca mulatta.*

Figure 5 represents our present information concerning the extents of the precentral (Ms I) and the supplementary motor areas (Ms II) in the chimpanzee, based on unpublished data from our laboratory. The precise boundary between the precentral and the supplementary motor areas is not clear. There is no tail area in the chimpanzee to assist in this differentiation as in the tailed monkeys. Nor do we have adequate

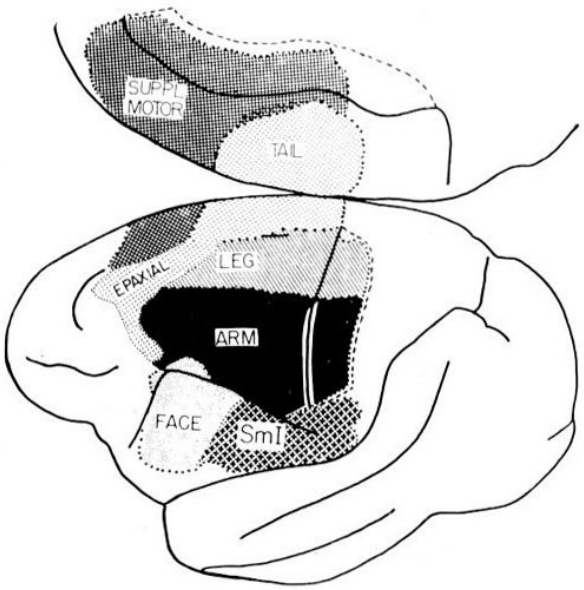


Fig. 4. — *Precentral and supplementary motor areas of* Ateles ater *according to Coxe* et al. *Face, arm, leg, tail and epaxial subdivisions of precentral motor area labeled. Supplementary motor area cross-hatched. Bottoms of central and cingulate sulci marked by dashed lines. A portion of the postcentral tactile area is also shown (Sm I).*

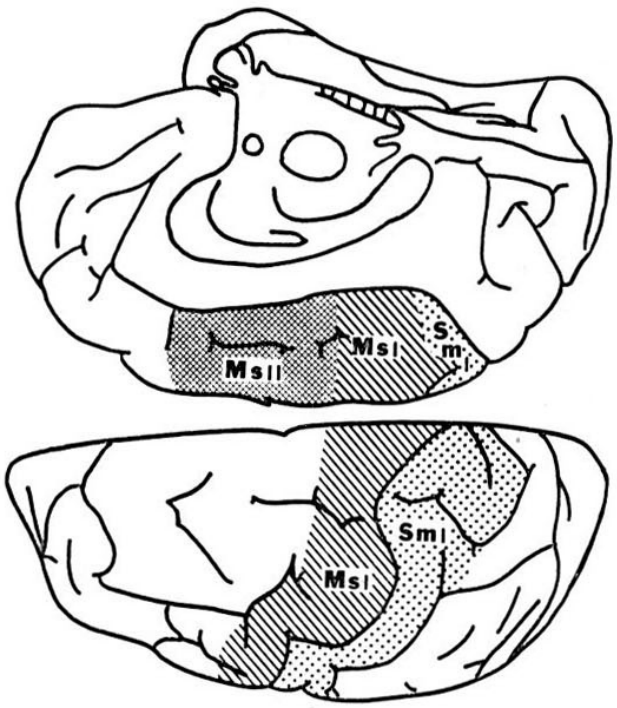
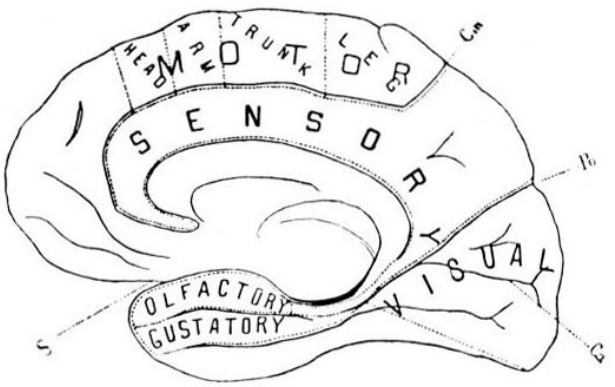
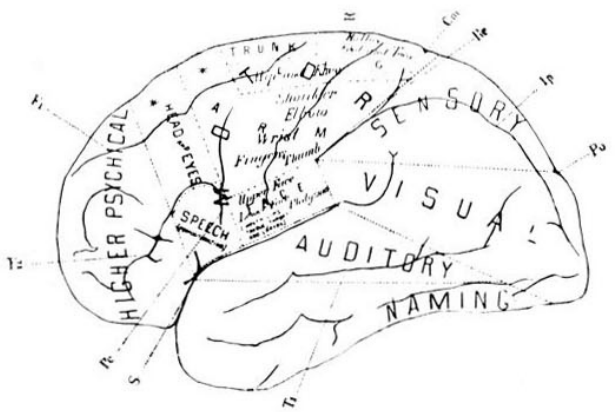


Fig. 5. — *Precentral motor (Ms I), supplementary motor (Ms II), and postcentral tactile (Sm I) areas of chimpanzee (Woolsey* et al, *unpublished data).*

data on the face subdivision of the supplementary motor area, nor any evidence of extention of the area onto the dorsal aspect of the hemisphere. These are defects primarily of insufficient material but may be due in part to high threshold of excitation in the face subdivision. Similarly, definition of the rostral border of the precentral motor area is not completely satisfactory, and it may extend somewhat farther forward. Again the epaxial musculature is represented rostrally but a complete map has not yet been made. Figure 5 also shows the extent of the postcentral tactile area as mapped on the mesial and dorsolateral aspects of the hemisphere.

In concluding this brief discussion of the precentral and supplementary motor areas, I should like to add a historical note and thus pay tribute to another of John Ful-





Figs. 6 and 7. — *Adaptation to human cortex of map of monkey brain, worked out by Schäfer and collaborators*[5]. *Taken from C. K. Mills*[4].

ton's interests. Among the first to draw attention to results obtainable on stimulation of the mesial aspect of the hemisphere, in the region now allocated to the supplementary motor area, was Schäfer and his collaborators. Their results were summarized in 1900 by Schäfer in the second volume of his *Textbook of Physiology*[5]. Not so well-known, perhaps, is an attempt to relate these early findings to man by direct transfer of the monkey diagram to the human brain. Figures 6 and 7 are taken from a review on cerebral localization published in *Brain* in 1889 by C. K. Mills[4]. It probably is the first figure illustrating representation of head and arm movements on the medial surface of the hemisphere of man.

SUMMARY

The work of recent years in the author's laboratory on the precentral and supplementary motor areas has in an indirect way had its origin, in part, in the work of the Fulton School.

Fulton's concepts of the motor and premotor areas, were derived primarily from the physiological and anatomical studies of the Vogts.

Reexamination of the details of organization of the frontal motor areas by the method of electrical stimulation brought forth evidence for two complete representations of the somatic musculature in frontal agranular cortex, which differed in several significant respects from the results of the Vogts and, in consequence they did not fit the areal parcellations of the motor and premotor areas used by Fulton. Comparison of Figures 1, 2 and 3 will convey the ways in which the motor and premotor areas differ from the precentral and supplementary motor areas.

The precentral and supplementary motor areas have also been studied in two other animals which greatly interested John Fulton: the spider monkey and the chimpanzee.

The author finishes his discussion by adding a historical note to pay tribute to one of John Fulton's interests; he points out the importance of Schäfer and coll.'s work for the later studies regarding motor and premotor areas, as in it the attention was drawn to results obtainable on stimulation of the mesial aspect of the hemisphere, in the region now allocated to the supplementary motor area.

RESUMEN

Este trabajo contiene los resultados de varios años de labor en relación a las áreas motoras precentral y suplementaria, labor comenzada a partir de los trabajos realizados por la escuela de Fulton. Los conceptos de Fulton estaban basados primariamente en los estudios de fisiología y anatomía de Vogt.

Reexaminados los detalles de organización del área motora frontal empleando el método de la estimulación eléctrica ha resultado evidente la existencia de dos representaciones de la musculatura somática de la corteza frontal agranular. Dichas representaciones difieren en algunos aspectos significativos de aquellas resultantes de los estudios de Vogt, y no se adaptan por lo tanto a la distribución de las áreas motora y premotora consideradas por Fulton. La comparación de las ilustraciones Nos. 1, 2 y 3 muestra las diferencias de las áreas motora y premotora con respecto a las áreas motoras precentral y suplementaria.

La representación completa del área motora precentral implica esencialmente la inclusión del área 6ª α de Vogt (la mitad caudal del área premotora); el área motora suplementaria, a su vez se extiende muy poco hacia el aspecto dorsal del hemisferio y no incluye por lo tanto, la porción del área

premotora de Fulton correspondiente al área 6ª β de Vogt. Se ha estudiado también las áreas motoras precentral y suplementaria en dos animales que interesaron particularmente a Fulton: el mono araña y el chimpancé.

El autor agrega al final una nota histórica para pagar tributo a uno de los intereses de Fulton. Señala los primeros descubrimientos de Schaefer y colaboradores. Subraya su importancia para el estudio actual, ya que ellos evidencian la posibilidad de obtener resultados en la región actualmente referida al área motora suplementaria.

RÉSUMÉ

Cet exposé contient les résultats de plusieurs années de travail se rapportant aux aires motrices précentrales et supplémentaires, commencé en partant des travaux faits par l'école Fulton. Les concepts de Fulton étaient en principe dérivés des études anatomiques et physiologiques des Vogt.

De nouvelles analyses des détails d'organisation de l'aire motrice frontale en employant la méthode de stimulation électrique ont manifesté l'évidence de deux représentations de la musculature somatique à l'écorce frontale agranulaire. Ces représentations étaient dans quelques aspects significatifs, différentes de celles appartenant aux résultats des Vogts, et, ne s'adaptèrent pas, par conséquent, à la distribution des aires motrices et prémotrices employé par Fulton.

La comparaison des illustrations 1, 2 et 3 fait voir les différences des aires motrices et prémotrices en rapport avec les aires motrices précentrales et supplémentaires.

Une représentation complète de l'aire motrice précentrale exige essentiellement l'inclusion de l'aire 6ª α de Vogt (la moitié caudale de l'aire prémotrice); l'aire motrice supplémentaire s'étend à peine vers l'aspect dorsal de l'hemisphère et n'inclut pas donc la portion de l'aire prémotrice de Fulton qui correspond à l'aire 6ª β de Vogt.

Les aires motrices précentrales et supplémentaires ont aussi été étudiées dans deux animaux auxquels John Fulton s'était beaucoup intéressé: le singe araign;e et le chimpanzé.

L'auteur finit en ajoutant une note historique pour payer tribut à un des intérêts de J. Fulton. Il le fait mention des premières découvertes de Schaefer et coll. et souligne leur importance pour cette étude actuelle, dans le sens que ces découvertes montrent la possibilité d'obtenir des résultats dans la région actuellement attribuée à l'aire motrice supplémentaire, par moyen de la stimulation de l'aspect mésial de l'hémisphère.

ZUSAMMENFASSUNG

Die Veroeffentlichung beschaeftingt sich mit den Ergebnissen einer jahrelangen Arbeit ueber die praezentrale und supplementaere motorische Zone, die ihren Ursprung in dem Werk der Fultonschen Schule hat. Fulton's Konzepte kommen in erster Linie von den physiologischen und anatomischen Studien der Vogts' her.

Die Wiederuntersuchung der Details der Organisation der frontalen motorischen Zonen durch die Methode der elektrischen Reizung brachte den Beweis fuer zwei komplete Repraesentationen der somatischen Muskulatur in der frontalen agranulaeren Rinde, die jedoch in verschiedenen bedeutenden Aspekten von den Ergebnissen der Vogts sich unterscheiden, und deshalb nicht in die Parzellierung der motorischen und praemotorischen Zonen passten, wie sie von Fulton angewandt wurden.

Der Vergleich der Figuren 1, 2 und 3 wird die Weise zeigen, in der die motori-

schen un praemotorischen Zonen sich von den prezentralen und supplementaeren motorischen Zonen unterscheiden.

Im Wesentlichen erfordert die prezentrale Zone, in der Vogt's Feld 6^a α (die kaudale Haelfte der praemotorischen Zone) einbezogen wird, damit die Repraesentation komplet ist, waehrend sich die supplementaere motorische Zone nur ein wenig bis in den dorsalen Teil der Hemisphere erstreckt und somit nicht jener Teil der Fultonschen praemotorischen Zone, der Vogt's Feld 6^a β entspricht, einbegriffen ist.

Die praezentralen und supplementaeren motorischen Felder wurden noch in zwei weiteren Tieren - dem Spinnenaffen und dem Schimpanzen studiert was John Fulton besonders interessierte.

Der Autor fuegt eine historische Note hinzu, um eines von John Fulton's Interessen zu ehren, indem er die ersten Entdeckungen von Schaefer und Mitarbeitern und ihre Wichtigkeit fuer die vorliegende Arbeit hervorhebt, wo auf ein Moeglichkeit hingewiesen wird, Ergebnisse zu erhalten in der Region die heute zu der supplementaeren motorischen Zone zugezaehlt wird, durch die Reizung der mesialen Seite der Hemisphaere.

Discussion: Dr. JAMES O'LEARY

Woolsey has presented admirably the changed concept of topical localization in precentral cortex between the time of Fulton and his own. In the quarter century gap which lies between these efforts to explore the precentral cortex there have been three innovations each adding materially to the much enhanced detail which developed out of Woolsey's later studies: the added flexibility of control permitted by electronic stimulators, replacement of ether by barbiturate anesthesia for experimental studies, and finally the refinement in mapping procedures made available through the discovery of evoked potentials. Since formerly electrical stimulation had been the only available method for delimiting cortical boundaries prior to ablation, the kind of neurological deficit said to follow differently placed precentral lesions had shown significant discrepancies between investigators. Such problems as arose are still in need of a final solution which cannot be expected for some time to come.

The studies of the Fulton group were much influenced by the architectonic maps of the cercopithecus brain as worked out by the Vogts. To the physiologist of Fulton's time the lines of anatomical demarcation sketched between areas, 4, 6, 8 and that other composite which was then called 'frontal association area', had the finality of the geodetic survey. Even sub-areas such as 6 *alpha* and *beta* were sometimes assigned different functions. Golgi architectonics had long been forgotten and using the 6 layer stratification the imaginative anatomist could parcel out as many fields as the rigidity of his criteria for dissimilarity invited. In many cases workers went overboard in designating minor fields, and each of these held the prospect of a proof of unique function. The cortical mosaic of that day was made up of tiles composed of masses of neurones which discharged in unison and by their mass effort produced each its unique effect. It remained for Lorente de No and later Lashley and Clark to deflate the significance of criteria used to distinguish the architectonic areas then in use; but faith in the Brodmann parcellation of the frontal lobe as a point of reference for stimulation and ablation studies had already become so entrenched that it spread through the text books and thus became ineradicable for years to come.

It was perhaps mere accident that in the well etherized monkey a distinct change in the pattern of motor response could be shown in an opposite extremity as one moved a stimulating electrode forwards from the central sulcus (area 4) into area 6. Disappearance of Betz cells is the distinguishing feature of area 4 as opposed to area 6, and for a long time the Betz cell region had been considered the chief locus

of origin of the pyramidal tract. At threshold the area 4 response so stimulation was typically a localized movement of a part of the face, hand or foot, whereas to get any area 6 response at all required a higher intensity and produced a torsion movement of the trunk with adversion of head (and eyes). Translate this older concept of motor versus premotor responses into the Woolsey map and one sees in the latter instead the homunculus of precise detail laid out upon the precentral gyrus with digits at the central sulcus and back protruding forwards past the anterior margin of area 4 well into area 6. Ablation studies based upon plotting the margins between the two fields at first indicated flaccidity and weakness from area 4 lesions which did not encroach upon 6 (Fulton), and spasticity from area 6 lesions. Later Hines and Richter were to show that spasticity could be produced by a simple transverse incision of cortex at the boundary between 4 and 6, while still later Woolsey and Travis proved the existence of the supplementary motor area on the medial wall of the hemisphere and that its ablation alone could produce contralateral mild, definite spasticity. This is about the way factual knowledge of the results of ablative lesions shapes up today.

Evoked potential studies have produced one other change in viewpoint in recent years, namely that the sensory cortex in back of the central sulcus is in actuality a sensori-motor cortex (at least in non-primate forms) giving evidence of excitability from stimulation of motor points as well as evidencing evoked potentials such as arise from distal stimulation. The strip which lies in front of the central sulcus is by the same token motor-sensory, producing evoked potentials as well as showing par excellence discrete contractions of extremity parts (more often distal than not).

The ultimate solution of the plan of organization of the motor cortex may be predicted to lie not with more knowledge of architectonics or the use of more erudite kinds of stimulation, better anesthesia, or even by the introduction of computers to do the work, but with new insights into interrelationships between patterns of firing in the separate neurones of closely aggregated cell assemblies such as lie beneath a stimulus point. We need to know how the pattern of on-going activity of such cell assemblies is changed by a stimulus. From present evidence it would seem probable that the instability thus introduced might persist for seconds before return to the steady state which preceded current flow. Such knowledge could ultimately explain the lability of cortical response and its kaleidoscopic changes in pattern under different conditions of anesthesia and using diferent parameters of stimulation. Collectively we characterize such changes as evidence of lability of the cortical pattern as opposed to a sharp-boundaried and unalterable mosaic which in the past it has been the inspiration of many workers to demonstrate. It is this very lability which is at the core of differences of opinion concerning the interpretation of results obtained using different methodologies.

The opposite extremes of stability and lability in the nervous system are well-exemplified in the behavior of two of the outstanding constituents of motor cortex - axons and synapses. A famous physiologist of the past generation once remarked that "the nerve fiber is a gentleman", appropriately praising the invariability of its response. Somewhat later George Bishop embroidered that idea by suggesting that "the synapse is a lady, fickle, temperamental, labile, promiscuous almost, and sometimes acting as if it had no inhibitions at all'. Those who have worked upon the motor cortex would agree, I believe, that she has all of the characteristics of a lady, causing, no doubt, her fascination for those who, except for attention to her whims, might find many other engaging problems much nearer ready for solution.

REFERENCES

(1) *Coxe, W. S.; Hirsch, J. F.; Benjamin, R. M.; Welker, W. I.; Thompson, R. F.*, and *Woolsey, C. N.*: Precentral and supplementary motor areas of Ateles. Physiologist, 1957, *1*:19.

(2) *Fulton, J. F.*: Physiology of the nervous system. Oxford University Press, New York, 1949, XII, 667 pp.

(3) *Fulton, J. F.*, and *Dusser de Barenne, J. G.*: The representation of the tail in the motor cortex of primates, with special reference to spider monkeys. J. cell. comp. Physiol., 1933, *2*:399-426.

(4) *Mills, C. K.*: Cerebral localisation in its practical relations. Brain, 1889, *12*:233-288; 358-406.

(5) *Schäfer, F. A.*: Textbook of physiology. Macmillan Co., New York, 1889-1900, 2 vols.

(6) *Travis, A. M.*: Neurological deficiencies after ablations of the precentral motor area in Macaca mulatta. Brain, 1955, *78*: 155-173.

(7) *Travis, A. M.*: Neurological deficiencies following supplementary motor area lesions in Macaca mulatta. Brain, 1955, *78*: 174-178.

(8) *Travis, A. M.*, and *Woolsey, C. N.*: Motor performance of monkeys after bilateral partial and total cerebral decortication. Amer. J. Phys. Med., 1956, *35*:273-310.

(9) *Vogt, C.*, und *Vogt, O.*: Allgemeinere Ergebnisse unserer Hirnforschung. J. Psychol. Neurol., Lpz., 1919, *25*: 277-462.

(10) *Woolsey, C. N.; Settlage, P. H.; Meyer, D. R.; Sencer, W.; Pinto-Hamuy, T.*, and *Travis, A. M.*: Patterns of localization in precentral and "supplementary" motor areas and their relation to the concept of a premotor area. Res. Publ. Ass. nerv. ment. Dis., 1952, *30*:238-264.

The Premotor Syndrome in Relation to Extrapyramidal Symptoms*

D. DENNY-BROWN

From the Neurological Unit, Boston City Hospital, and the Department of Neurology, Harvard Medical School.

The most unique attribute of John Fulton's many contributions to neurophysiology is the enormous enthusiasm he generated in a host of students and colleagues regarding every subject to which he turned his attention. The first outcome of the revolution he introduced into the study of cortical ablation in primates was an enormous surge of interest in the functions of the frontal lobe. The widened horizons that resulted in the following twenty years, ranging through many aspects of general behavior, mind and emotion are summarized in his Withering Lectures[1], his Salmon Lectures[2] and his lectures in Louvain[3].

In the short time at our disposal I would like to discuss an aspect of frontal lobe functions that was one of the earliest to secure John Fulton's interest, and of which the full significance is still not evident today.

In the course of their study of the plantar response resulting from lesions of the motor cortex in primates Fulton and Keller[4] found to their surprise that excision of the foot area in the motor cortex of monkey and chimpanzee failed to produce the expected evidence of spastic paralysis. Fulton and Kennard[5, 6] investigated the matter further and maintained that removal of the remainder of the agranular cortex (area 6 of Brodmann) was necessary to induce the postural and reflex changes of spasticity. Kennard, Viets and Fulton[7] put forward the view, revolutionary in 1934, that impairment of skilled movements, spasticity, forced grasping and vasomotor disturbance were together the result of damage to the "premotor cortex", corresponding to area 6 of the Vogt's, lying rostral to the area of origin of the pyramidal tract. Walshe[8, 9] stoutly defended the classical clinical tenet that spastic paralysis resulted from lesion of the precentral gyrus. The description by Sarah Tower[10] of flaccid paralysis following section of the pyramid lent strong support to the hypothesis of John Fulton, who insisted that spasticity is an extrapyramidal disturbance. The further experiments of Tower and Hines[11] whereby the "flaccid" limb following pyramid section in the monkey became spastic following ablation of the precentral gyrus on the same side, appeared to provide a final proof. However, spastic phenomena resulting from lesions of the pyramid in man have been reported in verified cases by Brown and Fang[12], and by Meyer and Herndon[13], reopening this whole question.

The situation is not quite so straightforward as John Fulton and his collaborators had supposed. Hines[14] showed that the area of monkey cortex, removal of which was necessary for the appearance of clasp-knife rigidity and clonus, was in fact a strip in the most rostral border of area 4, which she called area 4S. This area was in fact similar to that which Fulton and his associates had called area 6, for their ablations had been based on the mapping of the cortex of an African monkey by

* Assisted by grants-in-aid from the George Harrington Fund of Boston.

the Vogt's, where area 6 extends more caudally than in the macaque monkey. The area 4S was thought by Hines to be that from which widespread inhibitory effects could be produced, the suppressor band 4S of McCulloch and his associates[15]. In her most recent review of this subject Hines[16] still adheres to this view.

Our own studies of the relationships between areas 6 and 4 of Brodmann in monkeys were proceeding in Dr. Fulton's laboratory in 1936 when Dr. Marion Hines made her conclusions known to us. Publication of our own work[17] was therefore delayed until after that of Hines[14] had appeared. We differed from the conclusions of Hines in several respects. We agreed that spasticity of proximal joints (and therefore the hemiplegic posture) required ablation of the most anterior strip of area 4 (i. e. tissue containing large pyramids). Inhibitory effects could however be obtained with undiminished intensity from the remaining agranular cortex (area 6) when serial section showed that all Betz cell cortex had been removed. Conversely, if area 6 was alone removed the tendon reflexes were unchanged, though a soft plastic rigidity resulted in all muscle groups in the opposite limbs. Finally, in distinction from previous work we found that removal of a caudal strip of area 4, resulted in increased tendon reflexes in wrists and fingers, ankles and toes, with a mild type of resistance to increasing stretch of muscles. Dr. Fulton generously accepted these findings, which we jointly demonstrated in a chimpanzee, and to which he later referred as digital spasticity[18, 3].

The later work of Woolsey and his colleagues[19] showed a more orderly pattern of topographic representation of primary movements in area 4 with representation of proximal muscles in the most anterior strip of that area. It is possible therefore that what all these experiments were demonstrating was that removal of the areas for proximal joint movement resulted in proximal spasticity, and ablation of areas for acral parts resulted in distal spasticity. Though we have supported this view[17, 20], there are still some difficulties and inconsistencies. First, the fingers and toes, wrists and ankles, are much more spastic after removal of the anterior strip of area 4, where they are not directly represented. This was the basis of Fulton's first observations with Keller[4]. Second, Hines[14] maintained that the portion of the strip area 4S removal of which most consistently produced spasticity, is the most medial part. Denny-Brown and Botterell[17] found that removal of an excitable focus for a complex synergy in the region of the superior precentral sulcus was directly associated with the appearance of proximal spasticity.

Woolsey and his associates[19] later demonstrated the detailed pattern of foci for excitable movement of head, trunk and limbs in the "supplementary motor area", part of which lies in the medial 4S of Hines. (Fig. 1). Travis[21] found that removal of this supplementary area alone had no lasting

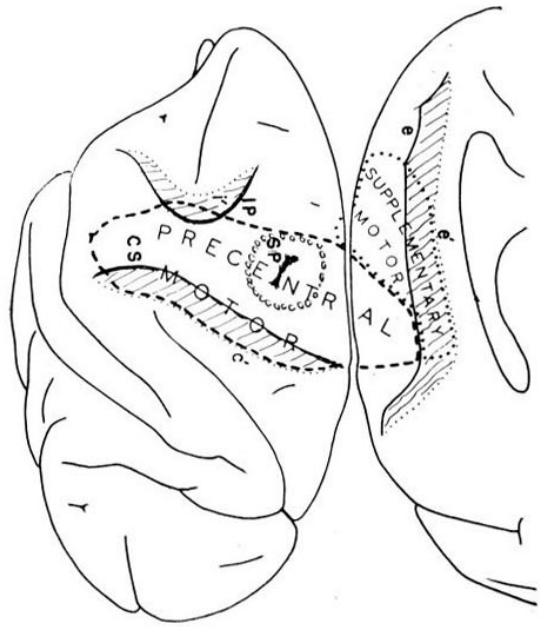


Fig. 1. — *Outline of hemisphere of macaque monkey to show the central fissure (CS) and precentral sulci (IP,SP) on the lateral surface and the cingulate sulcus (e) on the medial aspect. The anterior wall of the central sulcus, the posterior bank of the inferior precentral sulcus and upper bank of the cingulate sulcus are represented by shaded areas. To show the precentral area of Woolsey and the "supplementary area" of Woolsey. Attention is drawn to small superior precentral sulcus, or dimple, SP, and the area immediately surrounding it (small circles). Modified, from Woolsey[19] and Hines[16].*

effect, unless it was removed on both sides at once, when a "spastic" state of all limbs, without "significant" paralysis, was said to appear. Unilateral removal of the precentral motor cortex (area 4 of Brodmann) sparing the supplementary area, was stated by Travis [22] to produce no "significant" spasticity, but if the lesion was extended to include that part of the supplementary area lying medial to the precentral gyrus, spasticity of all limbs resulted. Hines[16] now appears to accept this effect as that which she described as the result of medial extension of her strip lesion. Reference to the map of Woolsey, however, shows that the foci of the supplementary area concerned here would be only those of the foot. Hines[14, 16] like Travis[21] insists that lesion of this area can cause severe and lasting spasticity without paralysis. Further, Hines relates spasticity to release from cortical inhibition of "tone", an undefined function that ignores the peculiar distribution of release in the flexor muscles of the arm and the extensor muscles of the leg. What is known of the supplementary area gives no clue to this prominent feature.

Degrees of Spasticity

Before we continue further we must define clearly what is meant by "spasticity" and "paralysis". In the past the text-book descriptions have focussed too exclusively on the condition known as "capsular hemiplegia". This condition is itself greatly variable, though a persistently flexed attitude of the upper limbs, and extended posture of the lower limbs is a prominent feature. It is usually associated with a type of resistance in the flexor muscles of the arm and extensors of the leg that begins to mount soon after beginning to stretch the muscle, reaches a peak then abruptly melts (the "clasp-knife" phenomenon). The tendon reflexes are easily elicited in the corresponding muscles, more rapid and of greater amplitude than normal. The tendon reflex is commonly repetitive to a single tap (clonic), and involves other synergists besides the muscle tapped (irradiation). At an optimum tension a tap or sudden stretch may elicit the repeated series of jerks called spinal clonus. In most accounts the variable degree of a similar resistance in the opposing muscle groups, triceps in the upper limb, hip flexors and hamstrings in the lower is ignored. In some of these e.g. triceps brachii the tendon reflexes may in some cases also be greatly exaggerated. In these muscles the resistance may be plastic in quality, i.e. present in similar amount at all lengths of the muscle. In the most spastic muscles a plastic resistance may be present upon which the spastic phenomena are superimposed. More importantly the clasp-knife quality may be absent, either because the resistance to passive stretch mounts progressively to a point where further passive movement is limited by increasing contraction (dystonia) or because of a fixed shortening of muscle or tendon (contracture). In short, the condition of capsular hemiplegia presents variable degrees of admixture of spasticity and the extrapyramidal sign we call dystonia[23]. The presence of "dystonia" can usually be recognized by the characteristic pronated or supinated posture of the hand and foot, and in thalamic lesions the spastic component of such dystonia can be seen in the typical spastic clawing of the fingers and toes.

The classical texts pay little attention to the beginnings of spasticity and dystonia, yet the recognition of these is part of the everyday experience of the neurologist in his search for signs of beginning disease. The clinician therefore has no difficulty in accepting as beginning spasticity the presence of a soft yielding resistance that appears only towards the end of passive stretch, and is associated with increased amplitude of tendon reflex. In this grade of change, clonus and its associated rapid tendon reflex, irradiation and clasp-knife phenomenon are absent. Yet such condition has commonly been referred to by physiologists as "flaccidity".

In her earlier work Hines[14, 24] sought to define paralysis in terms of the anatomists' classification of muscle function as prime mover, fixator, and antagonist. The function of area 4 was considered to be that of regulating "discrete" movements, "prime movers"[24]. Spasticity was defined

as increase in "tonus", or postural tone. The grasp reflex was thought by Hines to be the result of a loss of recriprocal innervation, so that the animal could not relax the flexors of the arms after removal of area 6. In the animal lacking area 4 of Brodmann bilaterally "proximal initiation of movement remains, distal initiation is gone. The prime movers do not begin a movement, the synergists (for example, the wrist extensors) are not as active, and the antagonists not as greatly contracted as in the intact monkey." (Hines[16], p. 507). Yet, Hines states that some such animals can pick up a small object between finger and thumb, and it is noted that these and other movements can still occur as part of generalized sequential movements such as scratching, where recriprocal innervation is intact.

The Supplementary Motor Area

The contention of Hines[14] that removal of the medial part of her strip 4S produced extreme spasticity without paralysis of movement presents a serious dilemma to the physiologist. If spasticity is a release phenomenon its appearance must mean that some corresponding function is lost. For Hines the lost function was inhibition, though Denny-Brown and Botterell[17] found the inhibitory response to stimulation to be unaffected by removal of the whole of area 4, and to be lost after careful removal of area 6, without damaging area 4. The same discrepancy appears in the findings of Travis[21] that ablation of the supplementary area is alone necessary to release spasticity, with no "noticeable paresis" when unilateral, and "practically no paresis" when bilateral. Both Hines and Travis found that bilateral lesions were necessary to produce clonus and spasticity. In the report of Travis[21] the supposed spasticity from supplementary area lesion presented curious features, for all limbs were held flexed in the first week and offered progressively greater resistance to passive extension. The tendon reflexes were difficult to elicit in this period, later becoming hyperactive in triceps, fingers and toes. Biceps, triceps and knee jerks were evidently difficult to obtain. Contractures of flexors developed, producing flexion deformities at elbows and knees. Such a condition is far removed from the phenomenon of cortical spasticity. We are unable to understand the contention of this author that "removal of the supplementary motor area seems to produce topographical localization of spasticity."

In four experiments personally carried out, including two bilateral ablations, I have not seen spasticity to result from ablation of the supplementary area. A soft plastic type of resistance to all passive movement in the contralateral limbs, but more evident in the flexors, is usually present when the animal is sitting. In one animal, with ablation of the total supplementary area on both sides at the same operation the result was remarkable. The animal walked with all four limbs flexed on the first day after operation, and frequently dragged the hind limbs or crossed them. On the second day and thereafter strong grasp reflexes were present in both hands, but the upper limbs were used naturally for walking, hopping, tilting reactions and climbing. The tendon reflexes in the upper limbs were slightly increased without spasticity. The lower limbs were used progressively better in the first week, but the ankles and toes and tail were paralysed, and the thighs were overadducted for two weeks. Visual and tactile placing were excellent in the upper limbs, but were absent in the lower limbs for two weeks, following which they gradually returned to normal. This transient weakness of the lower limbs was attributed to damage to the white matter under the leg area resulting from removal of the upper bank of the cingulate sulcus. In subsequent weeks the only residual defect was complete absence of all avoiding reflexes in the lower limbs, and of tactile avoiding response in the upper limbs, with slight spasticity of the tail. (Fig. 2). A soft plastic resistance was palpable in flexors (retractors) of shoulder, flexors of elbow and fingers, hip and knee, with abnormally rapid but small tendon reflexes in these muscle groups. There was no reflex irradiation and no clonus.

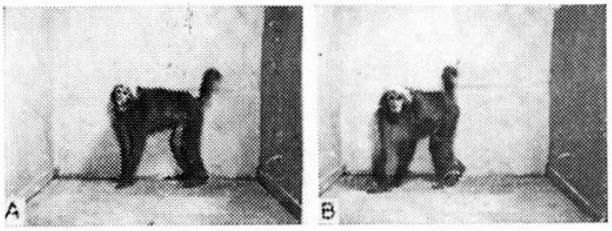


Fig. 2. — *Standing posture of monkey 3 weeks after complete bilateral ablation of both supplementary areas.*

Less extensive removal of the supplementary areas, but including the upper bank of the cingulate sulcus medial to area 4, and the medial strip of area 6 produced instinctive grasping in the hands and feet. A unilateral removal in another animal produced weakness of the opposite lower limb for two days and facile visual and tactile grasping for the remainder of the life of the animal, without other change.

In all these animals the lost function was instinctive tactile avoiding, which was replaced by instinctive grasping. This alteration of reaction was most remarkable. In response to any touch the affected hand grasped the contacting object. (Fig. 3, B, C). The delicate slight withdrawals that normally result from unexpected contact were entirely absent in hand and foot. Even with repeated pricking, or scratching the palm of the hand, the result was a grasp. A severe nociceptive stimulus to the dorsum of the hand, or to the forearm, resulted only in a slight simple flexion of wrist and elbow, comparable to a spinal reflex. There was no orientation to the stimulus in elementary withdrawal of this type.

Ablation of both cingulate gyri also abolished avoiding and in addition altered the whole behavior of the animal, by abolishing general avoidance reactions, but the result is not permanent. The experiments cited above convince us that the supplementary area is the focal point of motor projection of tactile avoiding reactions to the limb and body parts, though it is likely that the direction of fully appropriate avoidance behavior requires much more widespread cortical areas, particularly the cingulate gyrus[25]. Since we have found that the less elaborate avoiding reactions in the limbs could persist after pyramid section[26] we maintain that the supplementary area must have an extrapyramidal pathway.

Precentral Cortex

If now we return to the situation that initially intrigued John Fulton we find that, as Botterell and I described, spasticity is not absent following lesions limited to that part of area 4 lying immediately in front of the central sulcus. The flexors of the fingers and wrist, and the extensors of the ankles and toes, develop mild spasticity in the second to third week. Woolsey[19] uses the term "precentral cortex" to mean area 4 of Brodmann exclusive of his supplementary area. We shall use the term precentral cortex in this sense. If the lesion is restricted to the posterior part of this precentral cortex there may be no change whatever in motor power, performance, reaction to stretch or tendon reflexes at more proximal joints. Righting, hopping, placing and tilting can be completely unaffected. In the distal muscles the lost function can only be

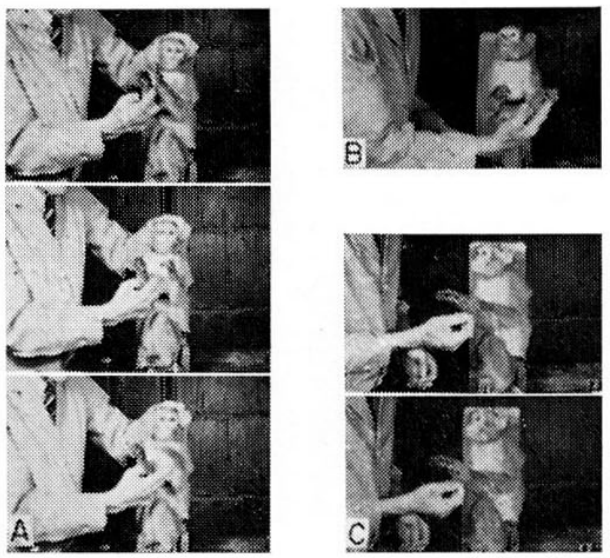


Fig. 3. — *A. Avoiding reactions to light contact (withdrawal of arm, external rotation at shoulder and extension of fingers). B. The grasping reaction to contact as a uniform response in the same animal 5 days after ablation of the right supplementary area. C. The reaction to pin prick (the hand grasps the point of the pin without withdrawing).*

defined as a loss of dexterity in performance. No particular movement is missing, and the finger and thumb can be used for grooming or picking up crumbs, though the movement is slow, not completely dissociated from movement of the other fingers, and requires visual guidance. The released function is an enhancement of the stretch reflex, evident as mild spasticity.

In our original experiments, and in many of the same type performed since for other purposes, we have not found any *abrupt* change in resulting paralysis or spasticity as a result of varying the extent of area 4 lesion. The more the oral part of area 4 is included in the ablation, the more are proximal muscles involved. In other words, we find no reason to set aside any special strip such as the area 4S of Hines. The anatomist[27] has had to abandon any supposed criteria for such a special strip. Gradually, in proportion as the movements at the shoulder and hip joint are involved not only do they in turn become spastic, but hopping and placing become defective and the gait and stance become stiff (Fig. 4). In addition a new and different reaction makes its appearance. This is the *traction reaction,*

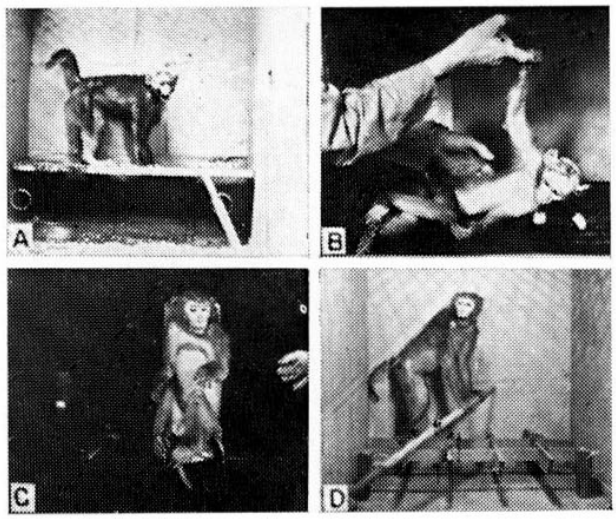


Fig. 4. — *Macaque monkey five months after bilateral ablation of area 4 of Brodmann sparing the supplementary area. B, showing traction reaction in the left arm. C, to show the clawed posture of the fingers and flexor posture of the arms (the legs have been spontaneously withdrawn in sitting posture, in spite of moderate extensor spasticity). D, shows the ability to place on a grid and stick by vision.*

whereby stretch of the retractors and adductors of the shoulder leads to an increased innervation of the flexors of elbow, wrist and fingers. The reaction is greatly enhanced if the flexors of the fingers are stretched at the same time. This traction reaction is greatly increased by certain neck postures, particularly by flexion of the neck and by turning toward the affected limb. It is greatly increased by lying on the opposite side (Fig. 4, B). This is the reaction that was called the grasp reflex by Fulton[18], Bieber and Fulton[28], Fulton and Dow[29], and Richter and Hines[30]. The grasp reflex of clinical neurology in an entirely different phenomenon that is not related to the neck or righting reflexes[31] and is not compatible with frank spasticity. The test of suspending the monkey from a rod [30, 16, 21] is not necessarily a demonstration of the clinical grasp reflex, though it does of course indicate a reflex disorder of grasping. In the clinic such a response of increased clawing of the fingers as a result of traction on the arm is regarded as spasticity. It is commonly seen in capsular hemiplegia, with associated clonic tendon reflexes or clonus. We therefore set aside the grasp reflex of clinical frontal lobe lesions, which in the monkey we find following lesion of areas 8 and 24 and the supplementary area.

In the experiments published by Denny-Brown and Botterell[17] the extent of ablation of precentral cortex on the medial surface was such as to just reach the lip of the cingulate sulcus at one point of opposite the middle of the precentral gyrus. Except for the first five experiments the cortex was removed by subpial resection by suction, a procedure we have used ever since that time. The only part of the supplementary area likely to be damaged by such an ablation is the leg segment. In subsequent experiments we have produced hemiplegic spasticity, with clasp-knife rigidity in the flexors of elbow and extensors of knee, clawed fingers and toes, and with clonic, irradiating tendon reflexes, by ablation of the whole area 4, with careful attention to complete sparing of the supplementary area of Woolsey (Fig. 5). In our experiments such "hemiplegic" spasticity develops only when that part of area 4 which surrounds the su-

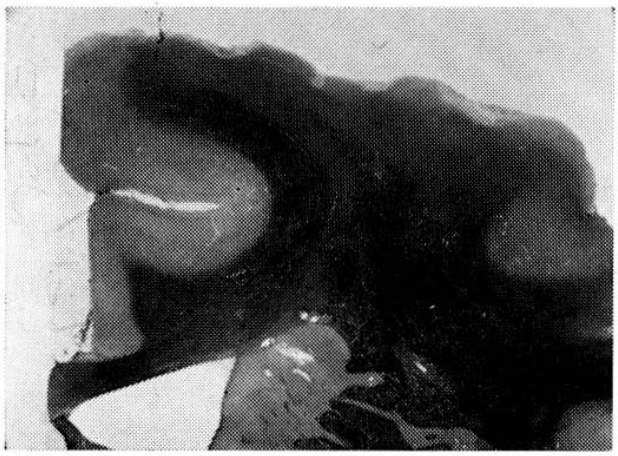


Fig. 5. — *Section through the lesion in the animal shown in Fig. 4. Loyez stain, to show sparing of cingulate sulcus and supplementary areas.*

perior precentral sulcus is included in the cortical ablation. When this area or parts of it remain, spasticity is less, and bipolar stimulation of points in its perimeter elicits extension of the opposite elbow, protraction of shoulder, flexion of the ipsilateral elbow, sometimes flexion of the opposite hip, with occasionally extension of the ipsilateral hip[15]. In full development this synergic movement is one of progression, initiated by the opposite arm. Stimulation can readily facilitate this movement as far forward as area 9 in intact cortex. Yet, in a series of histologically controlled ablations we showed that this proximal elbow and shoulder movement complex produced by stimulation is completely abolished when all the anterior border of area 4 is removed. This excluded the possibility that the synergy was the result of stimulating the intact supplementary area. Inhibition of spontaneous movement can also be facilitated by electrical stimulation to include parts of area 8 and 4, but it disappears completely if area 6 is carefully ablated, leaving the proximal extension synergy of area 4 intact behind the lesion.

Following section of the pyramid in the monkey we found[26] that extension of the contralateral shoulder and elbow, and flexion of the hip, could still be obtained by stimulation of the region of the superior precentral sulcus (Fig. 6). It was abolished by ablation of area 4 (Fig. 6, B) which led to the appearance of strong hemiplegic spasticity in the previously looser limbs. This synergy evidently corresponds to the "quadripedal progression" noted from stimulation of this area by Hines[16]. From area 6 inhibition of spontaneous movements under light anesthesia could readily be produced by stimulation after pyramid section. Along the medial border of the hemisphere, corresponding to medial area 6 we found that flexion of the opposite limbs, often rhythmical, could be produced by stimulation after pyramid section. This may correspond to the "grasping" mentioned by Hines, though we did not observe any activity that could be termed reaching. This flexion activity appeared to us to be identical with that normally produced by bipolar stimu-

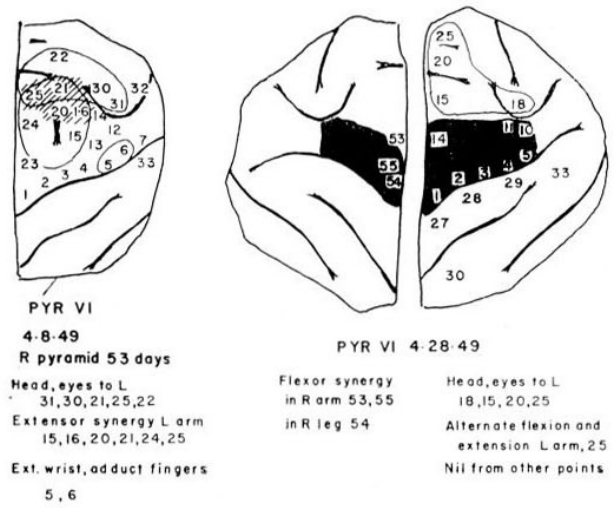


Fig. 6. — *Left, chart of bipolar stimulation of right motor cortex of macaque 53 days after section of right pyramid. A small movement of the left wrist and fingers escaped destruction. The encircled area enclosing points 24, 15, 16, 25 and 21 (centering on point 20) gave extension of the left arm and sometime flexion of right. The oval area enclosing points 25, 22, 21, 30 and 31 gave deviation of head and eyes to the left. Stimulation in the shaded area induced relaxation of all posture and spontaneous movement. All other points gave no response. To the right is the chart of both motor areas of the same animal exposed 20 days later (area 4 and most of area 6 having been excised following the first stimulation). Only the movement of head and eyes remains, except that a flexion alternating movement was obtained from point 25 (supplementary area).*

lation of the supplementary area. It was occasionally associated with a rhythmical grunting vocalization.

We therefore still conclude that a hemiplegic attitudinal pattern of flexion in the upper limb and extension in the lower limb, with increased adductor resistance, is superimposed upon the fundamental release of stretch reflex by inclusion of the region of the superior precentral sulcus in ablations of area 4 of Brodmann. This postural emphasis is associated with increased and irradiating reflexes, more evident in the more spastic muscles. It is not altered by making symmetrical bilateral ablation. The corresponding movement complex elicited by stimulation of these areas are still present after pyramid section.

The Hemiplegic Posture

In the monkey the hemiplegic posture of flexed upper limb and extended lower limb is presented when the animal stands or sits looking forward. It is intensified by lying on the back. The attitude is related to the posture of the head and neck. If the animal is suspended in air by the pelvis, and is slowly turned head down, this posture of flexion of the arms slowly gives way to extension of the arms as the neck is extended, and the lower limbs become flexed (Fig. 7, A, B). The reaction is dependent upon labyrinthine stimulation. Brain[32] long ago maintained that the flexed hemiplegic arm in man could be made to extend by leaning the patient forwards. The reason such reveral is not easily demonstrated in man is that sufficient neck extension has not usually been used for a long enough period (30 to 60 seconds).

Two additional observations are of special importance to the question of spasticity. When the attitude of the limbs change as a result of the maneuvers we have mentioned the reflex pattern also changes. Thus as the arm moves from the flexed to the extended posture the flexor spasticity at the shoulder, elbow and wrist disappears, and with it the clonic irradiation of flexor tendon reflexes, and the traction response in these muscles. Concurrently the extensor stretch reflexes are increased, and a stretch of the flexors of the fingers now induces a strong reinforcement of extensor posture (Fig. 7, C), transforming the limb into a pillar (the positive supporting reaction). The hind limb exhibits a weak traction reaction when the monkey sits in a chair, but gives an intense response if the animal is suspended with head extended (Fig. 7, D). If the neck is strongly and actively flexed while the head is held in normal horizontal position, the hind limb shows a strong positive supporting reaction and no traction response is then obtainable. The tendon reflexes of the extensors are then hyperactive. These reactions do not appear in full development unless the cortex surrounding the superior precentral sulcus is excised. They are most prominent in bilateral precentral cortex ablation without damage to supplementary area and then present remarkably consistent and delicate effects on the posture of the hands which are flexed when the animal leans backwards, extended as he leans forwards.

In the monkey with bilateral ablation of precentral areas there is also present after the third week a facile reaction to tilting, such that the arm on the side to which the

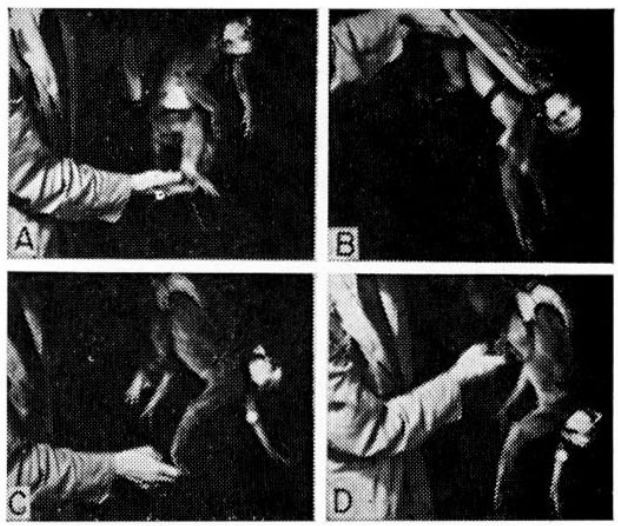


Fig. 7. — *Macaque monkey TM9, 5 weeks after bilateral ablation of area 4 to show A, beginning of positive supporting reaction of lower limb when held upright, B flexion of lower limbs and extension of upper limbs when held head down, C with supporting reaction now in upper limb, and D traction reaction in lower limb.*

Fig. 8. — *Tilting reaction in animal shown in Fig. 7 (TM9).*

animal is tilted abducts and the hand extends and pronates to give support (Fig. 8). The tilt reaction is graded, and proportional to the degree of tilt and can be arrested at any stage simply by arresting the degree of tilt. This is present when the animal is blindfolded and after section of 8th nerves.

It is therefore apparent that the accentuation of spasticity in any muscle group, and the posture of the hand and foot under these circumstances has meaning only in terms of the position of the head and body in space. Hemiplegic spasticity is a dynamic phenomenon, that cannot be wholly assessed in sitting or lying position.

From a very early stage of recovery from area 4 lesion ability to flex the limbs to withdraw from a noxious stimulus makes some recovery. In a late stage these avoiding movements may become adroit (Fig. 9), and may be elicited by contact (Fig. 9, B) or by visual threat, but never quite as delicate as those of the normal animal. These movements are chiefly of flexion of all joints in both limbs, with rotation at wrist and ankle. Individual movements of the fingers are usually in the nature of extension. Adversive movement of the head and contraction of the lips are also common. These avoiding movements are absent if the supplementary area is excised in addition to the precentral cortex. The animal is then more disabled and more spastic than after simple precentral ablation. The ability of the animal to extend the limb is recovered more slowly than after precentral ablation alone, but he can ambulate well if given enough exercise.

The way in which the limbs are used for prehension appears to be same whether an ablation of area 4 includes the supplementary area or not. At first the monkey makes sweeping extensions which hook the wrist around an object and pull it towards the mouth. The mouth is strained towards the object, and the associated flexion of the neck assists the weaker flexion phase of the movement, which is totally ineffective if the head is held turned toward the side of the lesion. The traction reaction has always appeared some days before such purposive movement occurs and the movement itself is facilitated by the same effects (neck flexion, contact of opposite side of body with ground) that facilitate the traction reaction. In the third and later weeks the closure of the fingers is facilitated by pressure in the palm of the hand, without traction on the limb, but this effect is only present when neck and head posture favour a brisk traction reaction. It is not a true

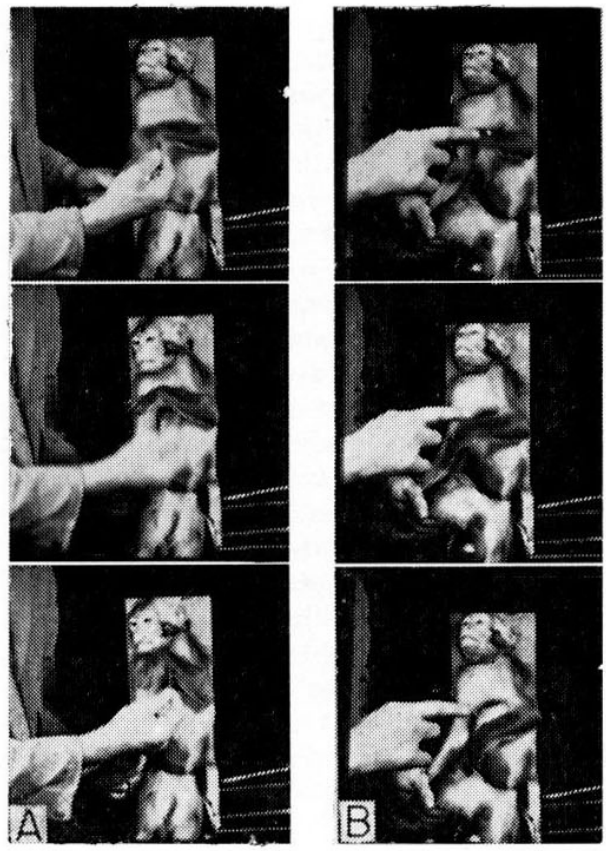


Fig. 9. — *A. Avoiding reaction to pin prick in left arm in monkey D.C.8 four weeks after ablation of right area 4, sparing supplementary area. B. Avoiding reaction to touch in the same limb (called "visual avoiding" because the eyes are not covered).*

grasp reflex for moving contact does not elicit the initial closing responses for a grasp reflex. When the head is extended the reaction disappears. At this time the monkey learns to oppose the thumb to the other fingers to pick up small objects. The movement remains clumsy, for all the fingers flex, and the orientation of the hand is awkward. He can get it to the mouth only by flexion of the head towards the hand, and with some flexion of the arm. The area 4 monkey cannot freely use opposition of the thumb and first finger in any position of the arm or in any position of the head.

Pyramid Section

The observations of Tower on the effect of section of the pyramid in the cat[33, 34] and the monkey[10] were indeed remarkable. Without wishing to detract in any way from their importance, we would point out that here again spasticity was defined as a maximal grade of the disturbance known to the clinician by that name. In the descriptions of Tower it is apparent that the limbs did not, after the first few days, present that grade of complete flail-like inertia that the clinician describes as "flaccidity". The tendon reflexes were described as slow but of wide amplitude, and sustained postures of flexion were present. To the experimentalist the condition of the limb is very different from the flail-like inert freely swinging limbs on the side of a recent hemisection of cervial spinal cord of the monkey. With Dr. Twitchell we ourselves attempted pyramid section of the monkey[26], encountering the same difficulties in avoiding damage to the lemniscus, that others have found. In six of 17 monkeys we succeeded in producing discrete lesions. The effect on movement is that which Tower described, namely a loss of the normal facility in reaching out for objects, yet with retained ability to hook the arm or wrist around a desired object and pull it towards mouth. The animals could walk and run, but the limbs were over flexed and adducted. Placing was absent. Tilting and hopping reactions were absent for a long period. Climbing was poorly executed because the fingers hooked only weakly onto cage wire. The most striking difference from a monkey with area 4 ablation was the absence or feebleness of the traction and positive supporting reactions and the greater intensity of adduction. The limbs correspondingly showed no hemiplegic posture, but only a weak flexion of the arm and hip when sitting, and an extremely poor positive supporting reaction with neck extended.

The stretch reflex was, however, not absent. From the 3rd to 10th day after pyramid section a soft resistance to passive extension of the elbow, wrist and fingers could be felt in the flexors towards the end of stretch. The same type of mild, soft resistance could be felt in the knee extensor. In the second week the retractors and adductors of shoulder, the extensors, flexors and adductors of the hip and the extensors of ankle and toes exhibited the same mild spasticity. A soft resistance could be felt in the extensors of the arm and the flexors of the lower limb. The corresponding tendon reflexes were at first slow and small, but

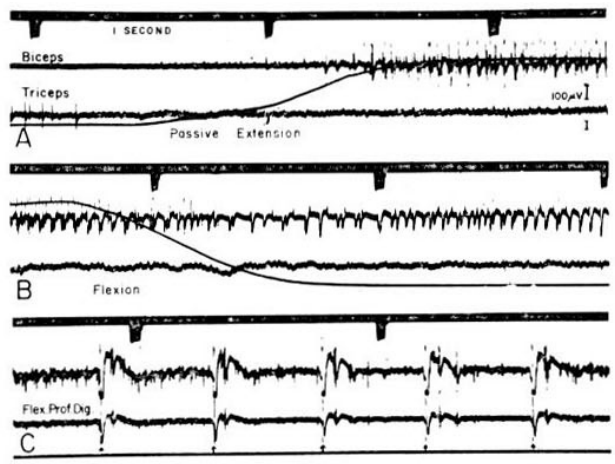


Fig. 10. — *Three strips of electromyogram, each with one second marker at top. A and B show the reaction in biceps and triceps of the left arm during a gradual extension of the elbow, A, followed by flexion, B. Right pyramid sectioned in the medulla 2 weeks earlier (PYR6). In C is shown a series of finger jerks recorded in deep forearm flexor (both leads from same electrode), in another animal (PYR9) with section of corresponding pyramid 19 days earlier. Note double and treble responses.*

in the second week became larger than in the opposite limb. In some animals the biceps, triceps and knee and finger jerks became of +++ grade, with occasional second beats. The arm was regularly held flexed to 90-100° when sitting and the fingers were curled in semiflexion. Electromyograms of biceps and quadriceps showed a regular response of a few units to stretch (Fig. 10), shortening reactions (Fig. 10, B) and repetitive tendon reflexes (Fig. 10, C). We therefore had to conclude that the stretch reflex was more active than in the normal relaxed limb.

If the lesion of the pyramid was submaximal so as to include only the middle third of the tract at the level of the olive (Fig. 11), the change in stretch reflexes was even more remarkable, though more transient. All muscles, both flexors and extensors, then became clearly spastic in the second week with clonic finger and toe jerks, positive Hoffmanns, and very brisk repetitive biceps and quadriceps tendon reflexes. By the fourth week, with increasing use of the limbs, this obvious spasticity subsided to a mild persistent increase over normal level. Yet the hemiplegic posture failed to develop.

We therefore conclude that *increase in stretch reflexes is indeed a pyramidal sign,* though the degree of increase is not such as to lead to any remarkable pattern of posture of the limbs apart from a slight tendency to general flexion and adduction. The element that is missing from the effect of precentral lesion is the postural bias of the traction and supporting reactions, with their associated spread of stretch effects and general heightening of stretch responses. There was more spasticity in the adductor muscles than follows area 4 lesion, with corresponding greater adduction of limbs in walking, and lack of tilt reaction.

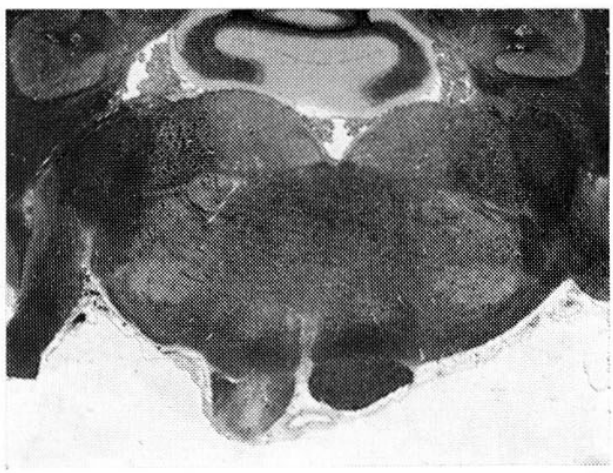


Fig. 11. — *Section of partial pyramid lesion in the animal (PYR17) referred to in the text.*

Parietal Ablation

Overadduction of all limbs is present after pyramid lesion but is not a feature of the upper limbs and is only transient in the lower limbs after pure area 4 ablation. Yet adductor spasticity is a feature of capsular hemiplegia in man, and it is of some interest to enquire whence this element is derived. If ablation of parietal lobe is combined with area 4 ablation spasticity is not only greater than from area 4 lesion alone, but the flexed posture of the arm and extended posture of the leg when sitting is greatly accentuated. It is then several weeks before the animal can flex the leg at all in righting to a sitting or standing posture. The animal is much more spastic than after precentral lesion alone (Fig. 12). He is completely helpless on a grid. In addition both arm and leg are strongly adducted causing difficulty in standing or hopping. There must therefore be an abductor function that was still present after area 4 ablation, but was lost after parietal lesion. The most obvious such function is the lateral tilt and hopping reaction. When the animal is sat on the haunches and suddenly tilted to one side the arm on that side rapidly abducts and externally rotates with extended hand in a movement to provide lateral support. If while standing the animal is propelled laterally the passive abduction of the limb on the side to which he is pushed leads to a step to the side (lateral hopping). After area 4 ablations all these reactions become extremely brisk after the second week. They also become very active after pre-and post-central gyrus ablation (areas

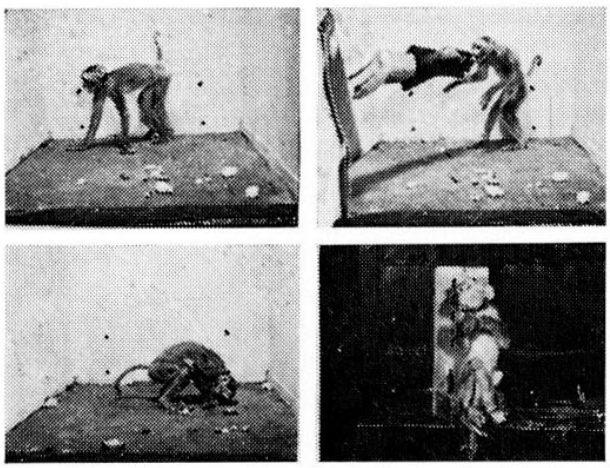


Fig. 12. — *Macaque monkey 5 weeks following ablation of pre- and post-central cortex, with verified sparing of supplementary cortex. To show intense spasticity with over adduction of limbs. He cannot use the arms or hands for eating.*

4, 3, 1, 2). If the posterior parietal cortex including areas 5 and 7 (intraparietal sulcus) is excised delicate tilting reactions are absent. After three or more weeks a turning of the whole body to the side to which the animal is tilted makes its appearance (a more coarse release reaction).

Ablation of these parietal areas alone does not cause spasticity. There is however some mild rigidity of plastic type (present all through passive stretch) greatest in the flexors of both limbs with an increase in tendon reflexes and, under some circumstances, some irradiation to the adductors[25]. We therefore find that adductor spasticity requires a general release of tendon reflexes *plus* the additional bias and facilitation produced by loss of the abductor reactions. In addition parietal lesion heightens any existing spasticity from precentral lesion.

It has been already mentioned that a peculiar feature of the effect of pyramid section, not present after area 4 lesion, is enhancement of the adductor stretch reflexes with profound depression of the tilt and hopping reactions. We conclude that this is due to the loss of those cortico-spinal fibers in the pyramid which are derived from parietal cortex, and which are not affected by area 4 lesion.

The Extrapyramidal Cortical System

The various modifications of body posture we have discussed above have led us to conclude that the fundamental release of stretch reflexes resulting from pyramid lesions is enhanced in different ways by the addition of lesions of supplementary area, area 6 of Brodmann, post-central gyrus and posterior parietal lobe. From each of these areas extrapyramidal mechanisms for modifying movement and posture must exist, besides that which lies within area 4 itself (the extensor synergy).

If our contention that the attitudinal aspects of spasticity are related to these various extrapyramidal factors is true, it should be possible to demonstrate this dystonic disorder alone, by selective damage to extrapyramidal cortex. With the assistance of Dr. Sid Gilman we have recently been able to achieve this by the simple expedient of removing both the precentral (areas 8 and 6) and postcentral (areas 3, 1, 2, 5) extrapyramidal areas on both sides (Fig. 13). The precentral gyrus, with its pial bloodvessels intact, and all medial and lateral arteries and veins, is left as an isolated bridge. The most medial portion of area 4, including the foot representation in supplementary area had to be left intact to avoid damage to elements of area 4. The result of this operation was most remarkable. The animal lay prone with the forelimbs tightly flexed against the body, the

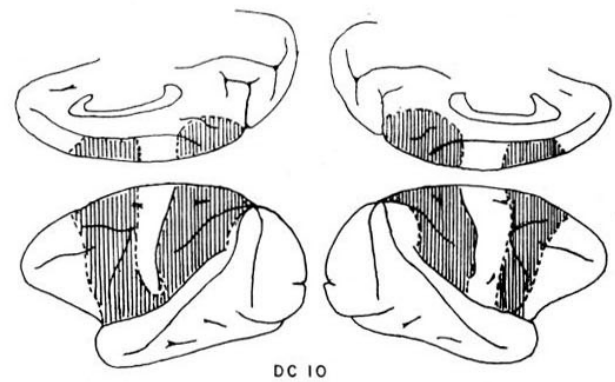


Fig. 13. — *To show the extent of ablation of cortex in monkey DC10, sparing the precentral area and its blood vessels.*

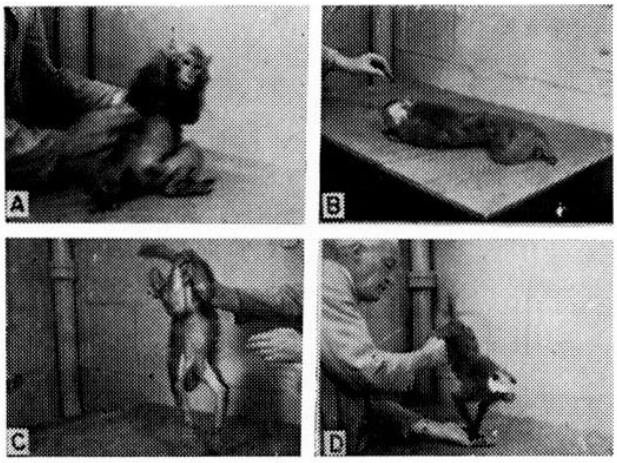


Fig. 14. — *The monkey DC10, ten days after bilateral ablation of the areas shown in Fig. 13. Note ability to right, but inability to stand (B), reversal of posture by inversion of animal (C), and weak positive supporting and head righting (D).*

hind limbs stiffly extended and abducted, in intense dystonia (Fig. 14). When first picked up this rigid posture remained for one to three minutes, then the limbs gradually extended. The extended posture of the fingers in this position was very unstable, alternately opening and closing as in athetosis. If he was held head up the arms flexed again, the legs extended further. If held head down, the arms extended and the lower limbs flexed and adducted (Fig. 14). When the animal was put on a surface the arms flexed and adducted immediately contact was made with them. The animal was unable to eat, the jaws remaining tightly clenched. The head was held rigidly, but the eyes followed the examiner. In all these postures any attempt by the examiner to pull the limb away from its dystonic position was met by an increasing resistance of springy quality, so that the limb would fly back to its original posture when released (Fig. 15).

This dystonic posture will be recognized as that which we reported to follow damage to the outer segment of globus pallidus in the monkey (Denny-Brown). The only difference is that cortical dystonia of the type we describe here is associated with preservation of righting, which was accomplished by a rotary movement of the hindquarters, and by certain fragmentary involuntary movements which will be described in detail elsewhere.

Positive supporting reactions were obtained in the dependent limbs (Fig. 14, D) but were not sufficiently strong to support the animal. Electromyograms revealed an intense neuronal discharge in biceps when the animal sat upright (Fig. 16, A), abolished by turning the animal head down, when dystonic discharge of the same characteristics appeared in the brachial triceps (Fig. 16, B, C). Against a background of dystonic discharge tendon taps produced only a very brief silent period (Fig. 17, B).

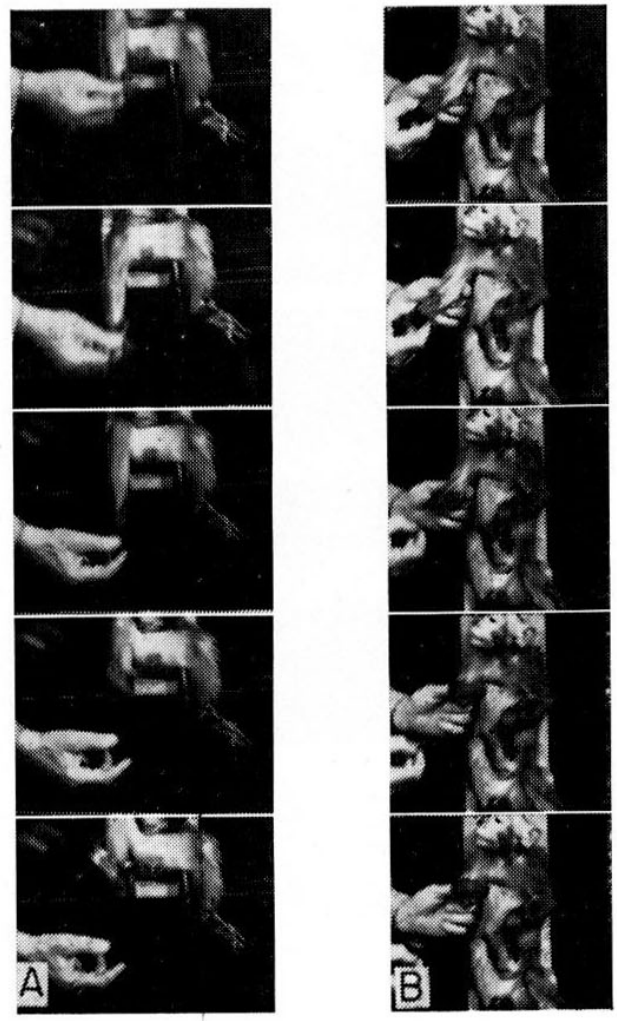


Fig. 15. — *As in Fig. 14 to show the springy quality of the flexion dystonia. In A the right leg is pulled down by the examiner and flies back to resting posture on release in the two lower figures. In B the right arm is pulled into extension and released (3 lower figures).*

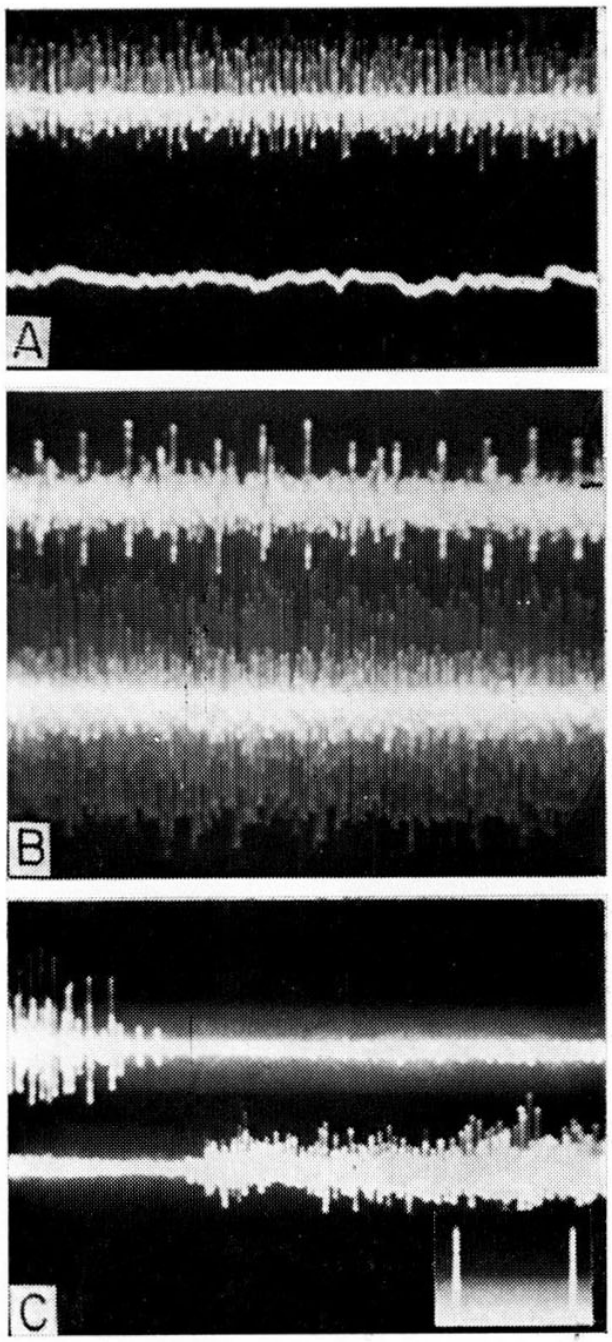


Fig. 16. — *Electromyogram of the animal DC10 biceps above, triceps below, 28th day after ablation made bilateral. In A the animal is suspended in air in sitting posture. The dystonic discharge is solely in biceps. In B the animal is suspended head down. The dystonic discharge has almost ceased in biceps and has now become intense in triceps. In C is shown the transition from head up at the beginning of the record to head down at the end. Time mark 0.5 sec. interval, height of stroke 500 microvolt.*

When by manipulating the posture of the animal the dystonia was lessened the tendon reflex was of normal intensity with normal silent period. If mild dystonia was already present, a tendon tap induced enhanced discharge following the jerk for periods of 5 to 10 seconds.

After section of both 8th nerves the dystonia changed to an intense flexion dystonia of the type seen after lesions of globus pallidus (Fig. 18). Each tendon reflex then elicited a remarkably prolonged tonic discharge. The dystonic posture was then constant.

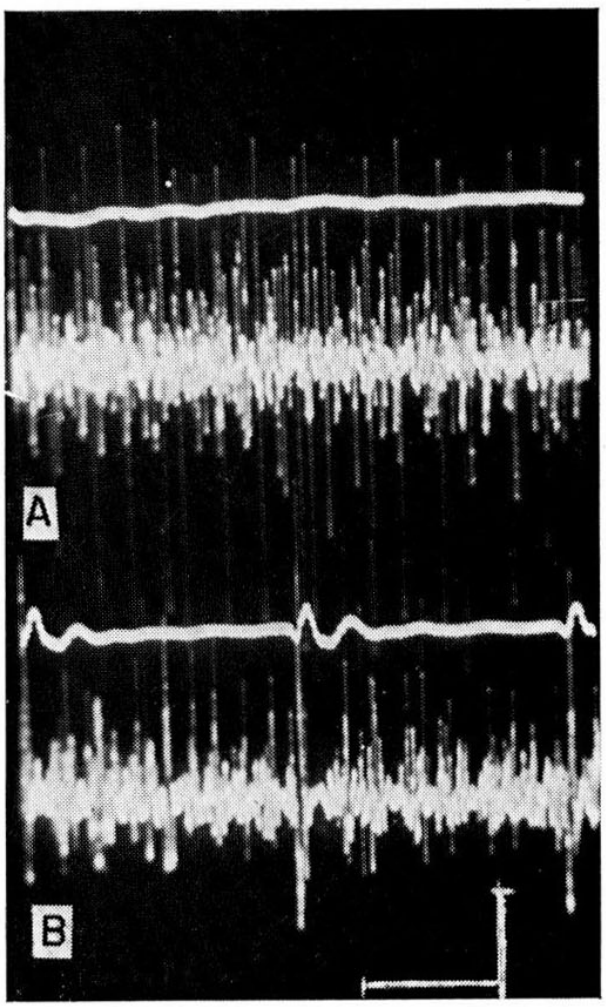


Fig. 17. — *As Fig. 16, two tracings of electromyogram of dystonia (each with mechanogram above) in biceps, animal sitting. A series of 3 tendon reflexes is shown in the lower trace. Note relatively small fast jerks with brief silent period. Calibration 0.5 sec., 200 microvolt.*

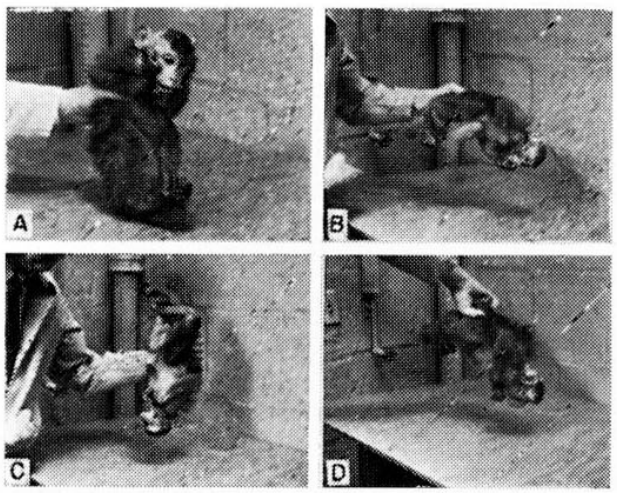


Fig. 18. — *The macaque monkey DC10, 4 weeks after bilateral ablation of extrapyramidal cortical areas and 3 days after bilateral 8th nerve section.*

This dystonia was seen in full development only when the lesion was bilateral. At the unilateral stage it was evident as moderate plastic flexion of the opposite limbs with extended fingers and toes. The extrapyramidal cortical motor mechanisms have therefore a considerable bilateral positive effect from each hemisphere.

The Multiple Components of Spasticity

It has been shown that the original observation of John Fulton of the absence of classical spastic hemiplegia from a lesion restricted to the areas of limb representation in the precentral gyrus is of great importance in the understanding of the factors underlying the cortical control of movement. The problem turns out not to be resolvable in terms of spasticity and flaccidity as such, or to Brodmann'a area 6 and 4, but to be related to multiple elements in the cortical control of spinal and brain-stem levels of organization. These can be independently affected by selective lesion of one or other part of the cortical areas controlling movement, or of the pyramid. We maintain that loss of pyramidal innervation leads to increase of the stretch and tendon reflexes, though the result is only the mildest type of clinical spasticity, without distinctive attitudinal pattern. Release of labyrinthine function is not associated with increased reflexes, or with the response to stretch that we call spasticity, but nevertheless profoundly influences any release of stretch reflexes resulting from any other lesion. Release of neck reflexes can result in irradiation of tendon and stretch reflexes. It is the combination of release of stretch reflexes with one or both of these other types of reaction that determines the most characteristic features of hemiplegic spasticity. Further complications are introduced by the release of body contact righting reactions that are associated with parietal lesion. Disintegration of control of tilting and hopping reactions is a secondary result of area 4 or parietal lobe lesion, leading to increase in spasticity of adductors. These reactions are modifications of body contact reflexes and proprioceptive responses, both of which are released. They are in part compensated for by either area 4 or parietal lobe, but are permanently lost when both are excised.

The supplementary motor cortex is involved in elaboration of withdrawal reactions, loss of which results in some release of the stretch reflexes of flexor muscles and increase in body contact responses. This change alone is not impressive either in terms of loss of motor performance or in spastic postures, though the grasp reflex and tonic flexion are present, but when added to the type of spasticity resulting from lesion of precentral motor area it aiso results in enhancement of spastic phenomena already present.

Paralysis is a relative term, implying loss of one or more of the many factors that contribute to movement. At the cortical level there is evidently an interaction between several different activators, some of which are antagonistic to each other. Loss of one factor leads not only to overactivity of others at cortical level (transcortical release, as in frontal lobe grasping), but to overactivity of the low level mechanism of the lost reaction (tonic innervation). It is remarkable that a total lesion of the hemisphere is less disabling than one where area 4 alone remains intact, for

in this case the precentral motor cortex retains its control of motor centers yet has lost its natural activating system in the extrapyramidal cortical areas. The outcome is then an intense dystonia such as we have described above. As we have already deduced from earlier studies[35] the pyramidal system is useless without its extrapyramidal substrate. The natural function of all these areas of cerebral cortex, whether they have pyramidal or extrapyramidal pathways, is interdependent. Release cannot be explained by loss of inhibition. Nevertheless it is possible to separate to a large extent the symptoms of loss of differentiation of extrapyramidal mechanisms from those that result from pyramidal defect. Each cortical area is related to the others in such a way that it does not appear possible to separate two independent motor systems. Rather it appears that the more complex are only specialized aspects of the less complex.

SUMMARY

John Fulton's application of modern neurosurgical methods to the study of the physiology of the primate brain inaugurated a new era in cerebral physiology. The original observation of Fulton and Keller of the absence of the usual phenomena of spasticity after removal of a limb area of precentral gyrus began a controversy over spasticity resulting from cerebral lesions that has still not been satisfactorily resolved.

The data regarding the functions of the pyramid, of areas 4 and 6 of Brodmann, and of the supplementary area of Penfield and Woolsey is briefly reviewed in the light of the author's own experience. Spasticity is found not to be a single entity but a basic state of mildly increased stretch reflexes, upon which are superimposed patterns of attitude reflecting the special extrapyramidal functions of the different motor areas concerned. The typical hemiplegic attitude (flexed upper limb, extended lower limb) is found to result from damage to the extrapyramidal component of area 4. A pattern of flexion of all limbs results from damage to the supplementary area. Adduction of the upper limbs results from parietal lesion. The total pattern of spasticity, with capsular lesion results from a combination of all these. The meaning of release of these various extrapyramidal patterns is briefly discussed.

RESUMEN

La aplicación de modernos métodos neuroquirúrgicos al estudio de la fisiología del cerebro de los primates por parte del Dr. John Fulton inauguró una nueva era en la fisiología del cerebro. La observación original de Fulton y Keller de la ausencia de los habituales fenómenos de espasticidad luego de la extirpación del área de la pierna de la circunvolución precentral originó una controversia sobre la espasticidad resultante de las lesiones cerebrales, que aún no ha sido satisfactoriamente resuelta.

Los datos concernientes a las funciones de las áreas 4 y 6 de Brodmann, y del área suplementaria de Penfield y Woolsey son brevemente considerados a la luz de la experiencia del autor. Se descubre que la espasticidad no es una entidad simple sino un estado básico de reflejos de distensión ligeramente aumentados, de acuerdo a los cuales se imponen esquemas de actitudes que reflejan las funciones extrapiramidales especiales de las diferentes áreas motoras involucradas. La actitud hemipléjica típica (miembro superior flexionado, miembro inferior extendido) aparece como resultado de una lesión en el componente extrapiramidal del área 4. Un síndrome de flexión de todos los miembros resulta de la lesión en el área suplementaria. La adducción de los miembros superiores resulta de una lesión parietal. El cuadro total de espasticidad por lesión capsular resulta de una combinación de todas ellas. Se discute brevemente el significado de la liberación de estos diversos esquemas extrapiramidales.

RESUME

L'application de méthodes neurochirurgiques modernes à l'étude de la physiologie du cerveau des primates, introduite par John Fulton, inaugura une ère nouvelle dans la physiologie du cerveau. L'observation originale de Fulton et Keller de l'absence des phénomènes habituels de spasticité après avoir enlevé une aire de la jambe de gyrus précentral, déclencha une controverse autour de la spasticité résultant des lésions cérébrales, qui n'a pas encore été satisfactoirement résolue.

Les données concernant les fonctions de la pyramide, des aires 4 et 6 de Brodmann, et de l'aire supplémentaire de Penfield et Woolsey est brièvement considerée, à la lumière des expériences de l'auteur. La spasticité n'apparaît pas comme une entité simple mais comme un état basique de réflexes en extension légèrement augmentés, selon lesquels s'imposent des schémas d'attitudes, qui reflètent les fonctions extrapyramidales spéciales des différentes aires motrices en question. L'attitude hémiplegique typique (membre supérieur en flexion, membre inférieur étendu) apparaît comme résultat d'une lésion dans le component extrapyramidal de l'aire 4. Un schéma de flexion de tous les membres résulte de la lésion dans l'aire supplémentaire. L'adduction des membres supérieurs résulte d'une lésion pariétale. Le schéma total de spasticité avec lésion capsulaire résulte de toutes ces lésions, combinées. On discute brièvement la disparition de ces divers schémas extrapyramidals.

ZUSAMMENFASSUNG

John Fultons Anwendung moderner neurochirurgischer Methoden, um die Physiologie des Primatengehirns zu studieren, eroeffnete eine neue Aera der Gehirnphysiologie. Die urspruenglich von Fulton und Keller gemachte Beobachtung des Ausbleibens der gewoehnlichen spastichen Phaenomene nach Abtragung eines Gliederfeldes in der praezentralen Hirnwindung, war der Beginn einer Uneinigkeit der Meinungen ueber die Spastizitaet durch zerebrale Laesionen, die noch nicht zur Zufriedenheit geloest worden ist.

Die Daten ueber die Funktionen der pyramidalen Zonen, der Brodmannschen Felder 4 und 6 und der supplementaeren Felder von Penfield und Woolsey sind kurz im Lichte der eigenen Erfahrungen des Autors gestreift worden. Man fand, dass die Spastizitaet keine isolierte Einheit ist, sondern der Grundzustand leicht vermehrter Dehnungsreflexe, welchen Haltungszustaende ueberlagert sind, bie besondere extra-pyramidale Funktionen der verschiedenen betroffenen motorischen Zonen widerspiegeln. Die typische hemiplegische Haltung (gebeugte obere und gestreckte untere Gliedmassen) sind das Ergebnis einer Schaedigung der extrapyramidalen Komponente des Feldes Nº 4. Die Flexion aller Gliedmassen entsteht durch die Schaedigung der supplementaeren Zone. Die Adduktion der oberen Gliedmassen entsteht durch Laesion des Scheitellappens. Der totale Zustand der Spastizitaet bei der Kapsellaesion entsteht durch die Kombination all dieser Schaedigungen. Es wird kurz die Entstehung all dieser verschiedenen extrapyramidalen Zustaende besprochen.

REFERENCES

(1) *Fulton, J. F.*: Functional Localization in Relation to Frontal Lobotomy. Oxford University Press, New York, 1949.

(2) *Fulton, J. F.*: Frontal Lobotomy and Affective Behavior. Norton Co., New York, 1951.

(3) *Fulton, J. F.*: Physiologie des Lobes Frontaux et du Cervelet. Etude Expérimentale et Clinique. Masson, Paris, 1953.

(4) *Fulton, J. F., and Keller, A. D.*: The Sign of Babinski. A Study of the Evolution of Cortical Dominance. C. C. Thomas, Springfield, Ill., 1932.

(5) *Fulton, J. F.*, and *Kennard, M. A.*: A study of flaccid and spastic paralyses produced by lesions of the cerebral cortex of primates. Res. Publ. Ass. nerv. ment. Dis. *13*: 158-210, 1934.

(6) *Fulton, J. F.*: Spasticity and the frontal lobes. A review. New Eng. J. Med. *217*: 1017-1024, 1937.

(7) *Kennard, M. A.; Viets, H. R.*, and *Fulton, J. F.*: The syndrome of the premotor cortex in man: impairment of skilled movements, forced grasping, spasticity and vasomotor disturbances. Brain *57*: 69-84, 1934.

(8) *Walshe, F. M. R.*; On the "syndrome of the premotor cortex" (Fulton) and the definition of the terms "premotor" and "motor": with a consideration of Jackson's views on the cortical representation of movements. Brain *58*: 49-80, 1935.

(9) *Walshe, F. M. R.*: The disorders of motor function following an ablation of part of the "leg area" of the cortex in man. Brain *58*: 81-85, 1935.

(10) *Tower, S. S.*: Pyramidal lesion in the monkey. Brain *63*: 36-90, 1940.

(11) *Hines, M.*: Control of movements by the cerebral cortex in primates. Biol. Rev. *18*: 1-31, 1943.

(12) *Brown, W. J.*, and *Fang, H. C. H.*: Spastic hemiplegia in man: lack of flaccidity in lesion of pyramidal tract. Neurolgy *11*: 829-835, 1961.

(13) *Meyer, J. S.*, and *Herndon, R. M.*: Bilateral infarction of the pyramidal tracts in man. Neurology *12*: 637-642, 1962.

(14) *Hines, M.*: The "motor" cortex. Bull. Johns Hopk. Hosp. *60*: 313-336, 1937.

(15) *McCulloch, W. S.*: Cortico-cortical connections, Chap. VIII of *The Precentral Motor Cortex* Ed. by P. Bucy. Univ. of Illinois Press, Urbana, Illinois, 1944.

(16) *Hines, M.*: The control of muscular activity by the central nervous system, Vol 2. Chap. XI, p. 467, in *The Structure and Function of Muscle*, ed. by G. H. Bourne, New York, 1960. Academic Press.

(17) *Denny-Brown, D.*, and *Botterell, E. H.*: The motor function of the agranular frontal cortex. Res. Publ. Assn. nerv. ment. Dis. *27*: 235-345, 1947.

(18) *Fulton, J. F.*: *Physiology of the Nervous System.* 2nd. Ed. Oxford University Press. New York, 1943.

(19) *Woolsey, C. N.; Settlage, P. H.; Meuer, D. R.; Sencer, W.; Hamuy, T. P.*, and *Travis, A. M.*: Paterns of localization in precentral and "supplementary" motor areas and their relation to the concept of a premotor area. Rev. Publ. Assn. nerv. ment. Dis. *30*: 238-264, 1952.

(20) *Denny-Brown, D.*: The frontal lobes and their functions. Chap. 2, p. 13, in *Modern Trends in Neurology*, ed. by A. Feiling, Hoeber, New York, 1951.

(21) *Travis, A. M.*: Neurological deficiencies following supplementary motor area lesions in *Macaca Mulatta*. Brain *78*: 174-198, 1955.

(22) *Travis, A. M.*: Neurological deficiencies after ablation of the precentral motor area in *Macaca Mulatta*. Brain *78*: 155-173, 1955.

(23) *Denny-Brown, D.*: *The Basal Ganglia and their Relation to Disorders of Movement.* Oxford University Press, London, 1962.

(24) *Hines, M.*: Significance of the precentral motor cortex. Chap XVIII in *The Precentral Motor Cortex*, ed. by P. Bucy, Univ. Illinois Press, Urbana, Illinois, 1944.

(25) *Denny-Brown, D.*, and *Chambers, R. A.*: The parietal lobe and behavior. Proc. Assn. Research nerv. ment. Dis. *36*: 35-117, 1958.

(26) *Denny-Brown, D.*, and *Twitchell, T.*: Pyramid section in the monkey (unpublished).

(27) *Bonin, G. von*, and *Bailey, P.*: *The Neocortex of Macaca Mulatta.* University of Illinois Press, Urbana, Illinois, 1947.

(28) *Bieber, I.*, and *Fulton, J. F.*: The relation of the cerebral cortex to the grasp reflex and to the postural and righting reflexes. Arch. Neurol. Psychiat. Chicago *39*: 435-454, 1938.

(29) *Fulton, J. F.*, and *Dow, R. S.*: Postural neck reflexes in the labyrinthectomized monkey and their effect on the grasp reflex. J. Neurophysiol. *1*: 455-462, 1938.

(30) *Richter, D. P.*, and *Hines, M.*: The production of the "grasp reflex" in adult macaques by experimental frontal lobe lesions. Res. Publ. Assn. nerv. ment. Dis. *13*: 211-224, 1934.

(31) *Seyffarth, H.*, and *Denny-Brown, D.*: The grasp reflex and the instinctive grasp reaction. Brain *71*: 109-183, 1948.

(32) *Brain, W. R.*: On the significance of the flexion posture of the upper limb in hemiplegia, with an account of a quadripedal extensor reflex. Brain *50*: 113-137, 1927.

(33) *Tower, S.*: The dissociation of cortical excitation from cortical inhibition by pyramid section, and the syndrome of that lesion in the cat. Brain *58*: 238-255, 1935.

(34) *Tower, S.*: Extrapyramidal action from the cat's cerebral cortex: motor and inhibitory. Brain *59*: 408-444, 1936.

(35) *Denny-Brown, D.*: On singleness of purpose. Arch. Neurol. Chicago *3*: 613-619, 1960.

Goal-seeking Controls Affecting Both Motor and Sensory Systems

R. B. LIVINGSTON, M. D.
National Institutes of Health. Public Health Service.
U. S. Department of Health, Education and Welfare,
Bethesda, Maryland.

INTRODUCTORY TRIBUTE TO JOHN FARQUHAR FULTON

Not long ago we were encouraged to establish a new laboratory at the National Institutes of Health. We proposed to call the new research enterprise The John Fulton Laboratory. This name would link the purposes and activities of the laboratory with Fulton's own research endeavors and would bring something of the luster of his name to Bethesda. Identifying the new laboratory with John Fulton would peg aspirations high and keep them high. The name would enable us in some measure to expiate ourselves from indebtedness to John Fulton: we could openly attempt to pass along to others something of the Fultonian spirit and style of fostering a laboratory "family". Fulton's name was desirable also because it would convey immediately the breadth of subject matter needed for studying integrative mechanisms of the nervous system. Yet, certainly, the name of John Fulton, without further qualification, would imply perhaps too much. It would imply not only a broad scope of biological and medical interests, but equally obviously, it would also imply scholarly devotion to humanism, bibliography, and academic internationalism. Therefore, a more limiting phrase should be added so that the full title would read "The John Fulton Laboratory of Neuro-Psycho-Socio-Biology".

Needless to say, officials of the NIH objected to an eponymic title for the laboratory: no other laboratory is presently named for anyone; the practice might provide a precedent; no telling what confusion could ensue if it became traditional to name laboratories for people instead of for diseases, or research disciplines. Ultimately, authorization was granted to call the new research entity the Laboratory of Neurobiology. By good fortune, our neighbors in the National Institute of Mental Health, Hal Rosvold and Mortimer Mishkin, also devotees of John Fulton, were able to change the name of their research program from Animal Behavior to Psychobiology. Together, then, the two groups would incorporate both the *neuro* and the *psycho* parts of neuro-psycho-socio-biology.

We recognized certain Fultonian organizational ingredients: laboratories and lab assistants were required, office-laboratories, animal quarters, and a space for discussion and writing which could be entirely surrounded by the most pertinent and exciting books. Another ingredient, equally in the Fultonian tradition, would be the manner of conducting the continuing intellectual search. Visitors would be entertained as royally as we could manage with the limited means of sandwiches and tea! Visitors and staff alike would be invited to share ideas in an atmosphere congenial for breaking away from conceptual traditions.

In this spirit, Ichiji Tasaki, Arnold Starr, Gabriel Frommer, Peter Carmel, Constantine Spyropoulos, Muneo Shimamura, Bo Gernandt, Konrad Akert, Rosalind Mari-

mont, David Galin, and other regulars, together with transient visitors from the United States and many other countries, joined in undertaking three interdependent lines of inquiry. One involved, of course, a diversified program of experimental research. Another involved analysis of scientific concepts developed by individuals who have contributed importantly to different areas of neuro-psycho-socio-biology. A third line

Fig. 1. — *John Farquhar Fulton, in the 1930s, a few years after accepting the chair of physiology at Yale University.*

of inquiry involved analysis of problems, the solution to which must depend on conceptual convergence of more than one research discipline.

It is not possible to present an account here of the total research undertakings of the laboratory; only two examples in one area of research will be submitted. But by way of introduction, I shall describe briefly how we conducted the biographical analysis and the problem-oriented interdisciplinary study.

Biographical Analysis

We undertook a careful review of the life and works of John Fulton and pursued a similar pattern, considering both the man and his ideas, among the following: Charles S. Sherrington, Karl S. Lashley, J. Hughlings Jackson, Ivan Petrovich Pavlov, Walter Rudolph Hess, William James, Ivan Mikhailovich Sechenov, B. F. Skinner, C. Judson Herrick, George Ellett Coghill, Adelbert Ames, Jr., and several others.

First, an illustrated biography was presented. Then we prepared as complete a bibliography of the man as we could uncover, and identified those publications which would best exemplify and characterize his principal conceptual contributions. These were grouped suitably for individual presentation of the man's main intellectual developments and research experiences in the origins of the man's ideas, in a form suitable for group study and discussion. Thereafter began the hard work: We attempted to characterize, as well as we could, mainly in the man's own words, what were his central scientific contributions. Next, we tried objectively to reinforce and amend these contributions according to evidence brought to light subsequent to his publications. Finally, we undertook to apply

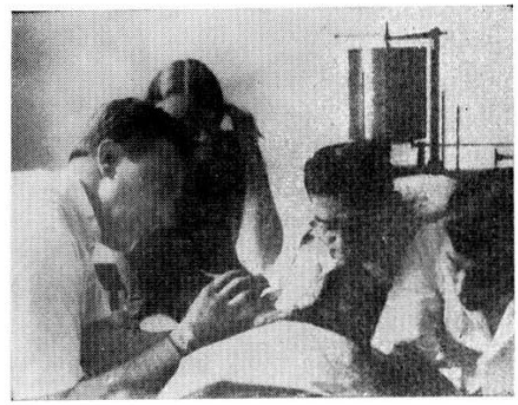


Fig. 2. — *Fulton, Margaret A. Kennard and Morris B. Bender, with the assistance of Leslie R. V. Kerby, conducting a typical exploration of motor cortex in monkey. (Photograph taken by Sir Geoffrey Jefferson during one of his visits to New Haven.)*

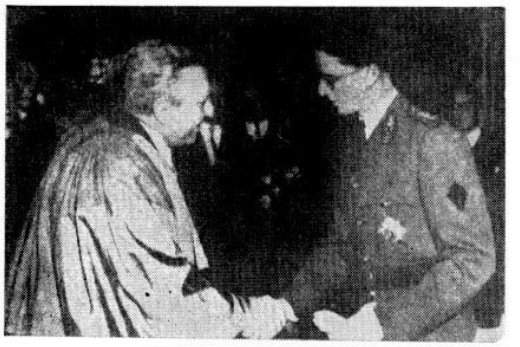


Fig. 3. — *Fulton, as* Titulaire de la Chaire Francqui *at the University of Louvain where, in 1951-52, he presented, in French, a series of lectures later published under the title* Physiologie des lobes frontaux et du cervelet, *shown receiving an honorary degree of Doctor of Medicine from the young King Baudouin.*

these amended conceptions to the major lines of research being pursued in the Laboratory of Neurobiology.

This effort was rewarding and refreshing. We encountered several concepts not currently in fashion which are nevertheless pertinent to contemporary scientific developments. We learned, furthermore, that a careful examination of a man's life and a serious analysis of his ideas gave us a warm, personal attachment to him, a lasting respect for his talents, accomplishments, and originality of views.

Interdisciplinary Seminars

About twice a year we sponsored working seminars for neurophysiologists and psychologists in the Washington area. Each of these seminars was made up of from 20 to 40 evening hours devoted to an interdisciplinary approach to a single subject or problem area, one broad enough so that no single research discipline could provide an adequate approach. For example, various individual seminars dealt with nervous mechanisms relating to perception, motivation, memory, visceral functions, and behavior.

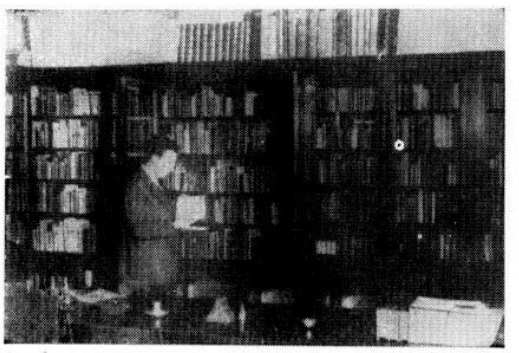


Fig. 4. — *Fulton, in the midst of his own irreplaceable collection of journals and monographs on the nervous system. Fulton possessed an uncanny ability to fly about in that room, locating particular citations almost instantaneously* à propos *nearly any topic in physiology, medicine or scholarly learning in general, and, equally characteristically, to modestly remember the material verbatim.*

Fig. 5. — *Fulton, with Lucia P. (Wheatland) Fulton, his charming, cordial and buoyant life's companion, under the portico of their home, 100 Deepwood Drive, in New Haven, Connecticut. Their home stands as a beacon radiating love and warmth to all corners of the world. As hosts, both at home and abroad, they probably contributed more to international understanding than any other individuals in academic life.*

Fig. 6. — *Fulton, with George D. Davis and Robert B. Livingston, operating on a chimpanzee in the physiology amphitheater, as a class demonstration of sterile operating procedures and stimulation of the excitable motor cortex; about a year before Fulton's retirement from the chair of physiology. (Photograph by Allen M. Sher, then a graduate student in physiology.)*

Each seminar series began with the most explicit and concrete lines of evidence and undertook group mutual education by easy stages until we were all venturing uncertainly in the *terra incognita* surrounding the headwaters of each of these grand problems. Through this concerted effort, we did not expect to contribute to the advancement of knowledge except among ourselves. Typically, these seminars disabused us of some of our commonplace heritage of misinformation and clutter of preconceptions, and assisted us in the development of more constructively adaptive attitudes and clusters of factual information relating to each of the subjects studied. Considerable satisfaction followed from the fact that we were all equally humbled by the magnitude of the problems we were addressing.

Our aim was consistently to seek *general principles relating to integrative actions of the nervous system.* We share confidence that this may be accomplishable through carefully examining the new patterns of regularity, coherence, and integration which emerge at each of the successively more complex levels of development and organization in biological systems. Such emergent patterns reflect properties that are not only integral to the successively more complex systems but which cannot be predicted from the properties of the parts of which they are composed. Emergent qualities of integration —emergent mechanisms of functional organization— these are what we seek to comprehend. Our search in reference to the nervous system was deliberately in the tradition of Fulton, and his intellectual antecedent, Sir Charles Sherrington. Perhaps the greatest stumbling block was encountered each time we attempted to shift the discourse from one level of complexity to another. The entertainment value of innumerable facts and concepts contained among descriptions and explanations pertinent to any given level of complexity is virtually inexhaustible; we invariably tend to become imbedded in the discourse of any given context.

It may possibly turn out that the integrative mechanisms of the nervous system

Fig. 7. — *Fulton, in the late 1950s, shortly after accepting the chair of the history of medicine at Yale University.*

as a whole will ultimately be revealed as a result of an adequate investigation of single neurons. Or, perhaps such insight will be found to depend predominantly or entirely on an understanding of subcellular molecular complexes, or the physico-chemical mechanisms underlying molecules, or atomic particles, or perhaps atomic field forces. We adhere to a different assumption: that each of the successively more complex systems from subatomic all the way to social levels of organization enjoys potentialities that cannot be completely expressed by the isolated parts of that system. We believe an adequate scientific pursuit of integrative processes must bridge the gaps between different levels of organizational complexity and among different traditions of scientific discourse. For a study of integrative mechanisms of the nervous system, this means, ultimately, familiarity with the physico-chemical, molecular, cellular, tissue, organ, individual physiological and psychological, and societal integrative patterns. We need to study complex systems *as systems,* and to examine them at several levels of complexity by way of finding illustrations of emergent patterns of integration. To members of the Fulton Society, it must be obvious why it would be an advantage to identify this kind of enterprise with the traditions of John Fulton.

SOME EXPERIMENTAL FINDINGS RELATING TO INTEGRATION

In the spirit of this meeting, I should like to submit interpretations relating to two lines of evidence which reveal some elementary mechanisms involved in integration of the nervous system. Perhaps these two lines of evidence would have been of interest to John Fulton. What we miss are his insights. The two examples are selected because they involve two distinctly different levels of organizational complexity; yet each reflects mechanisms of integration which may be characterized as "goal-seeking". Each deals with longitudinal systems involved in sensorimotor coordination along the neuraxis. At spinal and lower brainstem levels of integration, the mechanisms for signal handling are relatively fixed — built into the nervous system chassis, so to speak. They appear to be stable and arranged according to *species* rather than individual experiences. Thus, the same input-output patterns are presumed to perform in fundamentally the same way among all members of the species. The second line of experimental evidence also deals with lengthwise organization of the neuraxis, in this case, the auditory pathway. Here we find components which reflect modifiability of signal transmission according to particular experiences of the individual. Such modifiability appears to relate to phenomena of learning and memory. In both examples of integrative mechanisms —in the longitudinal sensorimotor coordination system and in the auditory pathway— the net effect seems to be directive towards enabling the organism to make more appropriate and simpler responses to a given environment.

I. LONGITUDINAL SENSORIMOTOR COORDINATION

The Segmental Reflex

Fig. 8 was sketched by hand by John Fulton. It appears in the monograph, *Muscular contraction and the reflex control of movement*[3], published when Fulton was 25 years of age and had recently completed his Doctorate in Philosophy at Oxford under Sherrington. The figure is entitled: "A diagram of a simple spinal reflex arc. The knee-jerk is believed to involve reflex paths of this order of simplicity". Let us briefly consider some of the concepts implied in our present day understanding of the "simple reflex arc". First of all, we can identify in Fulton's diagram the stretch *afferents* (s. a.) and the *efferent* unit serving muscle fibers. Such a reflex loop is known to be operationally circular, being completed both centrally and peripherally; centrally by nervous connections like those illustrated in Fulton's diagram, and peripherally by virtue of the fact that any given position or movement of the limb yields a characteristic sensory input pattern. Both the central and peripheral elements of the reflex

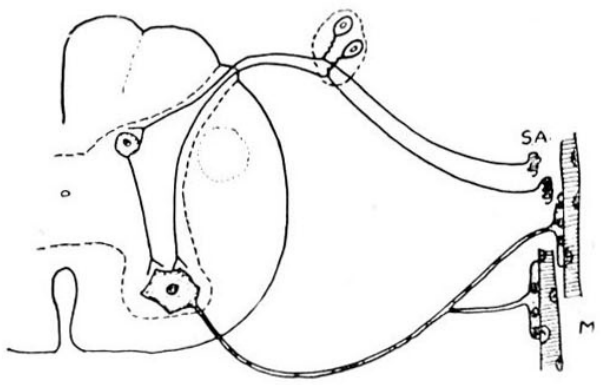


Fig. 8. — *A diagram drawn by Fulton, published in his monograph* Muscular contraction and the reflex control of movement[3].

loop are subject to interventions other than can be depicted at the segmental level; that is, the circuits involved are open rather than closed circuits. All this, of course, was well known to Fulton at the time that he drew this figure.

In recent years further complexity has been added: For example, *gamma* efferents which supply intrafusal muscle fibers in the muscle spindles. Excitation of these efferent units does not evoke an overt muscular contraction, but instead changes the tension in the intrafusal fiber which, in turn, alters the bias of sensory end-organs within the spindle. Fulton was deeply involved in the research leading up to this revelation. The gamma unit constitutes an *efferent fiber which rather directly subserves sensory functions.* Sensory signals, thus affected, feed back to further modify a wide variety of central motor controls. Other efferent fibers are known to discharge in the vicinity of individual sensory receptors in the skin and elsewhere. Stimulation of these efferents has been shown to markedly alter sensory input pattern[10]. In addition, there are both diffuse and localized chemical linkages which can also modify these neuronal activities. I refer to neurohumoral and neurohormonal agents which can modulate motor and sensory activity, both centrally and peripherally.

Finally, I invite your attention to a fact which was brought home to me first by Paul Yakovlev when he was at Yale. Looking at a diagram such as Fig. 8, Yakovlev said: "We can be reasonably unambiguous as to what is *afferent* and what is *efferent* when we are talking about peripheral nerves. But what about the 'internuncial neuron'? It is *efferent* from the point of view of the *dorsal* horn region of the spinal cord, but it is *afferent* from the point of view of the *ventral* horn of the spinal cord. The internuncial neuron lies directly along a path between input and output. It does not belong exclusively either to sensory or to motor functions. *This same ambiguity obtains for every other neuron wholly contained within the central nervous system".* Sherrington had earlier made the same observation with specific reference to Betz cells in the motor cortex. He said they are, in effect, "internuncial neurons" because they lie along a pathway between incoming and outgoing signals. They converge on the "final common path", the motor neuron, along with segmental and intersegmental internuncial neurons. Sherrington and Fulton were both cautious in assigning hierarchical relationships to central neuronal components and pathways: they were sensible of the fact that within the central nervous system neurons may be only relatively motor or sensory in function, even though they may be conveying signals in a clearly centripetal or centrifugal direction. Fulton described an occasion when Sherrington was asked what he meant when he referred to the cerebellum as the "head ganglion" of the proprioceptive system. Sherrington replied, "I meant only that the cerebellum is in the head".

Intersegmental Reflexes

Cajal, in Fig. 9 and Fig. 10, emphasizes the importance of the vertical organization of reflex coordination systems[1]. He depicts with drawings in *his* own hand, direct and indirect afferent-to-efferent projections which presumably can modulate reflex patterns along several segments of the spinal cord. Internuncial neurons as well as afferent fibers can spread both upwards along the neuraxis, thus providing

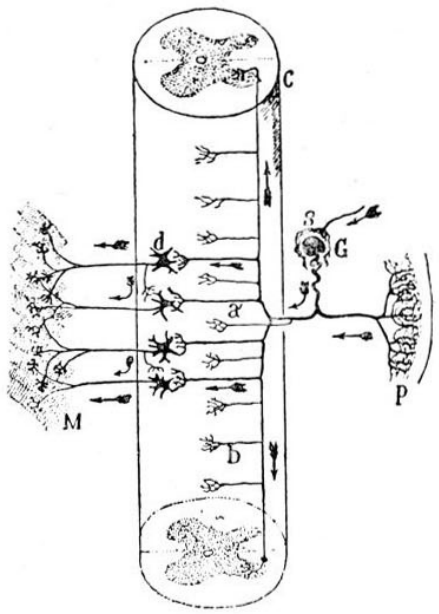


Fig. 9. — *A diagram from Ramón y Cajal*[1] *illustrating how action currents passing along a sensory nerve may be distributed up and down the spinal cord to yield both relatively direct and relatively indirect influences affecting motor groups which serve several segments of the spinal cord.*

longitudinal or intersegmental extensions for sensorimotor integration and reflex control.

Recently, Gernandt, Shimamura, and Akert have shown that stimulation of any dorsal root or mixed nerve yields a double reflex response which can be observed at every ventral root along the entire spinal column bilaterally [7, 8, 12, 13, 14]. These double responses are mediated by separate pathways: one —a *propriospinal system*— is relatively direct. It crosses and recrosses the spinal cord during its upward and downward trajectory and results in bilateral reflex responses at every level of output along the whole spinal cord. Reflex responses to excitation of this pathway are dispersed decrementally in time both up and down the spinal system of motor centers. Another pathway involves a more circuitous route through the medulla oblongata. This is a *spino-bulbo-spinal system.* Motor effects following excitation of this less direct longitudinal reflex pathway are always dispersed along the neuraxis, in time and spatially, beginning with the medulla oblongata, regardless of the site of stimulus input. In other words, this path has no outward (efferent) expression until after it has been relayed through the medulla oblongata. Because the system relayed through the bulb is more rapidly conducting than the more direct propriospinal reflex system, impulses arising from sensory fields in the *hindlimbs* can activate *forelimb* reflex responses simultaneously as a result of impulses travelling along both the propriospinal and the spino-bulbo-spinal pathways. This is true even though the latter pathway has a conspicuously longer route.

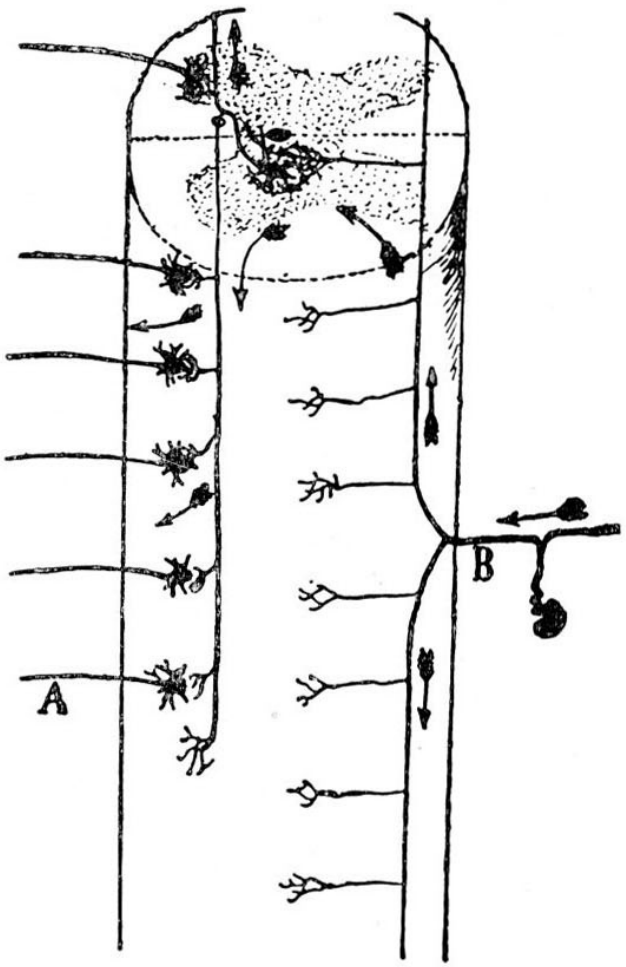


Fig. 10. — *Another diagram from Cajal*[1] *showing, in principle, how incoming impulses may ascend the spinal cord for some distance before communicating to an internuncial neuron which, in turn, may ascend or descend the spinal cord for some distance before affecting motor units serving several different spinal segments.*

Similarly, impulses arising from sensory fields in the *forelimbs* can activate *hindlimb* motor discharges simultaneously as a result of impulses travelling along both propriospinal and spino-bulbo-spinal pathways. It appears as though the spinal organization of conduction velocities were matched appropriately for simultaneous direct and indirect control of interlimb reflexes.

Akert and Shimamura[13] have shown that although spino-bulbo-spinal reflex responses are as widespread as propriospinal reflex responses, they are induced at lower stimulus intensities. Moreover, spino-bulbo-spinal reflexes are predominantly cutaneous in origin and give rise to reflex responses which induce predominately flexor motor effects. Spino-bulbo-spinal reflex responses can also be elicited by stimulation of muscle afferents; however, muscle afferent induction requires higher intensities of stimulation and yields less consistent responses. In the case of stimulation of muscle afferents from extensor muscles, extensor muscle contractions are yielded, in contrast to the general tendency for the spino-bulbo-spinal system to yield flexion reflexes. Both propriospinal and spino-bulbo-spinal reflexes are executed by activation of alpha motoneurons. The same individual alpha units respond to activation of either or both of these separately conducting systems.

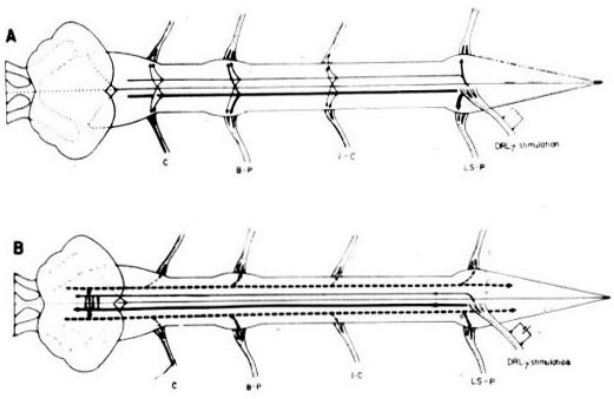


Fig. 11. — *Diagram from Shimamura and Livingston[14] illustrating two physiologically distinguishable longitudinal sensorimotor reflex systems, A: propriospinal, projecting bilaterally diffusely throughout the spinal cord, and B: spino-bulbo-spinal, projecting to the medulla oblongata from whence it returns, after relay in the bulb, to yield a descending cascade of motor responses along each side of the spinal cord throughout its length.*

Cranio-Spinal Reflexes

Shimamura has shown that a similar double system of projections —one direct, the other bulbar relayed— intercoordinates spinal with cranial, cranial with spinal, and cranial with cranial reflexes[12]. Thus, *all dorsal root and cranial nerve inputs and all ventral root and cranial nerve outputs are linked centrally by at least two distinguishable sensorimotor coordination systems.* These systems stitch together intersegmental and interlimb reflexes and provide foundations for important reflexes interconnecting the head with the body, and the limbs with each other.

Interpretations

It is evident that both proprioceptive and cutaneous sensory patterns arising in any limb can yield motor repercussions which affect the entire neuraxial motor column, cranial as well as spinal. Because the cellular clusters in the medulla oblongata involved in the bulbar relay overlap extensively with medullary visceral centers, it is highly probable that these relayed reflexes are also intimately inter-related with mechanisms governing visceral tonic and phasic activities. Perhaps the pattern of visceral activation thus induced will turn out to be that which is necessary to support the somatic reflexes simultaneously induced. This is a point which could easily be tested.

Coordination between limbs and between spinal and cranial sensorimotor mechanisms is so organized that responses relayed through the bulbar centers travel faster than those more directly projected. This would likely serve to advantage in both posture and locomotion and in any situation calling for head, body, and interlimb coordination. Shimamura and Akert have shown these systems to be responsive to stretch receptor mechanisms and neck proprioceptive mechanisms[13]. These longitudinal integrative

mechanisms are presumably available for many kinds of spinal reflexes and other patterns of coordinated postural control, such as righting reflexes, labyrinthine reflexes, flexion and extension reflexes, various rigidities, and the acts of sitting and standing, walking, hopping, and jumping.

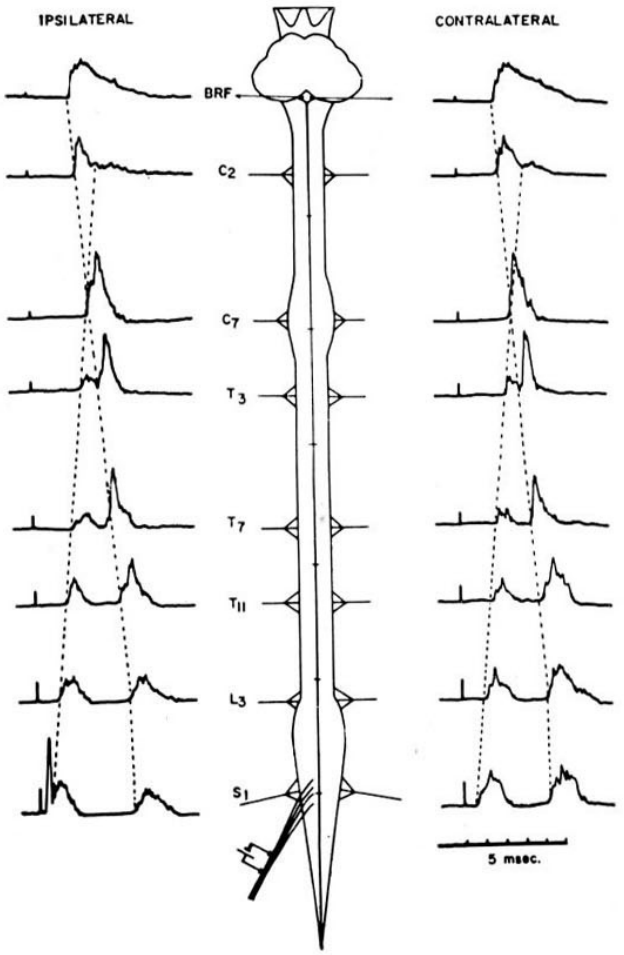


Fig. 12. — *Diagram from Shimamura and Livingston[14] illustrating the sequence of latencies for locally initial and locally delayed responses evoked by stimulation of dorsal root S_1. Five cm. distances along the spinal cord are indicated by short lines crossing the midline of the spinal cord. Oscilloscopic tracings from each of several ventral roots on both sides of the spinal cord are dispersed according to their relative distance from the S_1 segmental level of stimulation. Latencies of responses to propriospinal and spino-bulbo-spinal activation are identified by dashed lines which indicate, by their even slope, uniform but markedly different conduction velocities along the two systems. Note temporal convergence via both propriospinal and spino-bulbo-spinal systems of hindlimb impulses on forelimb motor neurons.*

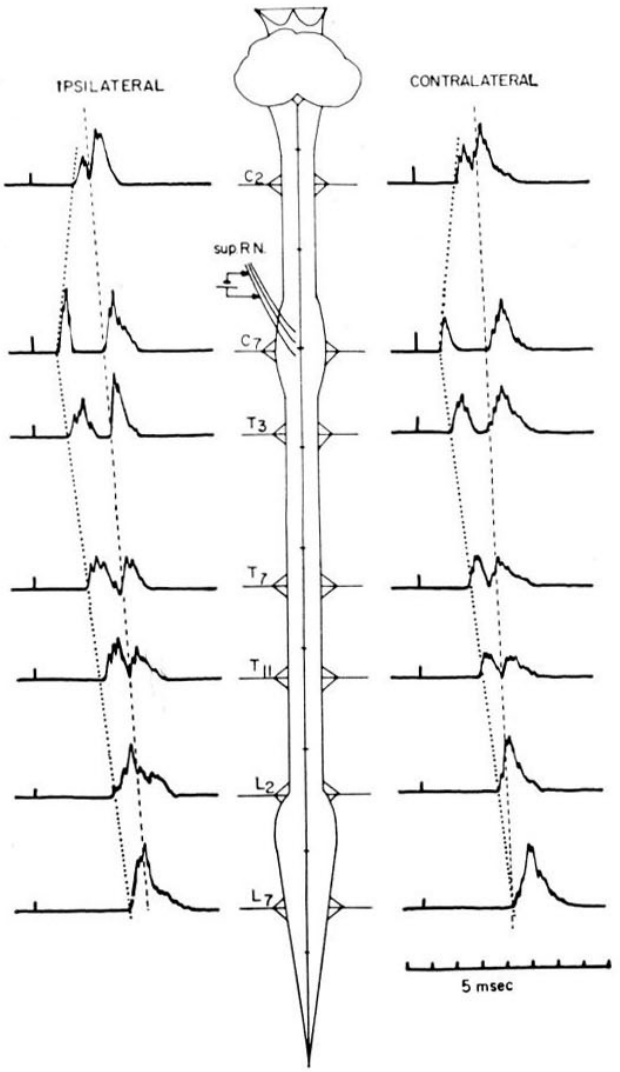


Fig. 13. — *Diagram from Shimamura and Livingston[14] as above, with stimulation applied to the superficial branch of the radial nerve. Here locally early (propriospinal) and locally late (spino-bulbo-spinal) responses converge from forelimb stimulation on hindlimb motor neurons.*

Local Sensory Influences Enhancing Longitudinal Reflexes

The effects of local sensory patterns on localized enhancement of such longitudinal reflex responses has recently been demonstrated[6]. The upper half of Fig. 14 shows a typical lumbosacral ventral root response to vestibular stimulation. This vestibulospinal reflex involves both directly (vestibulospinal) and indirectly (cranio-bulbospinal) relayed systems and represents a specific example of the craniospinal double

system of sensorimotor coordination already depicted. In the lower half of the figure, the joints in the cat's foot have been displaced dorsalward as in the position of normal weight-bearing. The change induced in local sensory influx by dorsal displacement of the foot has the effect of markedly enhancing the local ventral root response to vestibular stimulation. The enhancement can be produced by dorsal displacement of the foot and by working of the phalangeal and metatarsal joints of the foot. No other changes in posture or movement of the limb, muscular or cutaneous stimulation, noxious or otherwise, appears to have any such effect. This vestibulospinal reflex enhancement affects only motoneurons localized to that particular limb. In the absence of muscle relaxants, the combination yields stabilization and extension of that extremity. Long intersegmental integrative patterns are in this way closely coupled with local integrative patterns.

This localized enhancement of vestibulospinal reflexes may provide an underlying neurophysiological explanation for the so-called "magnet reaction", whereby the foot of a decerebrated animal suspended in air

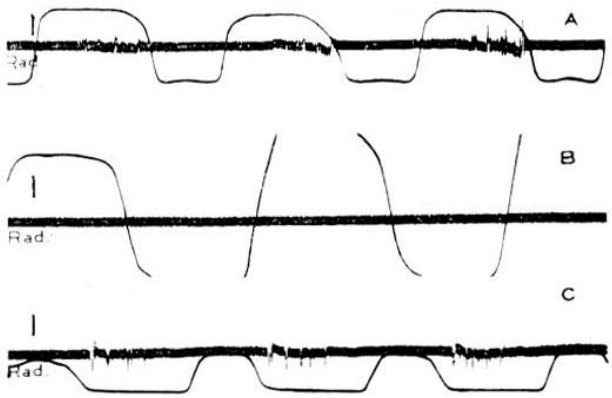


Fig. 15. — *Illustration (retouched) from Livingston, Paillard, Tournay and Fessard*[11] *showing the effect of immobilization of the wrist in blocking voluntary contractions of motor units of the extensors of the wrist. Heavy bar: electromyographic trace, light curve photoelectric tracing of excursion of fingers in a gesture of forceful prehension. A: control tracing showing short bursts of units associated with each gesture, B: during immobilization (with even more exaggerated and forceful movements of prehension) showing no responses of motor units, and C: following discontinuation of immobilization, the same units can be seen as in A, responding to each gesture of prehension, indicating that the needle electrodes have not been disturbed from their location. The manner of joint immobilization is immaterial for this experiment.*

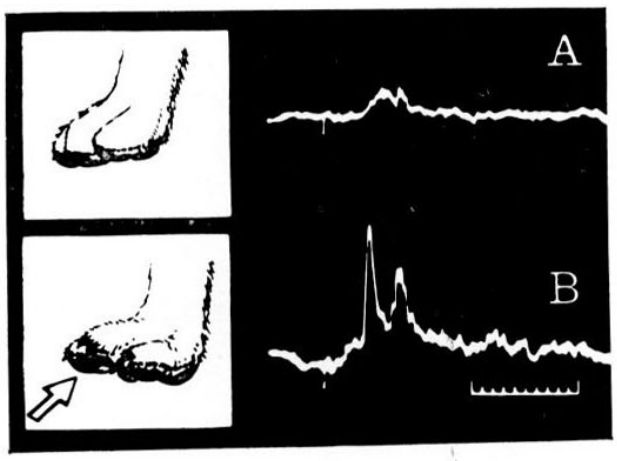


Fig. 14. — *Sketch and accompanying oscilloscopic tracings from Gernandt, Katsuki and Livingston*[6] *showing the effects of upward displacement of the foot which produces localized facilitation of vestibulospinal reflex responses. This provides a neurophysiological basis for the "magnet" response and would contribute to local autonomy and timing of weight bearing acts.*

will offer downward thrust against a supporting object and will appear to "follow" that object wherever it offers upward-weight-bearing support. Such localized reflex enhancement could contribute further goal-oriented potentialities to the mechanisms of posture and locomotion cited above. The two patterns, local and longitudinal, acting together, can "save" higher centers from having to organize the more complex signals that would otherwise be required for accomplishing the same performance in the absence of these "built in" reflex mechanisms. The local enhancement of longitudinal reflexes, moreover, guarantees *exact timing* of the local motor reinforcement, thus further sparing higher centers from more complicated central controls for the timing of responses and, at the same time, saving local muscular resources from unnecessary expenditures of energy.

Local Sensory Influences Blocking Voluntary Performance

By quite different means, it has been demonstrated in man that local sensory patterns can contribute not only to patterns of motor *activation,* as we have just seen, but that they may be responsible for a feedback signal which can impose *limitations* on otherwise potentially responsive motor centers. For example, Fessard and his collaborators[11] found that when the wrist joint is effectively immobilized, the extensor of the wrist cannot be excited into action by any amount of voluntary effort. With bipolar electrodes in place, it was possible to show that the same units respond before and after, but not during periods of complete wrist immobilization. If the wrist is not completely immobilized, voluntary contractions of the muscle may be resumed. This local sensory pattern, limiting responsiveness to voluntary command, has, for obvious reasons, not yet been shown to link up with the two longitudinal reflex patterns described above. But, it does clearly indicate how local sensory patterns may block access of "suprasegmental" motor signals to the motor units acting over an immobilized joint.

These blocking mechanisms would appear to play a protective role by reducing muscular spasm and associated pain as a consequence of immobilization of a broken limb or inflamed joint. Once again, local sensorimotor integrative mechanisms appear to be serving a biologically advantageous purpose by precluding the wastage of local energy expenditure against an impossible task. We have long known that local sensory patterns set the neuronal background for activating or facilitating reflexly and voluntarily induced motor performances, but here is a neuronal background which can *preclude* action.

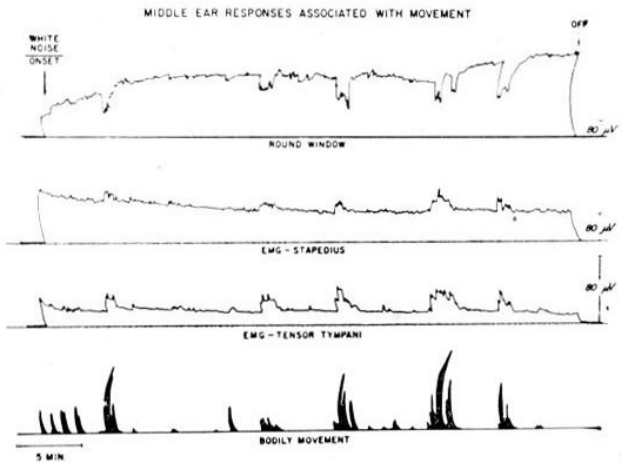


Fig. 16. — *Summated responses during prolonged white noise (85 db). Round-window, first trace; stapedius, second trace; tensor tympani, third trace; bodily movement, fourth trace.*

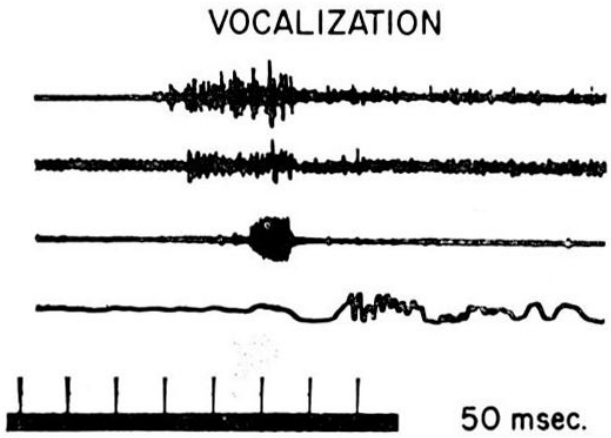


Fig. 17. — *Middle ear responses with vocalization. Stapedius EMG, first trace; tensor tympani EMG, second trace; round-window response, third trace; piezoelectric response indicating movement, fourth trace.*

Interpretations

The segmental reflex, such as the "simple knee jerk", was shown by John Fulton to be appreciably affected in terms of its latency, according to the length and levels of organization of the neuraxis available for indirect contributions to the "background" of activity against which the most direct (monosynaptic) reflex pathway is activitated[3]. He found that the latency to initial response is generally *shorter* when more of the neuraxis is available. *Local* sensorimotor coordination is in this way tonically affected by remote centers. In a like fashion, the results of activation of longitudinal systems can be significantly affected by local changes in sensory input.

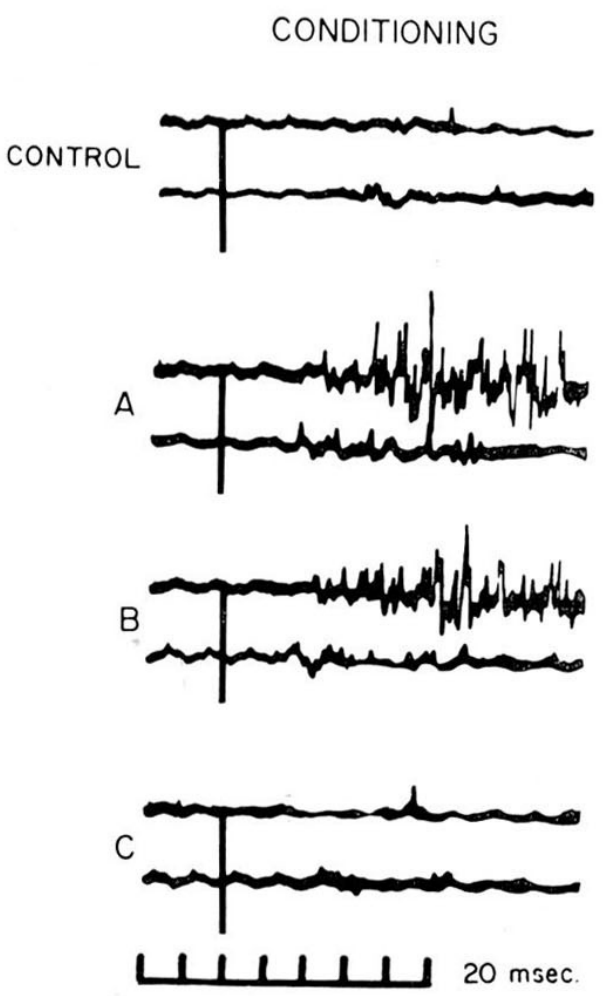


Fig. 18. — *Modifications of middle-ear muscle responses to low-intensity white noise (50 db). Vertical lines in control, A, and B indicate onset of low-intensity sound. Vertical line in C indicates onset of light flash in the absence of any sound stimuli. Stapedius EMG, upper trace; tensor tympani EMG, lower trace. Control responses show absent EMG to low-intensity sound. A, responses several seconds after one short burst of loud white noise (300 msec., 95 db); B, responses after a single pairing of the low-intensity noise with an electric shock to the animal's body; C, responses after several pairings of light flash with the same electric shock.*

Altogether, there is mounting evidence for patterns of sensorimotor coordination which in and of themselves appear to subserve portions of more complicated and entire purposes of sensorimotor coordination. Each level of organization of these elementary reflex patterns is coordinately linked to associated levels elsewhere along the neuraxis. These findings are not only embraced within the shadows of Sherrington and Fulton but they yield an impression that there still remains an almost infinite variety of comparable experiments which may throw additional light on our as yet inadequate understanding of the integration mechanisms underlying posture, locomotion, and simple coordinated acts.

The main generalization which seems to obtrude itself from what is now known about these systems is that *each of the neurophysiological patterns seems to be organized so as to provide integration favoring maximum local responsibility and control over fundamental acts.* This is accomplished in a way that increases the self-sufficiency of the local system and spares more remote centers for activities which John Fulton referred to as "long-circuiting". In the case of "higher (more cephalic) centers", this long-circuiting takes the form of more elaborate patterns of integration of more inclusive scope. This long-circuiting does not imply that the "higher centers" necessarily thereby assume either increasing responsibility or control. They may or may not be able to exercise such responsibility or control, depending on the degree of modifiability of the local integrative mechanisms. We shall see in the following section that something of the same sort appears to obtain in relation to the far more "dynamic and plastic" systems involved in auditory mechanisms.

II. INTEGRATION ALONG THE AUDITORY PATHWAY

Middle Ear Muscle Reflexes

Carmel and Starr have shown that middle ear muscles respond to low intensity as well as to high intensity sounds[2]. The middle ear acoustic reflex is proving to be not simply an auditory protective device, but a motor gesture which provides a continuous exquisite modulation of the acoustic environment. Interference with normal middle ear muscle responses, as obtained in patients with myasthenia gravis who are in need of pros-

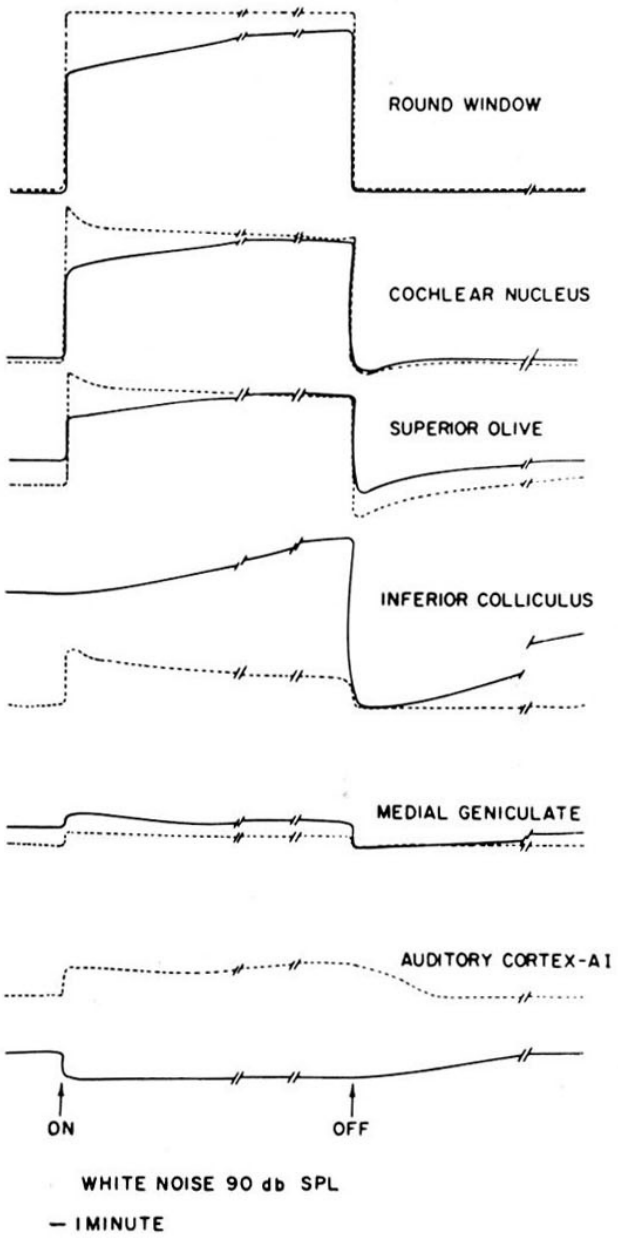


Fig. 19. — *Diagram from Starr and Livingston[15] illustrating schematically the form of averaged electrical responses, at each of several stations along the auditory pathway, in response to two hours exposure of steady loud noise. At the round window, cochlear nucleus, superior olive and inferior colliculus can be seen initial rising curves of somewhat different shape, but all relating to a gradual relaxation of middle ear muscles during the first 30-45 minutes of sound exposure. Higher stations do not show this middle ear muscle effect. Each station along the pathway has a characteristic pattern of response to the sound and several of the stations show equally characteristic after-effects following discontinuation of the sound.*

tigmine, or in patients who have recently sustained Bell's palsy, gives rise to hyperacusis. Prostigmine, in the former case will apparently abruptly relieve the symptom of hyperacusis. Middle ear muscle contractions are also induced by nonacoustic factors, such as bodily movements, vocalization, and stimulation of the external auditory canal. These effects can be demonstrated in totally deafened animals. Middle ear muscle contractions associated with vocalization precede onset of cochlear microphonics in response to the vocalization; therefore, the middle ear muscle contractions must be anticipatory and preparatory. In the newly born kitten, middle ear muscle responses associated with bodily movements appear earlier than does cochlear microphonic attentuation. Thus, already at the neuromuscular "segmental reflex" level of organization of auditory mechanisms, there is evidence for considerable complexity. As a result of their contractions, the middle ear muscles can exert marked effects on both round window microphonics and on central nervous system responses evoked by given stimuli.

Conditioning Auditory Responses

At higher levels of the auditory system even more dramatic complexities are observed. Hernández-Peón and his colleagues[9] and Galambos and his co-workers[4] were first to discover that, in conscious cats, click evoked responses recorded from the level of the *first central relay* along the auditory pathway, and from many other regions throughout the central nervous system, can be markedly affected according to an animal's direction of attention and its previous sensory experiences. Thus, while a cat is watching mice in a jar, or sniffing fishy odors from a tube, click evoked responses in the cochlear nucleus appear sharply attentuated[9]. Similarly, classical Pavlovian conditioning experiments yield attenuation or augmentation of such click evoked responses recorded from a variety of cerebral loci[4]. The conditioning can be extinguished and reconditioned repeatedly.

Recently, Starr has shown that acoustic responses to long lasting sound stimuli evoke patterns in lower auditory stations which are partially a reflexion of the middle ear muscle cochlear microphonic attenuation described above and partly are characteristic of the particular nucleus from which the recording is derived[15]. Every station along the auditory pathway appears to have an individualistic pattern of electrical activity in response to a stereotyped sound stimulus. Whereas transient sound signals can be recorded in widespread regions throughout the central nervous system, the long lasting sound signals seem to be limited to the classical auditory pathway. This implies that the *"extra-classical sensory pathways"*, borne by the reticular formation and other structures, *may be responding preponderantly to transiency of signals* —to change— without necessarily bearing signalling reference to the specific modality involved. In addition to the auditory controls exerted by middle ear muscle contraction, there are also purely nervous controls affecting the cochlea directly, by way of the Rasmussen olivo-cochlear bundle and by way of controls affecting the ascending sequence of central auditory synapses.

There are also marked *after-effects following cessation of long lasting sound stimuli.* These after-effects are characteristic for the individual nuclei within the auditory system. Galin has shown, moreover, that positive and negative reinforcement (food and mild electric shock) have opposite effects on the averaged electrical response to prolonged sound signals[5]. *Association of food with a given acoustic signal yields enhancement of the auditory response to that signal as recorded from the level of the medial geniculate body. Association of mild electric shock yields, in contrast, a reduction in amplitude of the acoustic response as recorded from the level of the inferior colliculus.* These effects can be repeatedly conditioned, extinguished, and re-conditioned. The contrasting modes of reinforcement have no obvious effects on stations other than those mentioned, that is, food reinforcement does not appear to affect responses recorded from the inferior colliculus, nor does mild electric shock appear to affect responses recorded from the medial geniculate body. Neither of these reinforcements affects the averaged electrical responses recorded from auditory stations below the inferior colliculus.

These findings of Galin, although recently broached and in need of his own further corroboration and extension, appear to me to open up an entirely new domain for neurophysiology and psychology. By means with which he is now pioneering, it may prove feasible systematically to localize and

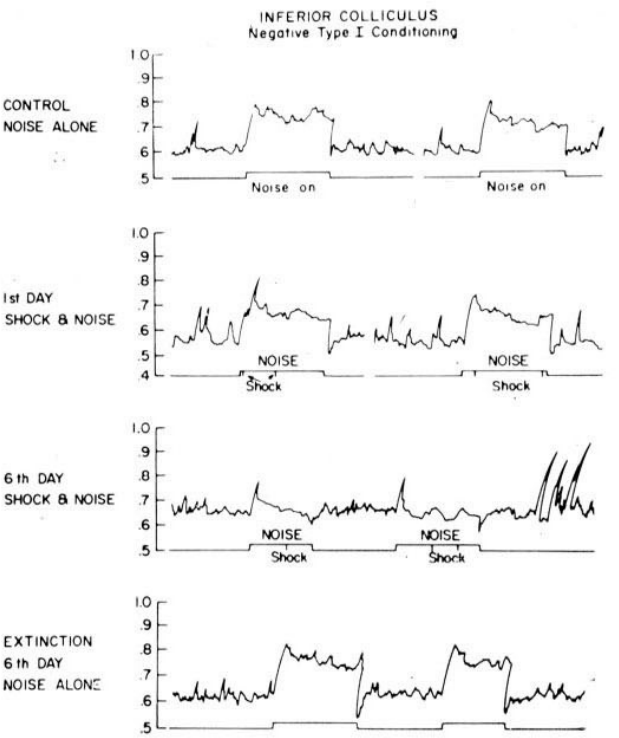


Fig. 20. — *Samples of averaged amplitude of electrical activity in the inferior colliculus in response to standard sound stimulus during negative (mild electrical shock) conditioning and (no shock) extinction trials. Read tracings left to right. Approximately two minutes continuous 65 db white noise indicated by rise in stimulus marking trace. Shock reinforcement indicated by vertical strokes of marking pen randomly spaced during noise presentations. Vertical scale has arbitrary units but approximately linear scale. Note increasing attentuation of inferior colliculus responses to sound during conditioning (shock) trials and recovery to initial values during extinction (no shock) trials.*

characterize the central nervous system correlates of pleasant and unpleasant perceptual experiences. The fact that positive and negative reinforcements have differential effects is most exciting. It calls to mind the Aristotelian view that "appetite" attaches to sensory experiences, giving the individual an impulse to seek that which is pleasant and to avoid that which is painful. In any event, these mechanisms appear to "contaminate" incoming signals with "value" already at the level of the midbrain and diencephalon.

Interpretations

Sensory signals obviously don't come cleanly into the "inmost portion of the brain". These signals are conspicuously affected according to previous experience, expectations, and the momentary flux of purposes. The effects of central control of sensory transmission may be achieved by means of systems of descending and efferent fiber projections closely associated with the inbound sensory pathways. Signals can be affected from their outset, from the point of stimulus contact with the body. Efferent fibers projecting to sensory endings can affect the receptivity of the end-organs. Thereafter, the signals may be affected at each point of synaptic relay along the entire centripetal system, not only along the trajectory projecting toward cortical receiving areas, but also wherever else such signals may be routed. Thus, the pattern, extent, and duration of central sensory signals evoked by a given sensory stimulus can be vastly and systematically altered according to dynamics intrinsic to the waking brain.

As in spinal and brainstem reflex control systems, the sensory pathways appear to be arranged for maximum local integration and self-sufficiency, together with closely interdependent integrative coupling with other levels of organization of the total system. Surprisingly, a great deal of the modulation of sensory signals appears to take place at lower levels than we previously had any reason to suppose. For example, in the auditory pathway by virtue of middle ear muscular contractions, considerable attenuation of a given acoustic signal may take place prior to activation of the cochlear hair cells. One can visualize important perceptual and behavioral implications relating also to acoustic after-effects, to modulations of amplitude and distribution of signals according to rewarding and punishing associations, and relating to the transient effects on sensory signalling elicited by shifting of the focus of attention.

Lower centers are proving to be more self-sufficient, responsible and controlling than previously supposed. Higher centers are apparently being "saved" for long-circuiting activities which, as in the case of spinal and brainstem reflex systems, are organized so as to enable enmeshing of simpler patterns into more elaborate and more holistic patterns of integration. This is apparently accomplished without loss of integrity, and, in some cases, controlling influence of lower centers on what signals may get up to or descend from higher centers. The higher centers are, in effect, enclosed within certain species invarient and individually varient filters. Higher centers are "victims" of distortion of incoming signals and limited in their outward expression by such filters. Central processes of perception and command are enclosed in a limited and partly plastic physiological capsule.

"Who" reads sensory signals and by what means they are read remain as much as ever a mystery; but it is now possible to surmise that sensory patterns may be "read out" at lower levels than we have previously assumed, just as the patterns for reflex control at spinal and brainstem levels appear to be locally self-sufficient, responsible and controlling for certain actions. The patterns of incoming signals organized along the sensory pathways apparently subserve designs that are partly inscribed according to past experience of the species, and partly shaped according to individual past experience. Each perceptual experience can in this way be compounded of the hereditary endowment which has yielded up the general plan and potentialities of cerebral circuits and the modifications in circuiting imposed by

idiosyncratic individual experience. This is consistent and reinforcing to psychological data.

We can now plainly see that individual experiences affect the character and extent of the invasion of the central nervous system by any given sensory signal. It is the feature of conditionability which strikingly distinguishes the integrative processes of the auditory pathway from those of the propriospinal and spino-bulbo-spinal pathways. Each of these two systems represents some probabilistically determined biological adaptation. The lower level expressions of such adaptation are organized so as to provide, for instance, a close coordination between generalized vestibular and localized afferent patterns for the support of the body's weight against gravity and to accomplish this with the maximum nicety of timing. This exemplifies an adaptation by hereditary endowment, built into the chassis in an "automatic" and serviceable form. This is a *species memory* inscribed into neuronal circuits by the constancy of terrestrial gravity. The auditory patterns of adaptation are also hereditarily organized, — all of the potentialities are species endowed. But the circuits retain extensive degrees of freedom for modifiability and consequently of the distribution of sensory signals according to whatever reward and punishment conditions may happen to be associated with a given acoustic environment. The mechanisms are rigged in such a way that a changeable acoustic environment can be accomodated. If the impacts of gravity, mass and inertia were as changeable as sound patterns, evolution might have yielded mechanisms for individually modifiable adaptations in systems of posture and locomotion.

For many decades neurophysiologists have been accustomed to the notion that sensorimotor coordination is essentially goal-seeking. Increasingly powerful evidence is impelling us towards the less familiar view that mechanisms underlying perception are likewise goal-seeking. Psychologists have long been convinced that we perceive the world around us according to a systematic bias of perceptual commitments founded upon our previous successes and failures in our cumulative collection of perception-dependent actions. In addition, the distinctions between "motor" and "sensory" mechanisms and between "higher" and "lower" processes of integration are losing some of their pristine clarity. Biologically, there is no proper dividing line between them or between "mind" and "body". These all enjoy a "harmonious coalescence", to use the felicitous expression with which John Fulton concluded his monograph *Muscular contraction and the reflex control of movement*[3].

SUMMARY

Two lines of experimental evidence relating to two different levels of organizational complexity of the nervous system are presented. The first level concerns sensorimotor coordination along the vertical axis of the nervous system. The experiments were conducted in the Laboratory of Neurobiology of the National Institutes of Health in Bethesda by Dr. Bo Gernandt of Stockholm, Dr. Muneo Shimamura of Sapporo, and Dr. Konrad Akert of Zürich. This vertical organization for sensorimotor integration involves two distinctive components both of which underlie interlimb, cranial, and spinal reflexes. The first component —propriospinal— is relatively direct and is characterized by impulses which cross and recross the spinal cord, being reflected in bilateral motor responses appearing in every ventral root all along the entire neuraxis. The other component —spino-bulbo-spinal— involves projections which ascend the spinal cord to the medulla oblongata, then reenter the spinal cord from above downward. The bulbar relayed system does not yield motor responses until after impulses have relayed in the medulla oblongata, and does not cross from one side to the other except at the level of sensory input and within the bulb. Therefore, res-

ponses to impulses conveyed along the propriospinal pathway are dispersed in space and time beginning from the point of sensory input, whereas responses to impulses conveyed along the spino-bulbo-spinal pathway are dispersed in space and time from the medulla oblongata *regardless of the locus of sensory input.* Because the bulbar relayed system is more rapidly conducting than the propriospinal system, impulses arising from sensory fields of the hindlimb reach the forelimb and impulses from the forelimb reach the hindlimb along both pathways simultaneously. A similar double system of projections inter-coordinates spinal with cranial, cranial with spinal, and cranial with cranial reflexes. Thus, the entire neuraxis is provided with these two distinctive longitudinal sensorimotor coordination systems which stitch together intersegmental and interlimb reflexes as well as the important reflexes interconnecting the head with the body. Examples are given of integrative actions characterizing longitudinal reflex coordination, emphasizing the relations between descending and local contributions to final motor performances.

A second line of experimental evidence concerns mechanisms involved in the auditory pathway which has also been examined as a vertical sensory integration system. This work has been carried out in the same laboratory by Dr. Arnold Starr, Dr. Peter Carmel, and Dr. David Galin, all of New York. First, they have shown that every station along the auditory pathway has an individual, characteristic pattern of electrical activity in response to a given sound stimulus. Second, whereas *transient* sound signals can be recorded in widespread regions throughout the central nervous system, continuing sound signals evoke continuing alterations in level of activity which are limited to the classical auditory pathway. This implies that the extraclassical sensory pathways borne by the reticular formation and other structures may be responding preponderantly to transiency of signals —to change— without necessarily bearing reference to the specific signalling system concerned. Third, certain lower stations along the auditory pathway are markedly affected by neuromuscular and central neuronal sensory control mechanisms and exhibit dynamic changes in response to a continuing steady sound stimulus. Fourth, the inferior colliculus and the medial geniculate body appear to exhibit a conditionable change in averaged electrical response to sound. Specifically, these two intermediate relay and integrative stations exhibit individually distinctive changes in averaged electrical response to sound when the sound is presented alone after the sound has been associated with the food or mild shock to the animal. The inferior colliculus shows a reduction of averaged electrical response to sound presented alone following punishing conditioning trials, whereas the medial geniculate appears to show an increase in averaged electrical response to sound presented alone following rewarding conditioning trials.

RESUMEN

En este trabajo son presentadas dos categorías de hechos experimentales en relación con dos niveles distintos de complejidad en la organización del sistema nervioso. El primero de esos niveles se refiere a la coordinación sensomotriz a lo largo del eje vertical del sistema nervioso. Las experiencias fueron llevadas a cabo en el laboratorio de Neurobiología de los Institutos Nacionales de Salud en Bethesda por los Dres. Bo Gernandt de Estocolmo, Muneo Shimamura de Sapporo y Konrad Akert de Zurich. Esta organización vertical de la integración sensomotriz implica dos componentes diferenciados que están en la base de los reflejos intermiembros, craneanos y medulares. El primer componente —propriomedular— actúa en forma relativamente directa y se caracteriza por impulsos que cruzan y vuelven a cruzar la médula, y que se traducen en respuestas motoras bilaterales que aparecen en cada una de las raíces ventrales que se encuentran a lo largo de todo el neuroeje. El se-

gundo —médulo-bulbo-medular— involucra proyecciones que ascienden por la médula hacia el bulbo raquídeo y que vuelven a penetrar luego en la médula en sentido descendente. El sistema bulbar no produce respuestas motrices hasta luego de haber liberado los impulsos en el bulbo raquídeo, y no cruza de un lado al otro excepto a nivel de la entrada sensorial y dentro del bulbo. De ahí que las respuestas a impulsos transportados por la vía propriomedular se dispersen en tiempo y espacio a partir del bulbo cualquiera que sea el punto de partida sensorial. Siendo el sistema bulbar un conductor más rápido que el propriomedular, los impulsos originados en los campos sensitivos de los miembros posteriores alcanzan los anteriores e impulsos de los miembros anteriores alcanzan los posteriores a lo largo de ambas vías simultáneamente. Un doble sistema de proyecciones similar intercoordina a su vez los reflejos medulares con los craneanos, los craneanos con los medulares y los craneanos con los craneanos. Así el neuroeje contiene estos dos sistemas diferenciados de coordinación sensomotriz longitudinal que enlaza reflejos intersegmentales e intermiembros así como también los reflejos importantes de interconexión entre la cabeza y el cuerpo. Se citan ejemplos de acciones integrativas que caracterizan el reflejo coordinador longitudinal, poniendo énfasis en las relaciones entre las contribuciones descendentes y las locales a la realización motriz final.

Un segundo aspecto de evidencia experimental se refiere a los mecanismos involucrados en la vía auditiva, que también ha sido examinada como sistema vertical de integración sensitiva. Este trabajo ha sido realizado en los mismos laboratorios por los Dres. Arnold Starr, Peter Carmel y David Galin de Nueva York. En primer lugar demostraron que cada etapa a lo largo de la vía auditiva tiene un trazado individual y característico de actividad eléctrica como respuesta a un estímulo sonoro determinado. En segundo lugar, mientras las señales sonoras *transitorias* pueden ser registradas en varias regiones diseminadas a lo largo del sistema nervioso central en su totalidad, aquellas señales sonoras contínuas evocan alteraciones contínuas a un nivel de actividad limitado a la vía auditiva clásica. Esto implica que los sistemas fuera de la vía auditiva clásica estarían guiados por la formación reticular y otras estructuras que podrían responder particularmente al espaciamiento de las señales, al cambio, sin que por eso esté implicado en el proceso el sistema específico de señales. En tercer lugar, ciertas etapas bajas a lo largo de la vía auditiva se ven considerablemente afectadas por mecanismos de control neuromuscular y de neurona sensitiva central y presentan cambios dinámicos como respuesta a un estímulo sonoro prolongado y contínuo. Cuarto, el colliculus inferior y el cuerpo geniculado interno hacen aparecer un cambio condicionable en la respuesta eléctrica promedio al sonido. Específicamente, estas dos etapas integrativas y estaciones intermedias presentan individualmente cambios diferenciados en la repuesta eléctrica promedio al sonido cuando éste se presenta solo, luego de haber sido asociado con la comida o un ligero golpe al animal. El colliculus inferior evidencia una disminución de la respuesta eléctrica promedio al sonido a continuación de intentos condicionados de castigo, mientras que el medial geniculado suscita un aumento en el promedio de respuesta eléctrica al sonido que se presenta aisladamente a continuación de intentos condicionados de premio.

RESUME

Deux catégories de faits expérimentaux ayant trait à deux niveaux différents de complexité organisationnelle du système nerveux sont exposées. Le premier niveau concerne la coordination motrice sensorielle le long de l'axe vertical du système nerveux. Les expériences ont été réalisées au laboratoire de neuro-biologie de l'Institut National de Santé de Bethesda par le Dr. Bo Gernandt de Stockholm, le Dr. Muneo Shimamura de Sapporo et le Dr. Konrad Akert de Zürich. Cette organisation verti-

cale de l'intégration motrice sensorielle met en jeu deux composantes distinctes qui toutes deux sont à la base des réflexes intermembres, craniaux et vertébraux. La première composante —propriospinale— est relativement directe et est caracterisée par des impulsions qui traversent et retraversent la moelle épinière, réfléchies sous forme de réponses motrices bilatérales qui apparaissent dans chaque racine ventrale le long du névraxe tout entier. L'autre composante —vertébro-bulbo-vertébrale— comprend des projections qui montent le long de la moelle épinière jusqu'au bulbe rachidien et, de là, pénètrent à nouveau dans la moelle épinière de haut en bas. Le système à relai par le bulbe ne produit pas de réponses motrices avant que les impulsions n'aient été relayées par le bulbe rachidien et ne se déplace pas d'un côté à l'autre sauf au niveau de l'entrée sensorielle et au sein du bulbe. Par conséquent, les réponses à des impulsions acheminées le long de la voie propriospinale sont dispersées dans l'espace et dans le temps dès l'entrée sensorielle, tandis que les réponses à des impulsions acheminées le long de la voie vertébro-bulbo-vertébrale sont dispersées dans l'espace et dans le temps à partir du bulbe rachidien *quel que soit le point d'entrée sensorielle.* Le système à relai bulbaire conduisant plus rapidement que le système propriospinal, des impulsions provenant de champs sensoriels des membres inférieurs parviennent aux membres supérieurs et des impulsions partant des membres supérieurs parviennent aux membres inférieurs par les deux voies simultanément. Un double système de projections semblable inter-coordonne les réflexes vertébraux et craniaux, craniaux et vertébraux ainsi que craniaux et craniaux. Ainsi, le névraxe tout entier possède ces deux systèmes distincts de coordination motrice sensorielle longitudinale qui relient les réflexes intersegmentaires et intermembres ainsi que les réflexes importants reliant la tête et le corps. L'exposé contient des exemples d'actions intégrantes caractérisant la coordination de réflexes longitudinale, mettant en évidence les relations entre les contributions descendantes et locales aux actes moteurs finals.

Une seconde catégorie de faits expérimentaux concerne les mécanismes intervenant dans la voie auditive qui a été également étudiée comme système d'intégration sensorielle vertical. Cette étude éffectuée au même laboratoire par le Dr. Arnold Starr, le Dr. Peter Carmel et le Dr. David Galin, tous trois de New York. Tout d'abord, ils ont montré que chaque étape le long de la voie auditive a sont état propre, caractéristique, d'activité électrique en réponse à un signal sonore donné. Puis, tandis que des signaux sonores *transitoires* peuvent être enregistrés dans des régions dispersées du système nerveux central, des signaux sonores continus provoquent des alterations continues du niveau d'activité qui sont limitées à la voie auditive classique. Ceci implique que des voies sensorielles extraclassiques portées par la formation réticulaire et d'autres structures peuvent répondre principalement à des signaux transitoires —à des changements— sans que pour cela il y ait nécessairement référence au système signalant spécifique impliqué. Troisièmement, certaines étapes plus basses le long de la voie auditive sont nettement affectées par des mécanismes de contrôle neuromusculaires et centroneuroneaux sensoriels et montrent des changements dynamiques en réponse à une impression sonore continue stable. Enfin, la crête inférieure des tubercules quadrijumeaux et le corps genouillé médial semblent montrer des changements conditionnés en réponses moyennes au son. Plus précisement, ces deux relais intermédiaires et étapes intégrantes montrent individuellement des changements caractéristiques de réponse moyenne au son lorsque le son se présente seul après avoir été associé avec de la nourriture ou un faible choc à l'animal. La crête inférieure des tubercules quadrijumeaux montre une réduction de la réponse électrique moyenne à un son se présentant seul après des expériences de conditionnement par punition, tandis que le corps genouillé médial paraît montrer une augmentation de la réponse électrique moyenne à un son se présentant seul après des expériences de conditionnement par récompense.

ZUSAMMENFASSUNG

Zwei Arten experimenteller Beweisführung werden behandelt, die sich auf zwei verschiedene Grade der organisatorischen Komplizierthiet des Nervensystems beziehen. Der erste Grad betrifft die Beziehung der sensiblen und motorischen Anteile entlang der vertikalen Achse des Nervensystems. Die Versuche wurden im Laboratorium für Neurobiologie des National Institute of Health in Bethesda von Dr. Bo Gernandt aus Stockholm, Dr. Muneo Shimamura aus Sapporo und Dr. Konrad Akert aus Zürich durchgeführt. Diese vertikale Anordnung der sensibel-motorischen Einheiten beruht auf zwei klar vor einander unterschiedenen Systemen, die beide den Reflexen zwischen den Extremitäten, den Gehirnreflexen und den Rückenmarksreflexen zugrundeliegen. Das erste System —das propriospinale— ist verhältnismässig direkt und ist charakterisiert durch Reize, welche im Rückenmark hin- und hergehen und welche in beiderseitigen motorischen Reaktionen resultieren, die in jeder Vorderwurzel entlang der gesamten Nervenachse auftreten. Das andere spino-bulbo-spinale System wird von Reizen benützt, die im Rückenmark bis zur medulla oblongata aufsteigen, dort wieder in das Rückenmark eintreten und sich dann abwärts bewegen. Das im Bulbus übertragene System führt erst dann zu einem motorischen Effekt, wenn Impulse in der medulla oblongata übertragen worden sind und kreuzt von einer Seite zur andern ausschliesslich in der Ebene des sensiblen Reizes und innerhalb des Bulbus. Folglich werden die Reaktionen auf Impulse, die die propriospinale Bahn benutzen, räumlich und zeitlich vom Punkt der Reizeinwirkung aus zerstreut, während Reaktionen auf Impulse, die die spino-bulbo-spinale Bahn benutzen, sich räumlich und zeitlich von der medulla oblongata verbreiten, also *unabhängig vom Ort der sensiblen Reizeinwirkung.* Da das im Bulbus übertragene System schneller leitet als das propriospinale System, erreichen Impulse, die vom sensiblen Gebiet der Hinterextremität ausgehen, die Vorderextremität, und die von der Vorderextremität ausgehen, erreichen die Hinterextremität gleichzeitig über beide Leitungsbahnen. Ein ähnliches Doppelsystem koordiniert wechselseitig die Rückenmarksreflexe mit den Hirnreflexen und Hirnreflexe mit anderen Hirnreflexen. So ist die gesamte Nervenachse mit diesen beiden deutlich von einander unterschiedenen, der Länge nach verlaufenden sensibel-motorischen Koordinierungssystemen versehen, welche sowohl die intersegmentären Reflexe und die zwischen den Extremitäten, als auch die wichtigen Reflexe, die wechselseitig den Kopf und den Körper verbinden, zusammenhalten. Beispiele komplizierterer Aktionen, welche für die Reflexkoordination nach oben und unten charakteristisch sind, werden angeführt, wobei die Beziehung zwischen absteigenden und lokalen Beiträgen und dem endgültigen motorischen Effekt besonders betont werden.

Eine zweite Serie experimenteller Beweisführung befasst sich mit Mechanismen in der Leitungsbahn des Gehörsinnes, die ebenfalls als ein vertikales System der Zusammenfassung sensibler Reize studiert wurde. Diese Arbeit wurde im gleichen Laboratorium von Dr. Arnold Starr, Dr. Peter Carmel und Dr. David Galin, alle aus New York, durchgeführt. Sie zeigten erstens, dass iede Stelle entlang der Gehörleitungsbahn in einer ihr eigenen, charakteristischen Art und Weise auf ein gegebenes Geräusch elektrisch reagiert. Zweitens, während *vorübergehende* Geräuschsignale in weiten Gebieten des gesamten zentralen Nervensystems registriert werden können, führen dauernde Geräuschsignale zu dauernden Änderungen in der Intensität des Effekts, die auf die klassische Hörleitungsbahn beschränkt sind. Das deutet an, dass die extra-klassischen sensiblen Bahnen, die auf der formatio reticularis und anderen Strukturen verlaufen, möglicherweise hauptsächlich auf vorübergehende Signale —auf Änderung— ansprechen und nicht unbedingt auf das spezifische Signalsystem abgestimmt sind. Drittens, bestimmte tiefer an der Hörleitungsbahn gelegene Stellen werden deutlich von neuro-muskulären und zentralneuronalen sensiblen Kontrollmechanismen beeinflusst und zeigen dynamische Änderungen, wenn

sie einem fortgesetzten Geräuschreiz ausgesetzt sind. Viertens, der colliculus inferior und das corpus geniculatum mediale scheinen eine beeinflussbare Veränderung des durchschnittlichen elektrischen Geräuscheffekts zu zeigen. Im besondern zeigen diese zwei Zwischenstationen für Übertragung und Zusammenfassung individuell ausgeprägte Änderungen im Durchschnitt der elektrischen Reaktion auf Geräusch, wenn das Geräusch allein einwirkt, nachdem das Geräusch auf das Tier zusammen mit Futter oder mit einem leichten Schock eingewirkt hatte. Der colliculus inferior zeigt eine Verminderung der elektrischen Durchschnittsreaktion auf Geräusch alleine, wenn schmerzhafte Bedingungsversuche vorhergegangen waren, während das geniculatum mediale eine Zunahme in der elektrischen Durchschnittsreaktion auf Geräusch, wenn alleine angeboten, zu zeigen scheint, wenn Versuche mit Geräusch unter angenehmen Bedingungen vorausgegangen waren.

REFERENCES

(1) *Cajal, S. Ramón*: Histologie du système nerveux de l'homme & des vertébrés. (French Ed. Trans. by Azoulay, L.) Paris, A. Maloine, 2 tom: xiv + 986 pp. (1909); 993 pp. (1911).

(2) *Carmel, P. W.* and *Starr, A.*: Acoustic and non-acoustic factors modifying middle ear muscle activity in waking cats. J. Neurophysiol., 1963, *26*: 598-616.

(3) *Fulton, J. F.*: Muscular contraction and the reflex control of movement., Baltimore, Williams and Wilkins Company, 1926, xv + 644 pp.

(4) *Galambos, R.; Sheats, G.* and *Vernier, V. G.*: Electrophysiological correlates of a conditioned response in cats. Science, 1956, *123*: 376-378.

(5) *Galin, D.*: Effects of conditioning on auditory signals. Chapter in: Fields, W. S. (ed.) Neurological aspects of auditory and vestibular disorders. Springfield, Ill., Charles C. Thomas, 1963, (in press).

(6) *Gernandt, Bo E.; Katsuki, Y.* and *Livingston, R. B.*: Functional organization of descending vestibular influences. J. Neurophysiol., 1957, *20*: 453-469.

(7) *Gernandt, Bo E.* and *Megirian, D.*: Ascending propriospinal mechanisms. J. Neurophysiol., 1961, *24*: 364-376.

(8) *Gernandt, Bo E.* and *Shimamura, M.*: Mechanisms of interlimb reflexes in cat. J. Neurophysiol., 1961. *24*: 665-676.

(9) *Hernádez-Peón, Scherrer, H.* and *Jouvet, M.*: Modifcation of electric activity in cochlear nucleus during "attention" in unanesthetized cats. Science, 1956, *123*: 331-332.

(10) *Livingston, R. B.*: Central control of receptors and sensory transmission systems, Chapter 31 (pp. 741-760) in: Field, J., Magoun, H. W. and Hall, V. E. (eds.) Handbook of physiology, Section 1: neurophysiology., Vol. 1, Washington, D. C., American Physiological Society, 1959, xiii + 779 pp.

(11) *Livingston, R. B.; Paillard, P. Tournay, A. et Fessard, A.*: Plasticité d'une synergie musculaire dans l'exécution d'un mouvement volontaire chez l'homme. J. Physiologie., 1951, *43*: 605-619.

(12) *Shimamura, M.*: Longitudinal coordination between spinal and cranial reflex systems. (Accepted for publication.)

(13) *Shimamura, M.* and *Akert, K.*: Peripheral nervous relations of propriospinal and spino-bulbo-spinal reflex systems. (Submitted for publication.)

(14) *Shimamura, M.* and *Livingston, R. B.*: Longitudinal conduction systems serving spinal and brain-stem coordination. J. Neurophysiol., 1963, *26*: 258-272.

(15) *Starr, A.* and *Livingston, R. B.*: Long-lasting nervous system responses to prolonged sound stimulation in waking cats. J. Neurophysiol., 1963, *26*: 416-431.

The Influence of Postural Set on Pattern of Movement in Man*

FRANK PIERCE JONES, Ph. D.
Tufts University. Institute for Psychological Research
Medford 55, Massachusetts, U. S. A.

In psychology, the term set is often applied to an attitude of expectancy which affects the character of a response by limiting its scope. You can recognize a set most easily on those occasions when the expected does not happen: when your mouth is prepared for sugar and receives salt; when the suitcase is full which you expected to be empty (or vice versa); when you go up stairs in the dark and there is one less step than you were prepared for.

Discussing such examples, Hebb says[5]: "If the higher animal responds in two different ways to the same total pattern of stimulation, it is because the activity of the central switchboard is not the same on the two occasions but "set" differently; as a result, the sensory input is routed to different muscles."

In this definition, Hebb seems to be confining the concept of set to the central nervous system and to be relegating changes in muscular tension to the response. As the word is commonly used, however, it comprises a postural (and muscular) as well as a central pattern. It is in this wider sense that I shall use the term in this paper — as a "postural change that precedes and accompanies movement". Set in this sense may be generalized as an "alertness response" or "orienting reaction" that prepares the organism for any contingency; or it may be specific and aimed at a particular goal. An example of the former is a basketball player who is prepared for his opponent's move although he doesn't know what that move will be. An example of the latter is a runner who is "set" for a race. In both cases the set must have an inward, switchboard component; but it also has an outward and visible component [2, 3, 4].

The inward component of set is (at present) accessible only to introspection**. The outward component is observable, however, and under certain conditions can be controlled experimentally. In this paper I am going to describe an empirical method for changing habitual set and demonstrate how the change affects a response pattern.

In the majority of experimental studies set has been measured by recording the changes in muscular tension in the part to be moved. Other measures which have been used are changes in heart-rate, breathing, and the galvanic skin response, which sometimes accompany expectancy and set. None of these measures gives a clear and unequivocal picture, though all seem related to the phenomenon. There is another index, however, which is regularly associated with the beginnings of specific goal-directed

* The research reported here was carried out at the Institute for Psychological Research under grants from the Carnegie Corporation of New York and the U. S. Public Health Service (Research Grant GM-04836) with the assistance of the Tufts University Fund for Research in Kinesthesis and the Katherine Bowditch Codman Fund. The x-ray photographs were obtained with the cooperation of Hal F. Knowlton of the Tufts University Health Service.

** The masking of the alpha rhythm in the electroencephalogram seems too general a phenomenon to be useful as an index of set, in the meaning which the term has been given here.

activities. When the stimulus for an habitual, learned movement is received, a change can frequently be observed in the relation of head to trunk. In some instances this change registers merely as an increase of tension in muscles attached to the head; in others, the head is measurably displaced. Though hard to detect with the naked eye, this preliminary head displacement can often be recorded photographically. Thus a photographic study of sitting posture in seven adult males showed a mean difference of ten degrees in the angle of the head depending on whether the subject expected to stand up or not after the picture was taken. The procedures were repeated four times for each subject. Though the position of the trunk remained virtually unchanged, the head angle changed in the same direction 25 out of 28 times[10].

The preliminary set of the head, which varies in pattern from subject to subject, persists into the movement itself. To study the relation between movement and set we have used a technique of color-coded multiple-image photography[7] in which markers of reflecting tape are recorded by a strobe light flashing at rates of five, ten, and twenty fps. Pictures are taken in profile (Fig. 1). The position of the head in space and its angle of rotation are defined by a one-inch strip of tape with the forward end centered in the Frankfort plane [6], half-way between the tragion of the ear and the lowest point of the orbit. The relation to the trunk is defined by a second marker attached over the seventh cervical vertebra. (Additional markers are attached to the trunk and limbs when more information is wanted). When the subject moves, the markers are recorded as a regular time-space pattern. (Fig. 2). This pattern shows both the initial set and the changes that take place during movement. A five-color filter wheel rotating in front of the camera in synchrony with the strobe changes the color of successive flashes. In the original

Fig. 2. — *Stroboscopic multiple-image photograph of movement from sitting to standing. Subject is wearing black jacket to provide more contrast for arm and neck markers. Strobe at 10 flashes per second.*

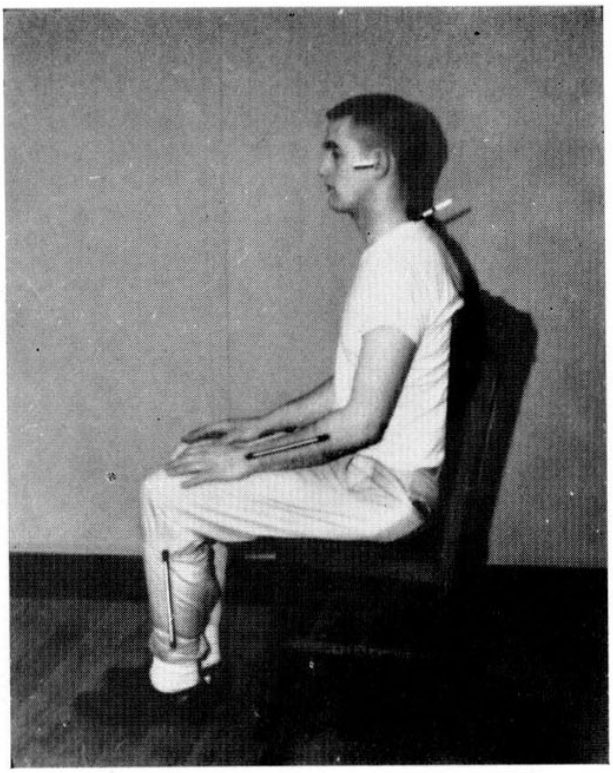


Fig. 1. — *Subject with markers of reflecting tape on head (in the Frankfort plane), neck (over the 7th cervical vertebra), lower arm, and leg.*

transparency from which the illustration for Fig. 2 was taken, color shows which points in the trajectories of different body parts belong together in time.

The movement from sitting to standing, which has been used to illustrate this paper, is well suited to photographic analysis. Though it is completed in a very brief time, it has a clear-cut pattern with a beginning, middle, and end. As a pattern, it has characteristic features which are apparently established early in childhood. Subjects show marked individual differences which persist from trial to trial. Patterns obtained in this way provide a wealth of quantitative data which can be treated graphically and statistically[8, 12].

Fig. 2 was obtained from a normal subject. In Fig. 3 the pattern is compared with three other normal and four abnormal patterns. To make the comparison, the transparencies were projected and successive positions of the head (at the center of the Frankfort plane) and the neck (over the seventh cervical vertebra) were traced off. The Frankfort plane itself was then drawn in for each position. Finally, the two trajectories were connected by straight lines at the beginning and end of the movement and at the point where the forward thrust of the head was greatest. The angle which these lines make with the horizontal measures the height of the head relative to the trunk.

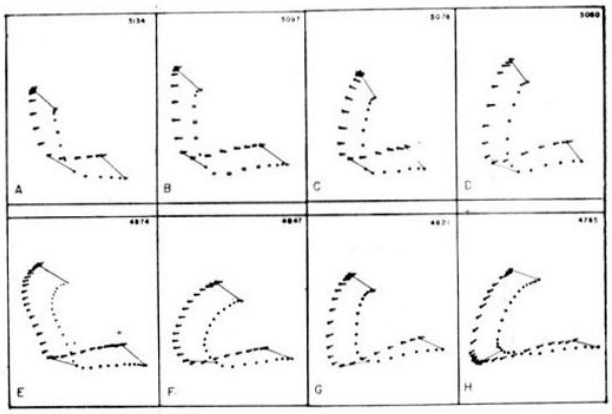


Fig. 3. — *Movement from sitting to standing. Comparison of four normal (A, B, C, D) with four abnormal (E, F, G, H) patterns. Trajectories of head and neck were traced from projected transparencies and connected at beginning and end of movement and at point where head is lowest relative to neck.*

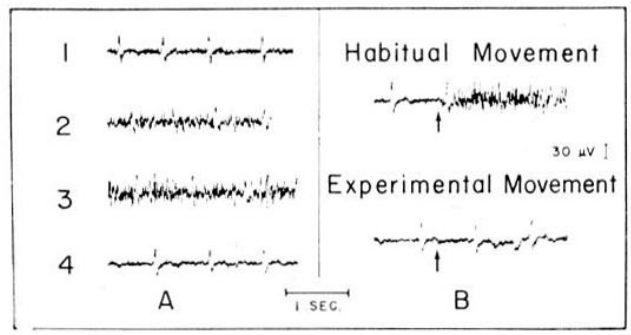


Fig. 4. — *Changing the postural set, I: Electromyograms to illustrate differences in the behavior of the sternomastoid muscle. A. Four sitting postures compared. In 1, the subject is sitting in his "most comfortable" posture; in 2, he has straightened up into a more erect posture; in 3, he is at his "greatest sitting height"; in 4, the poise of his head has been changed and he has been guided into the "experimental posture" described in the text. B. Habitual and experimental movements compared. In both patterns the subject is moving up from a relaxed to an erect sitting posture. Arrow marks signal for movement.*

The normal patterns were taken from graduate students at Tufts University, the abnormal from patients in the neurological ward of the V. A. Hospital in Boston *. Though there are individual differences within each group, the abnormals are distinguished from the normals by differences in velocity, in the head-trunk angle, and in the shape of the head trajectory. Data for the same movement performed as quickly as possible by the two groups of subjects (normal and neurological) have been given graphical and statistical treatment and are published elsewhere[13]. In these patterns, also, differences in head-

* Photographs were taken with the cooperation of Dr. F. A. Quadfasel, Chief of Neurology. The four subjects whose patterns are reproduced in Fig. 3 were diagnosed as follows: E (4874), Huntington's Chorea; F (4847), Cervical Spondylosis & Alcoholic Nutritional Polyneuropathy; G (4821), Basilar Impression and Obstruction on Vertebral Laminography; H (4765), Cerebellar Degeneration Secondary to Alcoholism.

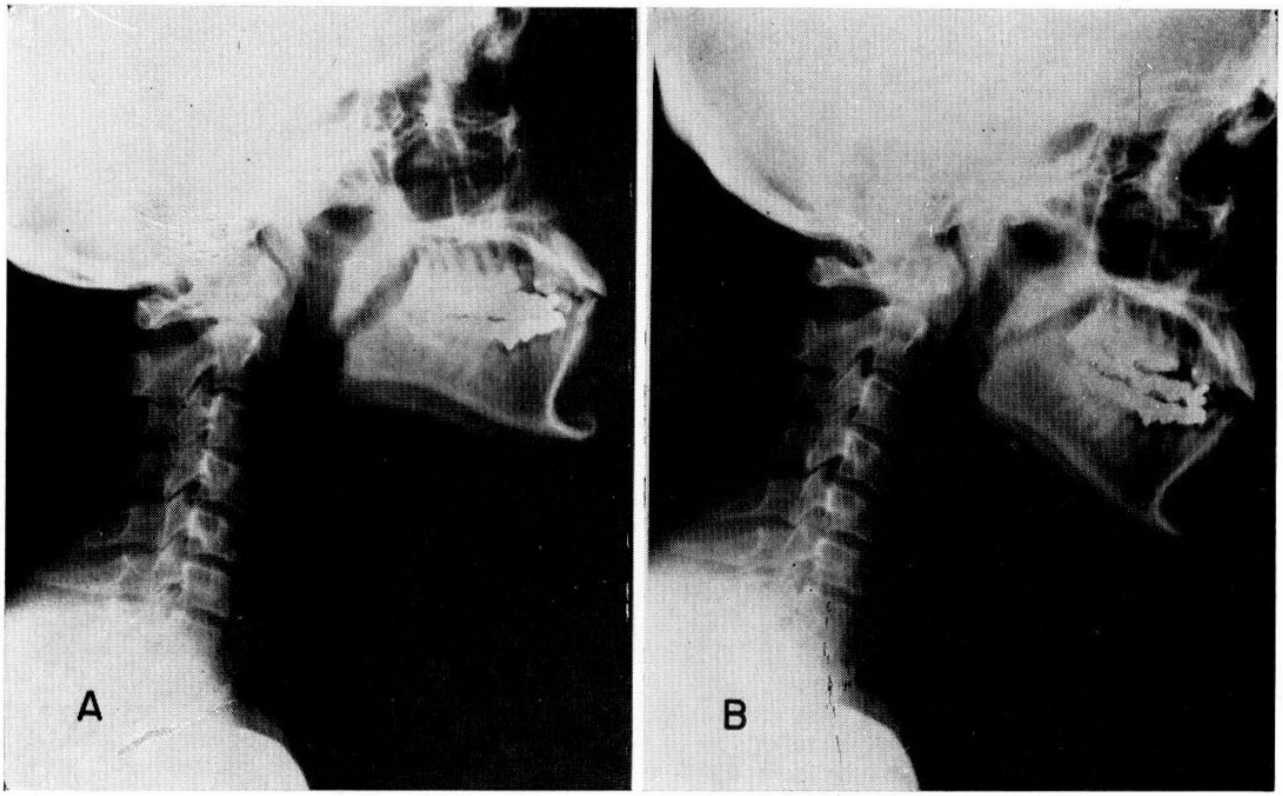


Fig. 5. — *Changing the postural set, II: X-ray photographs showing differences in poise of the head in two erect sitting postures.* A. *Habitual.* B. *Experimental. (The x-ray photographs were obtained with the cooperation of Mr. Hal F. Knowlton of the Tufts University Health Services.)*

trunk angle are closely related to differences in velocity and the shape of the head trajectory.

The shape of the head trajectory changes somewhat if the subject is asked to move more quickly or more slowly, but when speed is kept constant the trajectory remains essentially the same. It can be radically altered, however, by changing the poise (or tonic balance) of the head *. Since the concept of poise is dynamic rather than static, the poise of the head cannot be defined by spatial coordinates alone. The length and tension of the muscles and ligaments attached to it must also be taken into consideration. When a subject is asked to make some movement against gravity (e.g. by straightening up from a relaxed to a more erect posture) it has been found that a significant increase takes place in the tension of muscles attached to the head, especially the sternomastoid[10, 11]. With observation and practice, the experimenter learns to recognize this increase in neck-muscle tension. Once it has been recognized it can be inhibited by applying a light upward pressure around the insertions of the sternomastoids. The amount of pressure cannot be determined in advance but is regulated by the tensional response of the subject, which it is just sufficient to counterbalance. In the new posture, the subject's head feels lighter to the experimenter and is less resistant to passive movement. If, on the other hand, the pressure has been excessive or misapplied, stretch reflexes will be set up in the neck muscles and the head will be more difficult to move instead of less.

The effect of this procedure on the behavior of the sternomastoid muscle is

* This method for changing a postural set is derived from the procedures described by F. M. Alexander in *Constructive Conscious Control of the Individual*[1].

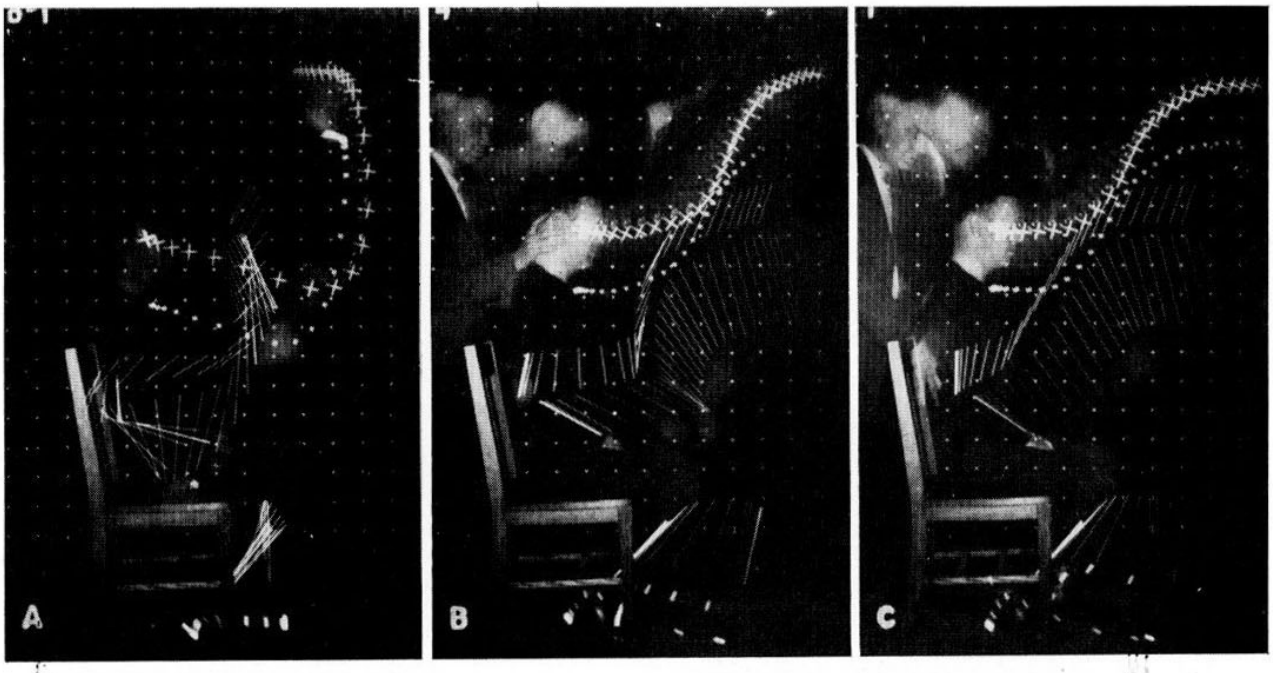


Fig. 6. — *Changing the postural set, III: Effect of change on gross movement pattern. Multiple-image photographs of movement from sitting to standing. Subject has markers of reflecting tape on head (centered in Frankfort plane), sternal notch, upper arm, lower arm, leg, right foot, left foot. Strobe at 10 fps.* A. *Habitual movement.* B. *Experimental 1; poise of subject's head maintained by experimenter throughout movement.* C. *Experimental 2; poise of subject's head changed before movement starts; movement initiated by experimenter with hand at subject's back.*

shown by electromyograms in Figure 4. The effect on the posture of the head and neck is shown by x-ray photographs in Figure 5. In the experimental posture on the right, the head has rotated slightly forward; the cervical curve has been slightly reduced; and the distances between the spines of the vertebrae are greater. It can be inferred that the extensors of the head and neck have lengthened[9]. This effect is tonic and tends to persist without conscious effort on the part of the subject.

Once the poise of the head has been changed by the means which I have described, a stimulus to move will elicit a totally different pattern of response. Figure 6A shows a subject moving from sitting to standing in his habitual pattern. He has markers on his leg, lower arm, upper arm, and sternal notch. The center of the Frankfort plane is marked with a small cross. Before the movement begins he sits in characteristic fashion with head tipped back. With the stimulus to move, the head is rotated rapidly forward and down to follow a pathway which approaches some of the abnormal patterns shown earlier. In B the postural set has been changed. The instructions to the subject (i.e. to stand up) are the same as before, but while he carries them out the change in the head balance is maintained by the experimenter. The upward movement begins almost at once, following an S curve which carries the subject into a walk. The change in the head trajectory is reflected by a change in the pattern of the arm. In C the movement is repeated. This time the subject's attention is called to the change in head balance and he is asked not to alter it but to allow the experimenter to initiate the movement with a hand at his back. As before, the pattern changes in all of its trajectories.

Apart from the differences in trajectory, the most striking feature of the experimental movement is the marked reduction in the feeling of weight observed both by the subject and by the experimenter. To the subject, his own weight, judged by the effort he puts forth to move, seems to be

reduced from a third to a half; a comparable difference is felt by the experimenter when he initiates the movement.

The operations for changing the pattern of movement can be performed with any normal subject. They can be demonstrated most easily, however, with a young adult. To get an idea of the range we took slow-motion photographs of 14 male undergra-

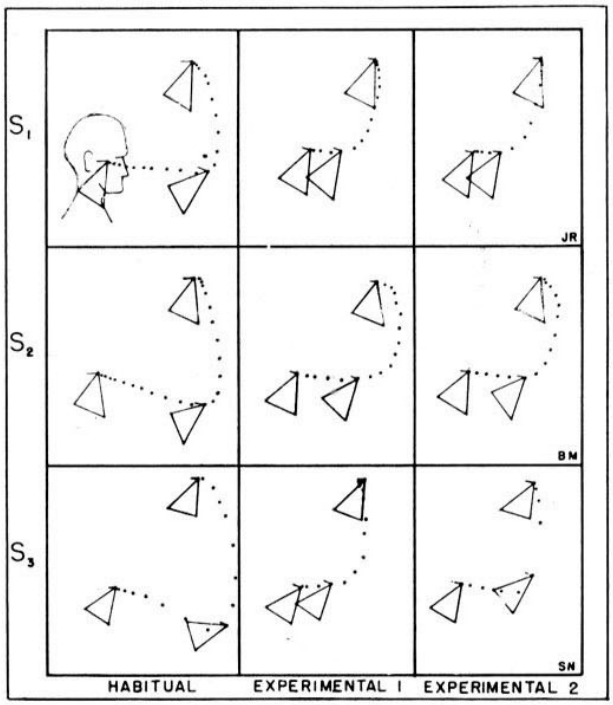


Fig. 7. — *Head trajectories of three subjects in movement from sitting to standing. Habitual movement contrasted with two experimental movements. Patterns traced from motion picture films. Triangles constructed by connecting sternal notch, 7th cervical vertebra, and center of Frankfort plane. Time interval app. 1/10 sec.*

duates. They wore swimming trunks, with markers of black tape on head, neck, trunk, and limbs. Pictures were taken in profile against a pair of coordinates. A mirror in the field gave a simultaneous record of the procedures from the front. Three habitual movements from sitting to standing were recorded, one at normal speed, one slow, and one fast. The postural set was then changed and two experimental movements were recorded, similar to those of Figure 6, B and C. A change in the predicted direction took place in all of the subjects. Seven of them were selected and the film strips edited for continuous viewing. The habitual movements show individual differences in the way the subject uses his head, trunk, and arms. All of these differences tend to disappear in the experimental movements, which have a smoother and more uniform character. One of the most striking changes appears in the facial expressions of some of the subjects when they notice the difference in the way they are moving. The head trajectories of three subjects for the first habitual movement and the two experimental movements were traced from the projected film and are reproduced in Figure 7.

The purpose of this paper has been to demonstrate a phenomenon which I believe to be of singular importance for understanding behavior, both normal and pathological. It is not my intention here to advance a theory of mechanism. Instead, I shall conclude by listing a number of general statements about the phenomenon which a theory of mechanism must take into account:

1. A learned movement is a function of the postural set that precedes it, and this set is a function of the spatio-tensional relation of head to trunk.

2. The inhibition during movement of a tendency to shorten muscles attached to the head facilitates extensor tonus in the trunk and limbs.

3. When the habitual set of the heads is inhibited in this way, the pattern of movement changes in the direction of greater simplicity and greater uniformity from subject to subject.

The sit-to-stand movement was chosen for demonstration because of its striking pattern. The operations by which the pattern was changed can be applied with similar effect to any movement. The only prerequisite is the consent of the subject to employ what Sherrington called "negative attention"[11], and inhibit his habitual response to the stimulus to move.

SUMMARY

In this study set is defined as "a postural change that precedes and accompanies movement." One index of postural set that can often be observed is a change in the poise or tonic balance of the head at the start of an habitual movement. A representative movement, that of sitting-to-standing, has been studied by color-coded, multiple-image photography. Pictures are taken on color film by repetitive strobe and coded in five colors by a filter wheel rotating in front of the camera. Subjects wear dark clothing. The relation between head, trunk, and limbs is marked with reflecting tape. This relation, which changes during the movement, is recorded as a time-space pattern on a single film to show both the postural set and the kind of movement that is associated with it. Patterns taken from normal subjects are compared with patterns taken from patients in the neurological ward of a Boston hospital.

Though the pattern of an habitual movement is characteristic of an individual and remains remarkably constant from trial to trial, it can be changed radically by changing the tonic balance of the head in such a way that the muscles attached to the head do not shorten during a movement. The procedure used in making the change is illustrated by multiple-image photographs, x-ray photographs, and electromyograms. The effect of the procedure on movement is demonstrated by slow-motion photographs of seven normal adults before and after the change in head balance. The patterns of movement can be seen to change in the direction of greater simplicity and greater uniformity from subject to subject. Head trajectories are consistently higher and shorter. It is concluded that "the inhibition during movement of a tendency to shorten muscles attached to the head facilitates extensor tonus in the trunk and limbs".

RESUMEN

En este estudio el término ajuste será empleado en el sentido de: "cambio postural que precede y acompaña el movimiento". Un índice de ajuste postural que puede observarse a menudo, es el cambio en el equilibrio tónico de la cabeza al comienzo de un movimiento habitual. Se ha estudiado un movimiento característico, el de levantarse de la posición sentada, por cronofotografía, distinguiendo las imágenes entre sí por el color, mediante la rotación frente al objetivo de la cámara, durante la toma de las vistas, de un filtro circular que presenta en forma sucesiva cinco sectores diversamente coloreados. Los individuos llevan ropas oscuras. Las posiciones de la cabeza, del tronco y de los miembros son señaladas con cinta reflejante. Estas posiciones, que varían durante el movimiento, son registradas como curvas espaciotemporales, sobre un solo film de manera de mostrar el ajuste postural y el género de movimiento con el cual está asociado. Se han comparado curvas provenientes de individuos normales con otras tomadas de pacientes neurológicos de un hospital de Boston.

Bien que el esquema de un movimiento habitual sea característico para cada individuo y prácticamente constante de un experimento a otro, se le puede cambiar radicalmente modificando el equilibrio tónico de la cabeza haciendo que los músculos que en ella se insertan no se acorten durante el movimiento. Las técnicas empleadas para obtener este cambio están ilustradas por fotografías estroboscópicas, radiografías y electromiogramas. El efecto del procedimiento sobre el movimiento está demostrado por vistas tomadas lentamente, de siete adultos normales antes y después del cambio en el equilibrio tónico de la cabeza. Se puede ver que los esquemas del movimiento tienden en todos los sujetos, hacia una mayor simplicidad y una mayor uniformidad. Las trayectorias de la cabeza

son siempre más elevadas y más cortas. Se llega a la conclusión que: "durante el movimiento si se inhibe la tendencia al acortamiento de los músculos insertados en la cabeza, se refuerza el tono de los músculos extensores del tronco y de los miembros".

RÉSUMÉ

Dans cette étude le mot ajustement(1) sera pris dans le sens de: "changement postural qui précède et accompagne le mouvement". Un indice d'ajustement postural que l'on peut souvent observer est le changement dans l'équilibre tonique de la tête au début d'un mouvement habituel. On a étudié un mouvement caractéristique, celui de se lever quand on est assis, par chronophotographie, les images étant distinguées l'une de l'autre par la couleur grâce à la rotation devant l'objectif de la caméra, pendant la prise de vues, d'un filtre circulaire présentant successivement cinq secteurs diversement colorés. Les sujets portent des vêtements sombres. Les positions de la tête, du tronc et des membres sont marquées au moyen de bandes refléchissantes. Ces positions, qui varient durant le mouvement, sont rapportées, sous forme d'un système de courbes spatio-temporelles, sur un seul film de façon à représenter et l'ajustement postural et le genre de mouvement avec lequel il est associé. On a comparé des courbes provenant de sujets normaux avec d'autres provenant de patients de la section neurologique d'un hôpital de Boston.

Bien que le schéma d'un mouvement habituel soit caractéristique pour un individu donné et reste remarquablement constant d'une expérience à l'autre on peut le changer radicalement en modifiant l'équilibre tonique de la tête de façon à ce que les muscles se rattachant à la tête ne se raccourcissent pas durant un mouvement. Les techniques employées pour obtenir ce changement sont illustrées par des photographies stroboscopiques, des radiographies et des électromyogrammes. L'effet du procédé sur le mouvement est démontré par des vues, prises au ralenti, de sept adultes normaux avant et après le chagement dans l'équilibre tonique de la tête. On peut voir que les schémas du mouvement tendent, chez tous les sujets, vers une plus grande simplicité et une plus grande uniformité. Les trajectoires de la tête sont toutes plus élevées et plus courtes. On en conclut que: "durant le mouvement l'inhibition d'une tendance à raccourcir les muscles attachés à la tête renforce le tonus des muscles extenseurs du tronc et des membres".

(1) En anglais "set".

ZUSAMMENFASSUNG

In dieser Arbeit bezeichnet Haltungsbereitschaft (set) die "Haltungsveränderung die der Bewegung vorangeht und begleitet." Ein Kennzeichen der Haltungsbereitschaft, das öfters beobachtet werden kann, ist eine Veränderung in dem Gleichgewicht oder der tonischen Balance des Kopfes zu Beginn einer gewohnten Bewegung. Eine representative Bewegung, die vom Sitzen zum Stehen, wurde durch farbverschüsselte Serienbildphotographie untersucht. Bilder sind mit wiederholendem Stroblicht in Farbe aufgenommen und durch ein vor der Kamera sich drehendes Filterrad in fünf Farben verschlüsselt worden. Versuchspersonen tragen dunkle Kleidung. Das Verhältnis zwischen Kopf, Rumpf und Gliedern ist mit reflektierenden Bändern markiert. Dieses Verhältnis, welches sich durch die Bewegung hindurch verändert ist auf einem einzelnen Filmstreifen als raum-zeitliche Bewegungsgestalt aufgenommen, um die Haltungsbereitschaft und die damit zusammenhängende Bewegungsart zu zeigen. Muster von normalen Versuchspersonen sind dann mit Mustern verglichen, die von

Patienten in der Neurologischen Abteilung eines Bostoner Hospitals aufgenommen worden sind.

Obwohl das Muster von einer gewohnten Bewegung charakterisch für die individuelle Person ist, und auffallend unveränderlich von Versuch zu Versuch bleibt, kann es radikal verändert werden wenn man die tonische Balance des Kopfes so verändert, dass sich die Muskeln am Kopfe nicht bei der Bewegung verkürzen. Das Verfahren das benutzt wird um diese Veränderung zu verursachen ist durch Serienbildphotographie, Röntgenaufnahmen und Elektromyographie illustriert. Die Wirkung dieses Verfahren auf die Bewegungsart ist durch Zeitlupenaufnahmen von sieben normalen Erwachsenen vor und nach der Veränderung der Kopfbalance dargestellt. Die Muster der Bewegungsart verändern sich, wie man sehen kann, in die Richtung der Einfachheit und grösseren Gleichheit von Versuchsperson zu Versuchsperson. Die Bewegungsbahnen des Kopfes sind stets hoher und kürzer. Man schliesst aus der vorliegenden Arbeit, "wenn man während der Bewegung den Hang zur Muskelverkürzerung hemmt, so wird der Muskelton der Streckmuskeln in Rumpf und Gliedern verstärkt."

Discussion by Dr. HAROLD SCHLOSBERG

The concept of "Set" has long played an important role in psychological theorizing. Sometimes it is a preparation for perceiving an ambiguous stimulus situation in a specific way. For example, the printed word LEAD may be interpreted as the name of a metal, or as a verb meaning "to guide." The meaning may be determined by context in ordinary reading, or by intructions, as when we ask for "The chemical symbol for the following." This type of set would seem to be clearly central (i.e., cortical), and is a very useful construct in experiments ranging from controlled association to problem solving; for example, it is very difficult to "make four equilateral triangles from six matches" because the person is "set" to stick to two dimensions, rather than to make a three-dimensional figure.

At the other extreme, "set" has been referred to response variables. Thus, the reaction time to a click will be more rapid if the *S* is told "PRESS THE KEY as soon as you hear the click," rather than "WAIT UNTIL YOU HEAR THE CLICK, then press the key." The assumption, for which there is some experimental evidence, is that the muscle tension in the right forearm is higher under the "motor set" than under the "sensory set." the trouble is that the tension (as measured by EMG) may also be higher in all muscles of the body, not just in those used in the response for which *S* is prepared.

A vast amount of research has been devoted to measuring the physiological changes involved in preparing to respond. There are changes in circulation, breathing, electrical skin resistance, and a host of other autonomic functions, but they tend to be general, rather than specific to the prepared response. Recently, these changes have been related to activation of the reticular formation by the Electroencephalographers. Dr. Elizabeth Duffy has given us a most useful summary of these changes in her Monograph, "The Energetics of Behavior." But her summary leaves us with the feeling that the pieces of the jigsaw puzzle don't fit together to give us a true picture.

What seems to be lacking is a specific or selective factor. All of the work on "Set" indicates that, in addition to an increased alertness (or general tension), there is activation of a specific system, with inhibition of competing systems. Thus, when one is instructed to respond with the class to which a member belongs, and he is given the stimulus word "DOG", the tendency to say "Animal" or "Mammal" is strengthened, and the tendency to name a specific example, as "Rover" is inhibited. Electroencephalographers are just beginning to study activation of specific systems; for example, on a recent visit to Sokolov's lab in Moscow, the writer was told that a sleeping dog would show activated brain waves when

his name was mentioned, but not to other words at equal loudness level.

This may seem remote from what Dr. Jones has told us, but I think it serves as a framework to understand his findings. When a person is told to "Stand Up," there is a generalized increase in activation, as a preparation for action. This shows clearly in many physiological indices, perhaps most clearly in an increase in skin conductance. But part of this increased level of activation is a tightening of the neck muscles, which is inconsistent with the specific postural response of getting up. By adjusting the head balance, Dr. Jones can modify this discordant key element, permitting the normal response to proceed smoothly.

But why do so many of us make this discordant tightening of the key postural muscles in the neck? Instead of sitting back in our armchairs, and blaming it on vertical posture, poor training, or natural perversion, we need more research; how do the head-neck relations develop in Phylogeny, and in Ontogeny? In other words, how do we get this way? Dr. Jones has given us excellent techniques to study these basic problems.

Discussion by Dr. GRAYSON McCOUCH

This field of work has been too long neglected by both physiologists and clinicians. I should like to pay tribute not only to the method of treatment described, but to the recording of its results. Dr. Jones has tactfully refrained from giving a reflex analysis, presumably counting upon the discussion.

When his normal subject was not guided, his head was tipped backward in the resting position. If you react as I do, and rise from such a sitting posture, you may notice a contraction of the muscles of the back. On standing the weight falls on the heels, requiring a sway forward to regain balance. If, instead, the head be tipped slightly forward, the muscles of the back relax and weight is thrown either on the ball of the foot, thus leading to a step forward, or between the heel and the metatarsophalangeal joint, giving a balanced standing posture. The unguided situation recalls a reflex described by Landau in infants from six months to two years of age. When in the prone position, the head is tipped backward by the righting reflex from the labyrinth. The neck righting reflex then induces contraction of the muscles of the back. In the upper extremities postural reflexes from both neck and labyrinth extend the arms. In the legs these reflexes are opposed. In only ten percent of the infants examined does the labyrinth dominate, giving extension at the hip. On bending the head forward, the muscles of the back relax and trunk and extremities are flexed.

Though submerged in the adult by suprasegmental factors, the neck reflexes continue to influence posture and play a significant role in the beneficial results induced by suitably guided posture of the head, which leads to balance with a minimum of muscular effort. With the head tipped backward, the postural neck reflex inhibits extension of the legs; with ventroflexion it favors such extension and thus facilitates standing. In the great majority of people, these neck reflexes are stronger than those from the labyrinth which oppose them.

Perhaps it is significant that the receptors for the postural neck reflexes are located in the intervertebral joints and not (as the textbooks persist in stating) in the muscles. If, instead of stressing relaxation, we emphasize correct posture, suitable muscular coordination may follow reflexly without further conscious guidance.

Dr. F. P. JONES

I want to thank Dr. McCouch and Prof. Schlosberg for their helpful and stimulating comments. Because of the short time at my disposal, I hesitated to advance a theory of mechanism. Since the question has been brought into the discussion, however, I

should like to express my view as briefly as I can. I believe that under civilized conditions the posture of the average person represents an imperfect integration of reflex and voluntary elements. In moving into the upright posture, (from sitting to standing, for example) the muscles which are used to orient the head shorten unduly; pressure is exerted on the neck; and the neck righting reflex is partially inhibited. When the movement is completed, the head has been righted, but the full extension of trunk and limbs which is appropriate to the upright posture in man has either not been achieved or has been achieved without reflex facilitation and can be maintained only with effort. When an animal is in the upright posture, gravity keeps the muscles and ligaments attached to head under stretch. In man, the same balance of forces operates, but it is a more delicate balance. If it is upset, the gravitational force, instead of restoring the balance, may be so directed that the head becomes a dead weight under which the cervical spine begins to sag forward and down. In this way, a vicious circle can be set up in which the attempt to right the head inhibits the neck righting reflex. The experimental procedures which I have described break into this vicious circle. The muscles attached to the head are kept from shortening and the righting reflex is facilitated as soon as movement begins. The result is an increase in extensor tonus which is perceived by the subject as a decrease in the feeling of weight.

The question has been raised how such a condition of malposture could develop and why it should continue. I have already suggested an answer to the first question. The answer to the second is partly to be found, I believe, in the principle of adaptation. Malposture does not "feel bad enough." When it is recognized as "bad", the problem is posed of how to change it. As Professor Schlosberg has pointed out, our problem-solving behavior is commonly hampered by sets. The example which he used of the match problem whose three-dimensional solution was blocked by a two-dimensional set is appropriate here. I believe that a three-dimensional set blocks the solution of the postural problem. The concept of "correct posture" as it is commonly held represents a spatial configuration of parts which can be achieved by will power and maintained by exercise. Posture, however, is not static but dynamic. The configuration of parts is continually changing. To allow for change, a concept of posture is needed which is organized in time as well as in space. The value of direct kinesthetic experience is that it serves to break up a set and thus facilitates the formation of a new concept.

REFERENCES

(1) *Alexander, F. M.*: Constructive Conscious control of the Individual. New York: E. P. Dutton, 1923; London: Chatterson, 1946.

(2) *Allport, F. H.*: Theories of Perception and the Concept of Structure. New York: Wiley, 1955. Chap. 9.

(3) *Berlyne, D. E.*: Conflict, Arousal, and Curiosity. New York: McGraw-Hill, 1960. Chap. 4.

(4) *Gibson, J. J.*: A critical review of the concept of set in contemporary experimental psychology. Psychol. Bull., 1941, 38, 781-817.

(5) *Hebb, D. O.*: A Textbook of Psychology. Philadelphia & London: W. B. Saunders, 1958, 63.

(6) *Howells, W. W.*: The designation of the principal anthropometric landmarks on the head and skull. Am. J. Phys. Anthrop., 1937, 22, 477-495.

(7) *Jones, F. P. & O'Connell, D. N.*: Color coding of stroboscopic multiple-image photographs. Science, 1958, 127, 1119.

(8) *Jones, F. P. & O'Connell, D. N.*: Posture as a function of time. J. Psychol., 1958, 46, 287-294.

(9) *Jones, F. P. & Gilley, P. F. M.*: Head balance and sitting posture: an x-ray analysis. J. Psychol., 1960, 49, 289-293.

(10) *Jones, F. P.; Gray, F. E.; Hanson, J. A., & Shoop, J. D.*: Neck-muscle tension and the postural image. Ergonomics, 1961, 4, 133-142.

(11) *Jones, F. P.; Hanson, J. A., & Gray, F. E.*: Head balance and sitting posture II: the role of the sternomastoid muscle. J. Psychol., 1961, 52, 363-367.

(12) *Jones, F. P., & Hanson, J. A.*: Time-space pattern in a gross body movement. Perceptual and Motor Skills, 1961, 12, 35-41.

(13) *Jones, F. P..; Hanson, J. A.; Miller, J. F., & Bossom, J.*: Quantitative analysis of abnormal movement: the sit-to-stand pattern. Am. J. Phys. Med., 1963, (in press).

(14) *Sherrington, C.*: Selected Writings (edited by Denny-Brown). New York: Harpers, 1940, 312-313.

The Motor Systems in Convulsive Movements*

A. EARL WALKER, M. D.
From the Division of Neurological Surgery, the Johns Hopkins University School of Medicine, Baltimore, Maryland.

Introduction

The motor manifestations of an epilepsy have been the most impressive aspect of convulsions. The mechanisms, which predicate their pattern, have been little studied. Dr. Fulton, who was so fascinated by the organization of the motor systems, observed the counterpart of convulsions in the course of stimulation of the motor cortex when afterdischarge occurred. It was such studies which confirmed the basic organization of the motor representation in the cerebral cortex suggested by observations of the march of a focal seizure.

Cortical Patterns

The patterns of convulsive seizures have seemed, at times, more complex than might be accounted for by a simple spread through the cerebral cortex. Certain aspects of the march have suggested a servo-mechanism which involves a projection to and a feedback from subcortical structures, usually considered to be the thalamus or reticular formation. The circuit relays facilitatory or inhibitory impulses to the primary focus and adjacent areas. To test this hypothesis a series of animals were prepared by ablation of the arm cortical motor and sensory areas. Since the application of an epileptogenic agent (penicillin) to the leg or face area was known to cause a rapid spread of electrical discharges along the motor cortex, and a clinical spread of the seizure to arm or leg, if a subcortical feedback were operative, after the ablation, such cortical stimulation should result in the same spread.

In all experiments, focal jerking was induced locally in the appropriate contralateral part of the body. In the case of injection into the motor leg area twitchings occurred in the opposite foot and toes; on only one occasion was there any evidence of spread to the arm. In that case (MM-III), the excision was found to have removed the inferior part of the arm area leaving intact the upper half. Even in this case, however, no twitching was noted in the contralateral face. In each of these animals, at a terminal experiment, cortical recordings were made after an epileptogenic focus was induced by the injection of 3,000 units of a freshly mixed aqueous penicillin into the motor area. Localized spiking occurred within 2 or 3 minutes, and was confined to the area above or below the ablation (fig. 1).

If penicillin was applied to one area (the face) and then subsequently to the leg area above, the independent spiking occurred in these two areas for a period of hours without becoming synchronous nor showing interference phenomena at any time. It may then be assumed that the Jacksonian march is a cortical phenomena and not dependent upon subcortical circuits for its propagation.

Role of the Cortico-Spinal Tract

Although the pyramidal or cortico-spinal tract has been assumed to be the pathway mediating convulsive movements, experimental studies have been inconclusive. In

* Aided by a grant from the National Institutes of Health, Bethesda, Maryland.

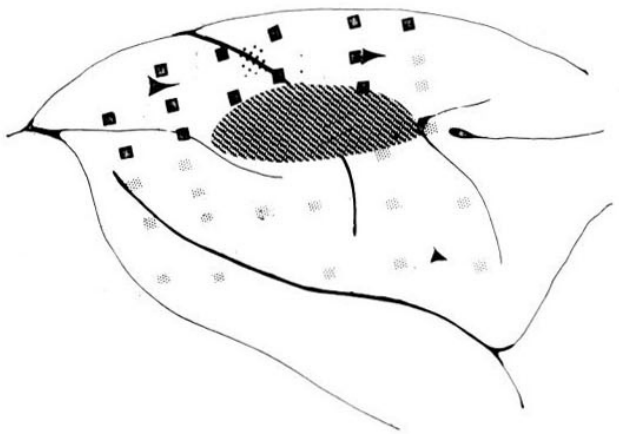


Fig. 1. — *(M-22) Sketch of the right hemisphere of a macaque monkey to show the site of a previous ablation of the motor and sensory cortex. Along the leg area of the central sulcus, penicillin was applied (dotted square) and spiking occurred at the electrodes indicated by solid black, but normal activity at the electrodes indicated by stippling.*

an endeavor to answer this question the left cerebral peduncle has been cut in seven monkeys and subsequently focal attacks were induced by the application of penicillin to the motor cortex, and generalized seizures produced by bilateral temporal electroshock, and intravenous metrazol.

In five animals with partial sections of the peduncle penicillin applied to the motor cortex induced focal fits on the sectioned side but the digits of the right hand participated very little. In the animals with more complete section, clonic fits could not be induced on the involved side. In the fits induced on the normal side, the hands participated with clonic flexions of the fingers.

In five animals generalized seizures were induced by electric shock. These fits usually characterized by initial flexion followed by clonic flexor contractions in all four limbs and face. In general, the right limbs did not participate as vigorously as the left. In the two animals with the most complete sections of the peduncle, the right limbs had little jerking. However, when 3 cc. of a 10 percent solution of metrazol was injected, intravenously, the generalized seizure was almost symmetrical, even in an animal which had a complete section of the peduncle. This is in line with a previous observation that the convulsion induced by intravenous metrazol in a unilateral hemispherectomized monkey involved all four extremities so symmetrically that one could detect only a slight difference in the convulsive movements of the two sides (fig. 2).

Thus, in pedunculotomized animals with a considerable portion of their corticospinal and frontopontine tracts severed, the motor pattern of focal and generalized seizures differ quantitatively and qualitatively from the attacks induced in normal animals. The difference between generalized seizures induced by electric shock and metrazol is striking and points to a different substrate for the two types of seizure. It would therefore seem that the corticospinal tract is not essential for the generalized seizure induced by metrazol, although in its absence the fine peripheral pattern of the focal attack is lost.

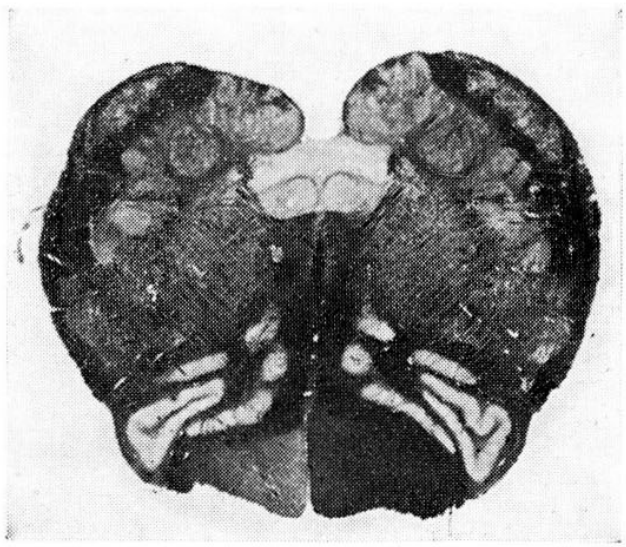


Fig. 2. — *Section through the medulla to show the marked demyelinization of the pyramid following a section of the left cerebral peduncle. Bitemporal electro-shock produced a generalized tonic-clonic convulsion except that the right fingers were not involved, and the attack stopped in the right limbs before the left. Penicillin applied to the left motor cortex induced only slight jerking in the right forearm and virtually none in the digits, although the attack became generalized.*

Role of the Reticular Formation

A number of papers have discussed the influence of the reticular formation upon cortical epileptic activity. A series of experiments have been carried out on six monkeys in which the brainstem reticular formation has been destroyed, unilaterally and bilaterally, in order to study the effect upon induced focal and generalized seizures.

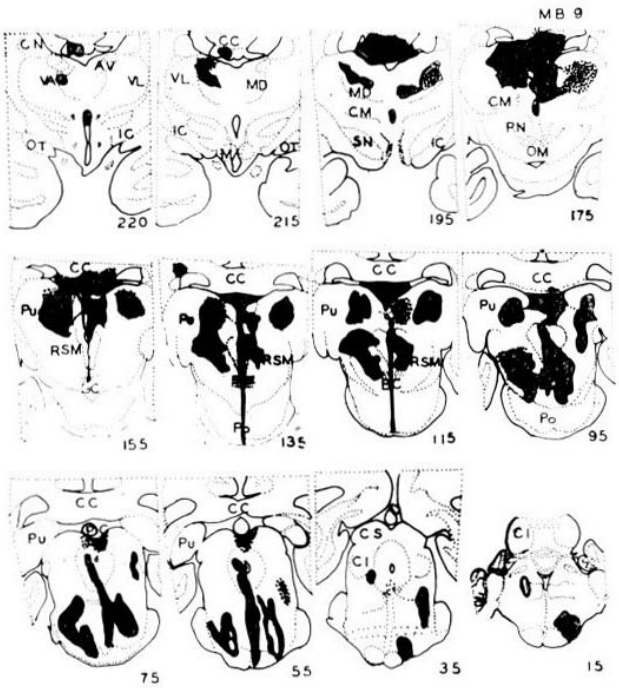


Fig. 3. — *Representative serial sections of the brain stem to show the extent of the lesion. Following these lesions penicillin applied to the motor cortex induced a focal clonic convulsion, and bitemporal electroshock a generalized tonic-clonic seizure. The abbreviations used in this illustration are as follow: AV, ventral anterior nucleus; BC, brachium conjunctivum; CC, corpus callosum; CI, inferior colliculus; CM, centrum medianum nucleus; CN, caudate nucleus; CS, superior colliculus; IC, internal capsule; MD, medial dorsal nucleus; OM, oculo-motor nucleus; OT, optic tract; PO, pons; PU, pulvinar; RN, red nucleus; RSM, mesencephalic reticular substance; SN, substantia nigra; VA, ventral lateral nucleus.*

Three of four animals had focal convulsions similar to control seizures and one animal had a less severe, although otherwise similar seizure. Generalized convulsions induced by electric shock in two of the five animals differed in no apparent manner from the control electroshock seizures. In two animals the tonic phase of the attack was essentially the same before and after the mesencephalotomy but the clonic phase was reduced in severity and time (fig. 3).

It must be admitted that the lesions produced in these animals did not in any case completely destroy the pontine or mesencephalic reticular formations. Yet, in several instances they were sufficiently large to involve the major part of the reticular formation. In spite of such lesions, both generalized tonic-clonic seizures could be induced by bitemporal electroshock and focal clonic convulsions by chemical stimulation of the motor cortex. The attacks induced in the animals with lesions of the reticular formation were somewhat less intense and shorter than those in normal animals. From such crude experiments one can only conclude that the basic pattern of focal and generalized fits in not greatly altered by the presence of extensive lesions of the brainstem reticular formation. This does not imply that the reticular formation plays no role in convulsive activity for the reticular influence on the cerebral cortex seems to be ambivalent, at times inhibitory and facilitatory. This ambivalent effect of the reticular formation acts upon the pattern of convulsive seizures without depreciating the importance of the influence of the reticular formation upon the general excitability of the cerebral cortex.

SUMMARY

The findings in these experiments are compatible with the thesis that the basic pattern of the tonic-clonic seizure is present in the brainstem and spinal cord, and may be activated by synergistic discharge of pyramidal and non-pyramidal fibers or by discharge of either system alone.

RESUMEN

Los hallazgos surgidos de estas experiencias son compatibles con la tesis según la cual el esquema básico del ataque tónico-clónico está presente en el tronco cerebral y en la médula y puede ser activado por descargas sinergísticas de fibras piramidales y no-piramidales o por descargas de cualquiera de los sistemas aisladamente.

RÉSUMÉ

Les découvertes extraites de ces expériences sont compatibles avec la thèse selon laquelle la crise tonique-clonique est présente dans le tronc cérébral et dans la moëlle, et peut être activé par des décharges synergistiques de fibres pyramidales et non-pyramidales, ou bien par des décharges de chacun de ces systèmes isolément.

ZUSAMMENFASSUNG

Die Ergebnisse dieser Experimente stimmen mit der These ueberein, dass die Grundform der tonisch-klonischen Kraempfe im Hirnstamm und dem Rueckenmark aktiviert werden koennen durch die synergistiche Entladung der pyramidalen und nicht pyramidalen Fasern oder durch die Entladung eines jeden einzelnen Systems fuer sich.

Discussion Dr. ROBERT B. AIRD

On the basis of acute and chronic lesions of various parts of the motor system — motor cortex (8 cases), cerebral peduncle (7 cases), reticular formation (5 cases), and other brainstem lesions, the pathways involved in the transmission of focal and generalized, experimentally induced epileptic seizures in the macaque monkey will be analyzed. In general, lesions of the cortico-spinal tract modify the pattern of the seizure, whereas, lesions of the reticular formation modify the degree and severity of the attack. Even complete hemispherectomy does not abolish the attack on the contralateral side, and only modifies its pattern to a minor degree. It seems that the basic pattern of clonic convulsive movements is represented in the lower brainstem and spinal cord and is activated by excessive discharge of the pyramidal and/or non-pyramidal fiber systems. The mechanism which transforms the normal functioning of the motor systems into tonic and clonic activity will be discussed.

Doctor Walker has presented convincing evidence for the cortical spread of focal epileptic discharge in explanation of the Jacksonian march. Our own evidence on the spread of epileptic discharge from a focus in humans would be in keeping with this. With EEG techniques which show the local spread of cortical discharge from a focus and which simultaneously record the development of mirror foci and generalized cerebral activity that so commonly accompanies gross epileptic foci, it was apparent that the first event to develop upon activation of the focus is concerned with the local cortical build-up and spread of the focal discharge. The development of mirror foci and of generalized dysrhythmias almost invariably appear at a later stage. This sort of evidence, of course, does not prove the presence or lack of a feed-back mechanism from subcortical structures in explanation of the Jacksonian march, but it strongly suggests that subcortical structures are not significantly involved. Doctor Walker's

study affords the final, direct evidence needed to prove this point.

I was even more interested in Doctor Walker's mesencephalotomies aimed at testing the role of the reticular formation in the propagation of focal and generalized seizures of cortical origin. Our own studies had suggested that some midline center in the brain stem, which controls the bilateral synchrony of the electrical activity of the two cerebral hemispheres, was involved in the propagation of focal epileptic discharge, at least in the development of generalized grand mal convulsions. This was suggested since such seizures developed, following the activation of an epileptic focus, only when a generalized dysrhythmia, which still retained its essential bilaterally synchronous pattern, became so exaggerated that it obscured the primary focus. Since this situation did not occur in seizures that remained focal, we had concluded that the brain stem center which mediates the synchrony of the electrical activity of the two cerebral hemispheres was not significantly involved and that this center, which we have called the 'pacemaker', was not required for the propagation of focal seizures. I should add that we are not certain that our 'pacemaker' center is the same as the reticular formation, although the latter possesses the anatomical requirements, namely, the location in the midline of the brain stem and the diffuse projections to both cerebral hemispheres. Regardless of this point, Doctor Walker's mesencephalotomies would probably have involved our 'pacemaker' since more recent brain splitting studies have indicated that it is likely at the pontine level and possibly lower levels.

Doctor Walker's evidence with respect to generalized convulsions was obtained by electric shock applied bitemporally. This, of course, is a different situation from our own, where the propagation of generalized seizures from a focus was studied. Nevertheless, the development of generalized convulsions in the presence of gross, bilateral mesencephalotomies would suggest, as Dr. Walker has concluded, that such convulsions do not depend upon a reticulo-cortical spread for their production as a result of bitemporal electroshock. The question remains as to whether or not massive, bilateral stimulation, as produced by bitemporal electroshock, is not directly or indirectly producing a cortical stimulation which in effect is the equivalent of the generalized cortical activation we have observed when generalized convulsions are produced by the propagation of focal epileptic discharge.

I have two other questions for Dr. Walker:

Your results indicate that the clonic phase of the generalized convulsions produced by bitemporal electroshock was reduced in severity and time. Did you measure the convulsive threshold of the animals before and after mesencephalotomy? In other words, is it possible that in keeping with the reduced severity and duration of the seizures, the convulsive threshold for electroshock was raised following mesencephalotomy?

If the convulsive threshold were raised, i.e., if stronger bitemporal electroshock stimulation were required, one might wonder about a spread of cortical stimulation which would fit in with the alternative interpretation I have suggested for your findings, namely that massive, bitemporal electroshock may produce a cortical stimulation which may approximate the generalized cortical activation we have observed when generalized convulsions develop as a result of the propagation of focal epileptic discharge.

My last question concerns EEG recording during your experiments. If such recording was obtained, were generalized dysrhythmias observed following focal chemical stimulation and bitemporal electroshock? Also, following such stimulation, was there a recorded activation of the brain stem below the mesencephalotomies? It would appear from your conclusions that the latter would be likely. It may be that cortical activation is not required as a component of the propagation of generalized convulsions and that brain stem activation secondary to focal cortical and electroshock stimulation may be the answer, as you have suggested. In this case the cortical

activation as we have recorded it would be a secondary accompaniment of the propagation of generalized convulsions from a cortical, epileptogenic focus, and not an integral part of the mechanism of propagation. For the reasons suggested in my previous question, however, I still have some doubts on this point. It seems to me that one of your statements which took care not to depreciate... "the importance of the influence of the reticular formation upon the general excitability of the cerebral cortex" was well taken. I am still not convinced but that the cortex may serve an important role in most patients in the propagation of focal epileptic discharge in the production of generalized convulsions. This might be true as a feed-back mechanism to prolong and intensify such convulsions, even if it did not serve a primary role in the production of generalized convulsions.

The Organization of the "Motor System"

HENRICUS G. J. M. KUYPERS*, M. D., Ph. D.
Department of Anatomy, Western Reserve University
School of Medicine, Cleveland 6, Ohio.

The era of neurological studies, in part symbolized by Fulton's name, has contributed considerably to the knowledge of cortical motor mechanisms. At the present time the attention has shifted. Nevertheless some of the questions of those days remain and some of the roads that were opened have not been traveled to the very end. Therefore, it seems most appropriate indeed to move the spotlight temporarily back to the classic "motor system". However, in doing so the impression is gained that the motor mechanisms attributed to the central cortex might have dominated excessively our concept of the "motor system" and thus might have prevented us from grasping the totality of its organization. Therefore, a more holistic concept of the motor system seems to be called for. In order to arrive at such a concept we propose to approach the system from below rather than from above, from the spinal segment instead of the motor cortex, because the motor potentialities of the higher centers of the brain are ultimately an expression of their degree of access to the motor neurons.

The spinal segment, our basic point of reference, contains motor neurons, neurons giving rise to long ascending pathways and propriospinal neurons. The axons of the latter terminate, among others, upon motor neurons and upon other propriospinal neurons (Cajal, 1955).

The motor neurons are located in the ventral and lateral parts of the ventral horn and display a somatotopic organization (Sprague, 1948; Romanes, 1951). The interneurons leading to motor neurons are located primarily in the zone intermedia and the dorsomedial parts of the ventral horn. This internuncial zone seems to be also somatotopically organized (fig. 1 a). The interneurons leading to motor neurons of distal musculature are primarily located laterally (Bernhard & Rexed, 1945), whereas those leading to motor neurons of proximal musculature are probably located ventromedially. Furthermore, the interneurons leading to flexor motor neurons are located primarily dorsally (Lloyd, 1944). Those leading to extensor motorneurons therefore might be located primarily ventrally.

The brain has immediate access to the internuncial zone throughout the entire spinal cord via the corticospinal and the long subcorticospinal pathways. The cortical fibers in the monkey (fig. 14C) terminate throughout the internuncial zone and in the lateral motoneuronal cell groups (Chambers and Liu, 1958; Kuypers, 1960). On the other hand in the cat these fibers terminate primarily in the dorsal and lateral parts of the internuncial zone (Szentagothai, 1941; Lloyd, 1941; Chambers and Liu, 1957). The long descending fibers from the brain stem in the monkey terminate almost exclusively in the internuncial zone (Kuypers, et. al., 1962). Generally speaking, these long fibers can be grouped into a lateral and medial pathways according to their spinal termination. The lateral pathway terminates primarily dorsally and laterally in the internuncial zone. The bulk of its fibers seems

* Supported by the U.S.P.H.S. Grant Nº B1131 (C5).

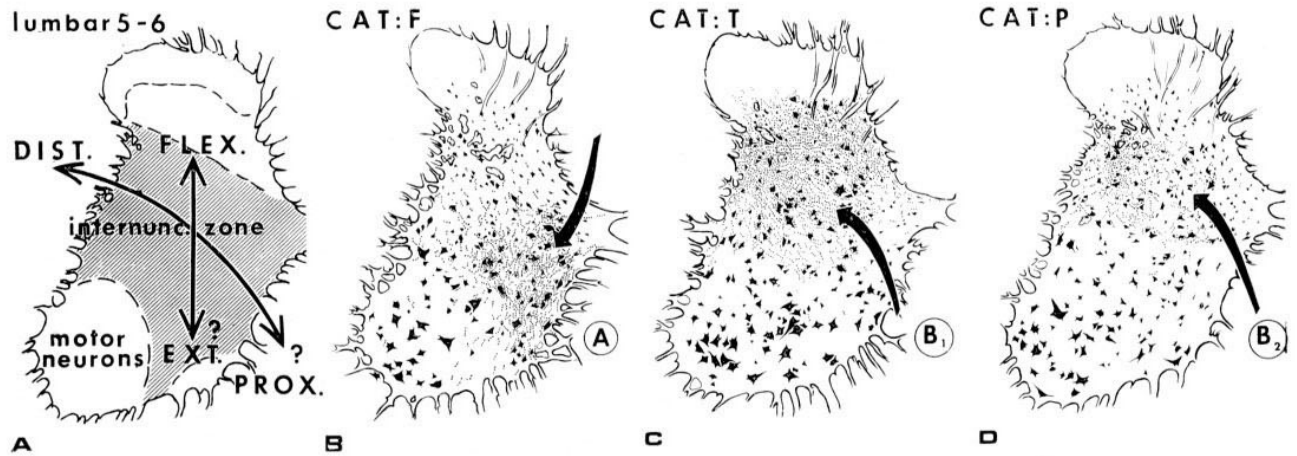


Fig. 1. — *a) Somatotopic organization of the internuncial zone. b) The spinal distribution of the degenerating fibers of the ventromedial subcorticalspinal pathway (System A) in the spinal gray of cat F. c) The distribution of the degenerating pyramidal fibers in the spinal gray of cat T (System B1). d) The distribution of the degenerating fibers of the lateral subcorticospinal pathway (System B2) in the spinal gray of cat T (lesion, as in fig. 9b).*

to orginate primarily in the red nucleus although some fibers may be derived from other sources (Crosby, et. al., 1962). The medial pathway terminates primarily ventrally and medially in the internuncial zone and originates primarily in the ponto-bulbar medial reticular formation, the vestibular complex and the interstitial nucleus of Cajal. A largely similar trend in the spinal termination of the descending fibers from the brain stem has been reported in the cat (Schimert, 1938; Lloyd, 1941; Szentagothai, 1941; Staal, 1961; Petras, 1963). Therefore in this very animal two fundamental motor systems might be distinguished according to their termination in the spinal cord: A) a medial system which originates in the brain stem (medial subcorticospinal pathway, Kuypers et al., 1962) and terminates medially and ventromedially in the internuncial zone, and B) a lateral system which consists of two components: B_1, the corticospinal pathway and B_2, the lateral subcorticospinal pathway, both of which terminate in the dorsal and the lateral parts of the internuncial zone. The organization of this zone, proposed above, suggests that the former system (A) influences most directly trunk and proximal extremity musculature including extremity extensors, whereas the latter system (B) influences most directly distal extremity musculature including extremity flexors. This hypothesis is somewhat similar to that brought forward by Evans and Ingram (1939) and seems to be supported by other findings (among others, Tower, 1935; Sprague & Chambers, 1953, 1954; Orioli & Mettler, 1956, 1957; Pompeiano & Brodal, 1957). It was tested physiologically in the cat. The two systems were interrupted separately at upper medullary levels. The impairments following these lesions were studied for 3 to 8 weeks. Subsequently the animals were sacrificed and the lesions as well as the distribution of the descending degenerating

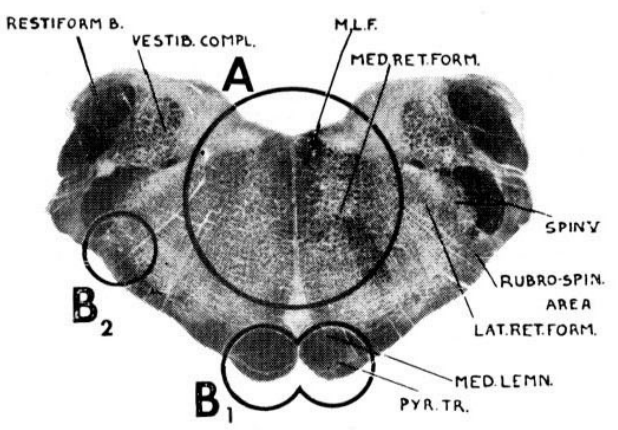


Fig. 2. — *The approximate location (A, B1, and B2) of the respective descending systems in the lower medulla oblongata of the cat.*

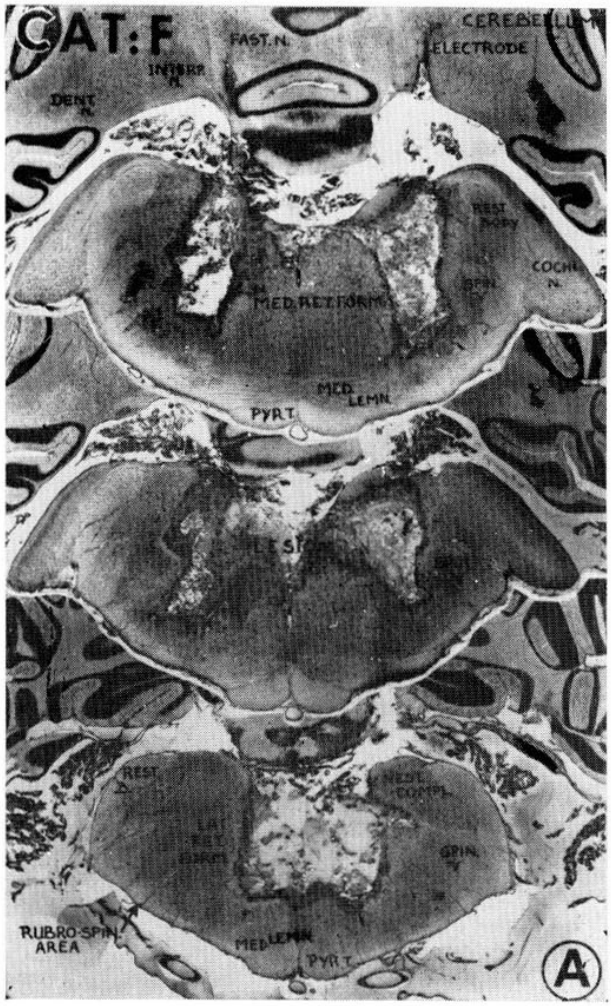


Fig. 3. — *Microphotographs of the medulla oblongata of cat F (Nissl stain). Electrolysis of the core of the medulla. The lesion extended slightly more rostrally than shown. Some involvement of the over laying parts of the cerebellar vermis occurred.*

fibers were studied anatomically by means of the Nauta-Gygax (1954) silver impregnation technique. The medial system (A) was interrupted most extensively in three cats, by electrolysis of the core of the brain stem at upper medullary levels. The medial longitudinal fasciculus, the medial reticular formation, and the lateral vestibulospinal tracts were interrupted bilaterally in these cases (fig. 3). The lateral systems (B) or either of its components (B1, B2) were interrupted as follow: a) The pyramidal tracts (B1) were transected either unilaterally or bilaterally in a total of five cats. In case of a unilateral transection, damage to the medial lemniscus was avoided as much as possible. b) The lateral subcorticospinal pathway (B2), which carries at least the rubrospinal fibers, was destroyed electrolytically in four cats at ponto-medullary levels (fig. 9 b). Some additional involvement of the lateral tegmentum, the spinal trigeminal complex, and the vestibular complex invariably occurred. c) A bilateral interruption of the pyramidal tracts (B1) was combined in three cats with a unilateral destruction of the rubrospinal area (B2) at ponto-medullary levels. Control lesions sparing the rubrospinal area were made in three cats. d) The bulk of the fibers of pathway B_2 apparently arises form the red nucleus. Therefore, the mesencephalic tegmental decussations including the rubrospinal one were interrupted in two cats by a midline cut, through the base of the skull (fig. 11). Later one pyramidal tract (B1) was also transected.

A. The animals in which the medial system (A) had been interrupted were alert but could not right themselves for several days. Nevertheless they used their extremities almost immediately (fig. 4) and began to move themselves about in this manner. The extremity withdrawal reactions were studied especially in the forelegs and were elicited by pin pricks or by pinching of the paws. Initially, withdrawal involved only distal flexion and hardly any shoulder pull was exerted (fig. 5 a, b). This impairment of the withdrawal was probably due to internuncial shock because it gradually diminished. Yet, the shoulder pull was still weaker than normal two to three weeks

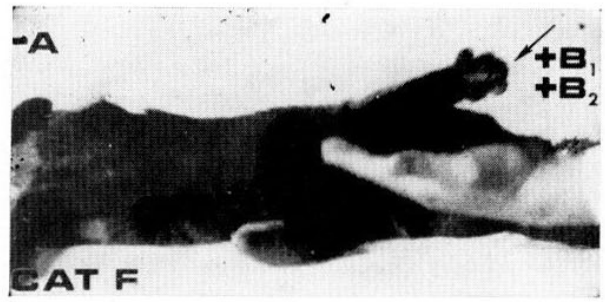


Fig. 4. — *Cat F, five days after electrolysis of the core of the medulla (involvement System A). Note: little tendency to right, but normal activity in the extremities (right foreleg). For the location of System A see fig. 2, for its termination see fig. 1B.*

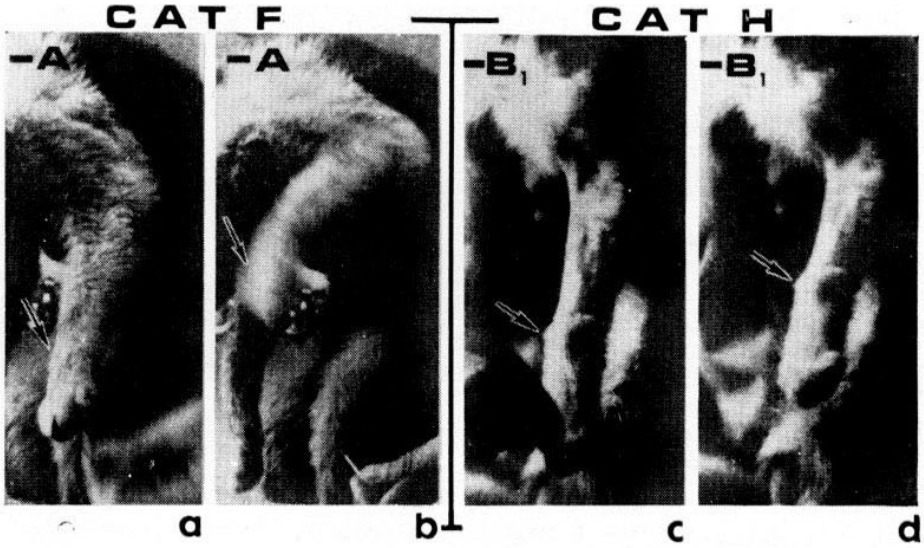


Fig. 5a and b) — *Foreleg withdrawal in cat F after electrolysis of the medulla (fig 3) (involvement of System A). Note the pronounced wrist and elbow flexion c and d) Foreleg withdrawal after pyramidotomy (involvement of System B1). Note the lack of wrist flexion and the limited elbow flexion.*

after surgery. At this time, the animals were "standing" but the trunk sagged between the scapulae as if only loosely suspended. The extremities, especially the hind legs, tended to display less extension than normal primarily in the proximal joints, (fig. 6 a), and the animals frequently assumed a crouching position. When progressing towards a goal, a severe trunk and neck ataxia occurred. However, despite these severe impairments, proximal in character, the animal began to hop and to place on contact and visually two or three days after surgery. After three weeks these extremity responses could be elicited almost as readily as in the normal animal (fig. 7). The animals could walk bars very well and without slipping, if the body was supported (fig. 8).

B. A totally different impairment, distal in character, was caused by the interruption of the lateral system (B) or either of its components (B1, B2). In these cases initially a distal weakness occurred most obvious in the wrist. The extremity withdrawal reactions were initially dominated by proximal movement (shoulder pull). Little elbow and hardly any wrist flexion occurred (fig. 5 c, d). This was probably due to internuncial shock as these movements gradually returned. Yet, the withdrawal flexion remained weaker than normal in many cases. These changes were more pronounced after transection of the corticospinal tract (B1) than after the destruction of the lateral subcorticospinal pathway (B2). This difference may be due to the greater number of fibers contained in the former tract than in the latter (fig. 1 c, d). In all cases hopping and placing were impaired, but primarily the latter were studied.

The bilateral interruption of the pyramidal tracts (B1), including parts of the medial lemniscus (fig. 9 a), initially abolished contact placing. Visual placing was either abolished or reduced to marching movements without distal flexion. However, a considerable recovery occurred. Two weeks after surgery both contact and visual placing again was executed with adequate flexion, although the threshold for eliciting

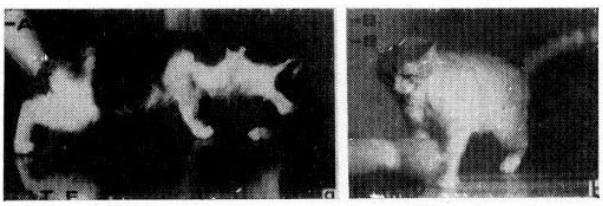


Fig. 6a and b). — *Illustration of the differences in posture after a) electrolysis of the core of the medulla (involvement of System A) in cat F, as compared to b) interruption of the mesencephalic tegmental decussations combined with pyramidotomy (involvement of System B1 and System B2) in cat V. Note that cat F (a) displayed less extremity extension than cat V (b).*

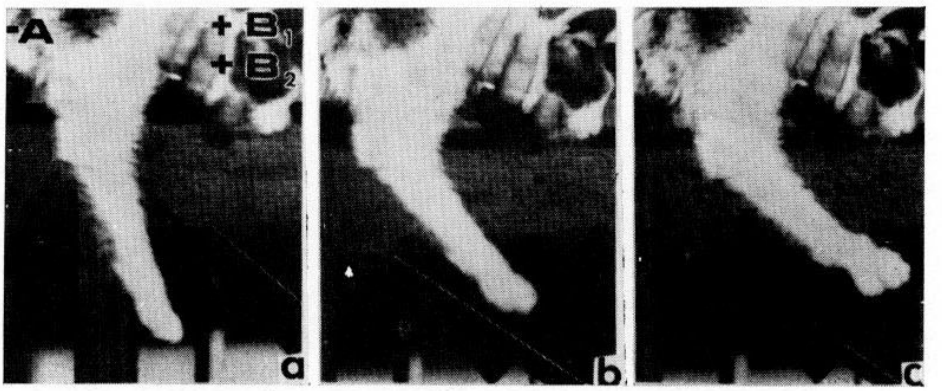


Fig. 7. — *Visual placing in cat F, fourteen days after electrolysis of the medulla oblongata (involvement of System A).*

these responses remained above normal (fig. 9 a, b, b'). Other impairments, more subtle in nature, could be noticed only after unilateral interruption of the pyramidal tract (B1). In these cases simultaneous tactile contact of the paws resulted only in placing of the non-affected extremity ($+B_1$, $+B_2$). Yet, the affected one ($-B_1$, $+B_2$) could place on contact with adequate flexion if the other was restrained. Furthermore, in reaching for the table while placing visually, the affected extremity ($-B_1$, $+B_2$) was held lower and placed later than the normal one (weaker elbow flexion). Finally, a preference for the non-affected extremity was exhibited during struggling, playing or fighting. Similar findings have been described by Tower (1935) but the placing reactions in her animals seemed to have been less vivid than in ours. In regard to the contact placing reactions, this difference might be due to a difference in the involvements of the medial lemniscus.

A somewhat similar but less severe impairment followed unilateral interruption of the lateral subcorticospinal pathway (B2) at ponto-medullary levels (e.g. fig. 9 b). During the first few days simultaneous contact of the forelegs either elicited no placing in the affected extremity ($+B_1$, $-B_2$) or placing was executed more slowly and involved less wrist flexion than in the nonaffected limb ($+B_1$, $+B_2$). In visual placing the affected extremity was held slightly lower (weaker elbow flexion?) and frequently touched the table. Moreover, visual placing in some instances seemed to be more readily elictable in the normal than in the affected extremity. A similar but bilateral impairment in extremity motility was noticed after interruption of the mesencephalic decussations. However, after both types of lesions an almost complete recovery

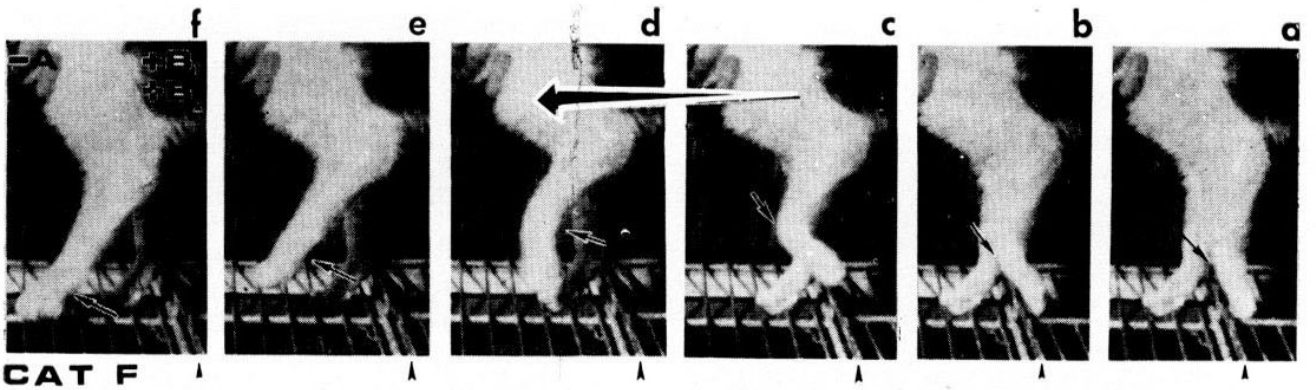


Fig. 8. — *Left foreleg movement in cat F, during bar walking. Progression from right to left. The body was supported. Note that the extremity motility seems totally unimpaired and that the right foot clutches the bar properly.*

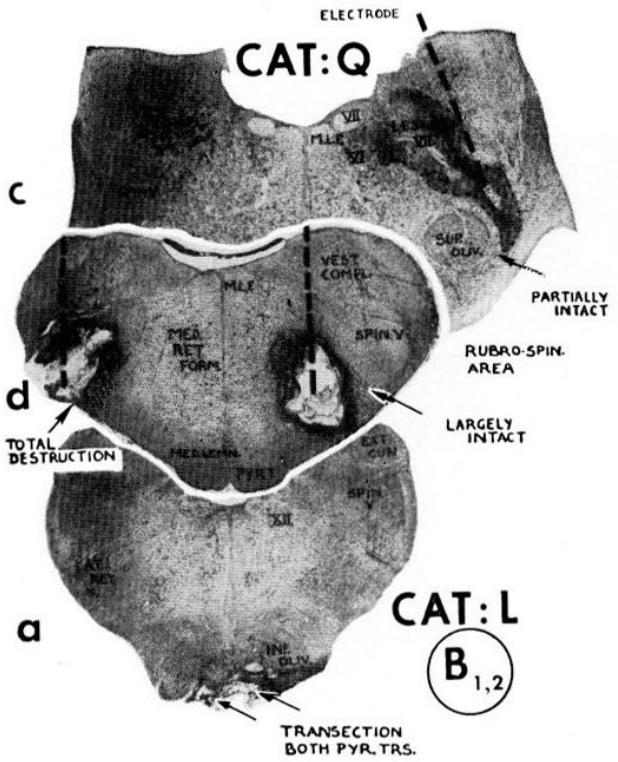


Fig. 9a and b). — *Microphotographs of the medulla oblongata of cat L (Nissl stain). Bilateral pyramidotomy a) was combined with two medullary lesions b). The left lesion destroyed the rubrospinal area, the right one largely spared it. Position of the electrodes indicated by dotted lines. Maximal impairment only in left extremities. c) medullary lesions in cat Q, destroying the lateral tegmentum but leaving the rubrospinal area partially intact. After additional bilateral pyramidotomy, a maximal impairment occurred, followed by an extensive recovery.*

occurred within 6 to 10 days (fig. 12 a). These findings seem to support those of Marshall (1934).

The impairment in extremity motility was aggravated by combined interruption of both components (B1 and B2) of the lateral system as achieved in either procedure c, (fig. 9 a, b) or procedure d, (fig. 11) and relatively little recovery ensued. When in these cases the less affected extremity (either $-B_1$, or $-B_2$) was not restrained, the maximally affected extremity ($-B_1$, $-B_2$) did not participated actively in visual placing (fig. 10 c, d; fig. 12 b, c) provided the animal was not overly excited. If the former was restrained, some visual "placing" occurred but the actual placing reaction was achieved primarily by proximal movement. In those cases in which contact placing was tested, the threshold was above normal and the placing movements tended to be proximal in nature involving little active distal flexion. The impairment of motility could also be demonstrated by placing the animal with the neck against the table (fig. 12 d). Under these circumstances the partially affected extremity (either $-B_1$ or $-B_2$) was placed by triple flexion but the maximally affected one ($-B_1$, $-B_2$) never achieved placing. Instead it made rhythmic movements of progression, which consisted of a limited flexion and extension in the elbow accompanied by alternating extension and flexion of the toes. However, hardly any wrist flexion occurred. The relative lack of extremity flexion could also be demonstrated by bringing the foreleg in extension onto the back. The partially affected extremity ($-B_2$) was brought back by triple flexion while only abortive attempts to triple flexion were noted in the maximally affected one ($-B_1$, $-B_2$). Yet, in this position little resistance was encountered in flexing this extremity passively. Finally, the maximally affected extremity ($-B_1$, $-B_2$) was far less agile than the less affected one (either $-B_1$ or B_2); and even when fighting in anger displayed a lack of flexion especially in the wrist (fig. 13 e, f, g). However, the impairment appeared to be a little less severe after procedure d, than after procedure e. The impairment persisted till death, in some instances 10 weeks after the second lesion.

The appearance of the full fledged impairment seems to depend at least upon the interruption of both the pyramidal tract and the fibers in the rubrospinal area at pontomedullary level. This thought emerged from the fact that full fledged impairment did not develop if a bilateral pyramidotomy was combined with a medullary lesion adjoining the rubrospinal area but largely sparing it (fig. 9 b). On the other hand, if the rubrospinal area was partially destroyed (fig. 9 c) a maximal impairment appeared, but a considerable recovery then followed. Interestingly enough the same holds true if a partial lesion of the py-

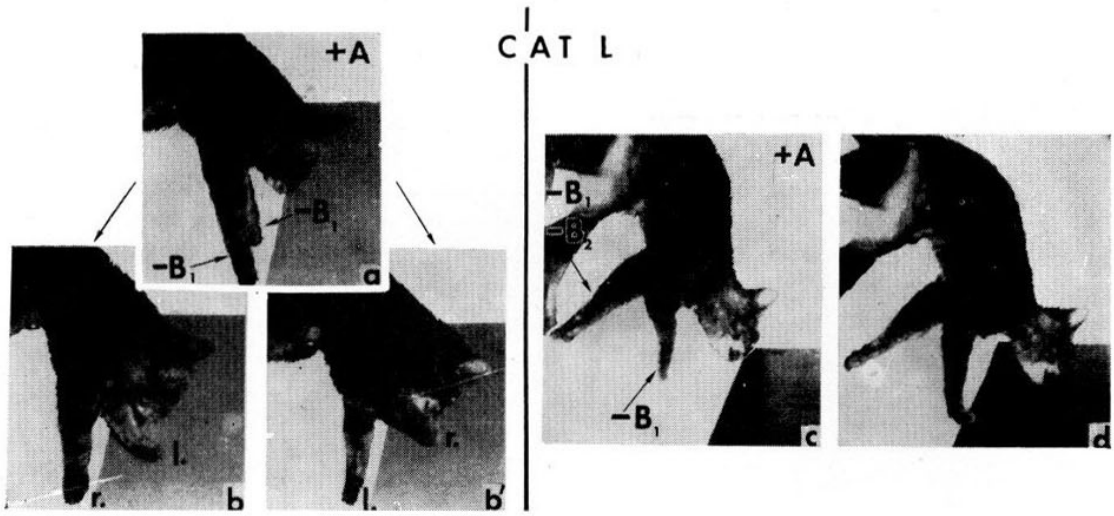


Fig. 10a, b and b'). — *Cat L, visual placing after bilateral pyramidotomy. Placing was achieved by both legs independently. c and d) Visual placing in cat L after additional medullary lesions (fig. 9b). The left leg did not participate actively.*

ramidal tract (B1) was combined with the total interruption of the rubrospinal area (B2). Therefore, the two components of the lateral system (B1, B2) to some extent seem to function vicariously and the motor recovery following the interruption of the one apparently depends on the presence of the other. Finally, maximal impairment also occurred either in case of a transection of the lateral funiculus at the first cervical segment or in case of a destruction of the rubrospinal area and the lateral tegmentum at the ponto-medullary levels, in combination with a transection of the corticospinal tract immediately distal to its decussation.

The present study is by no means exhaustive and a more detailed analysis undoubtedly is called for. Yet, the present findings seem to suggest that the two systems as defined anatomically truly represent two functionally different channels by which the brain has preferential access to two functionally distinct organizations in the muscular apparatus. Moreover, via these channels the brain seems to steer preferentially a) postural activities and progression and b) distal activities of the extremities respectively.

This concept of the organization of the motor system obviously abandons the classic pyramidal and extrapyramidal categories and in many respects is similar to that advocated by Bucy (1957). It is supported by the fact that both the motor cortex and the cerebellum seem to display a similar anatomical — physiological organization. For example, the cells of origin of the medial and the lateral "motor systems" of the present description receive fiber projections primarily from the medial and the lateral subdivisions of the cerebellum respectively (Jansen & Brodal, 1954; Cohen et al., 1958; Carpenter et al., 1958) which are functionally related to proximal and distal movements respectively (Chambers & Sprague, 1955). A similar topographical design can be detected in the organization of the motor cortex. Its rostral and caudal parts are functionally related to proximal and distal movements (Woolsey, 1958) and the fiber projections of these parts tend to be directed respectively to the medial and the lateral systems (Kuypers, 1958 a, 1960; Mettler, 1935).

The "motor" system in the Rhesus monkey is probably organized in a similar fashion. The subcorticospinal pathways terminate largely in the same manner as in the cat (Kuypers et al. 1962) and they apparently can maintain an important range of behavior in the absence of the cerebral cortex (Travis and Woolsey, 1956). On

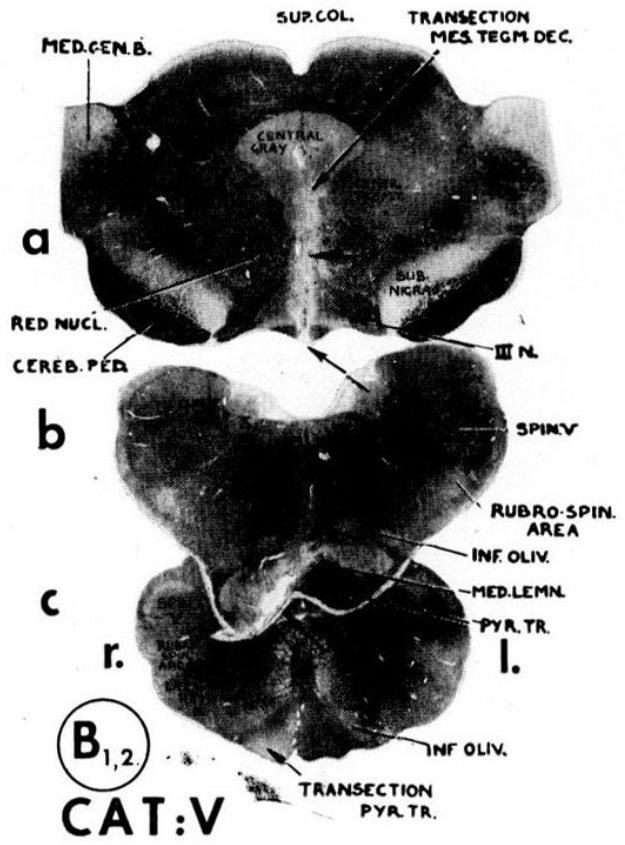


Fig. 11. — *Microphotography of brain stem sections of cat V (Weil stain) a) Cross sections through the mesencephalon. Note the transection of the mesencephalic tegmental decussations. b) Cross section through lower medulla. Note unilateral pyramidotomy involving parts of the medial lemniscus. c) Cross section through the caudal end of the medulla oblongata. Note demyelination of the affected pyramid.*

the other hand little is known in regard to the functional differences between the two subcorticospinal (A and B_2) pathways in this animal. Yet, the similarities between the present findings in the cat and those of Orioli and Mettler (1956, 1957) in the monkey suggest that at least the lateral subcorticospinal pathway (B_2) functions in both animals in a similar fashion. This is also supported by our findings in one Rhesus monkey which was ear marked for anatomical studies. When this animal was four months old, one corticospinal pathway (B_1) was almost totally interrupted. After one year, during which the animal regained almost normal motility, the lateral parts of the lower medulla were transected bilaterally including both lateral subcorticospinal pathways (B_2). Subsequently, the animals movements while climbing were only little impaired. Yet, it appeared very difficult for the animal by means of his maximally affected extremity ($-B_1$, $-B_2$) to take food held in front of him, whereas the partially affected extremity ($+B_1$, $-B_2$) was most agile in these circumstances. In retrospect this impairment seemed very similar to that observed in the cat following the combined interruption of both components of the lateral system (B_1, B_2).

Despite the similarities in the subcorticospinal connections in the cat and the monkey, the corticospinal connections are strikingly different. The cortical fibers in the cat (fig. 14 a) are distributed primarily

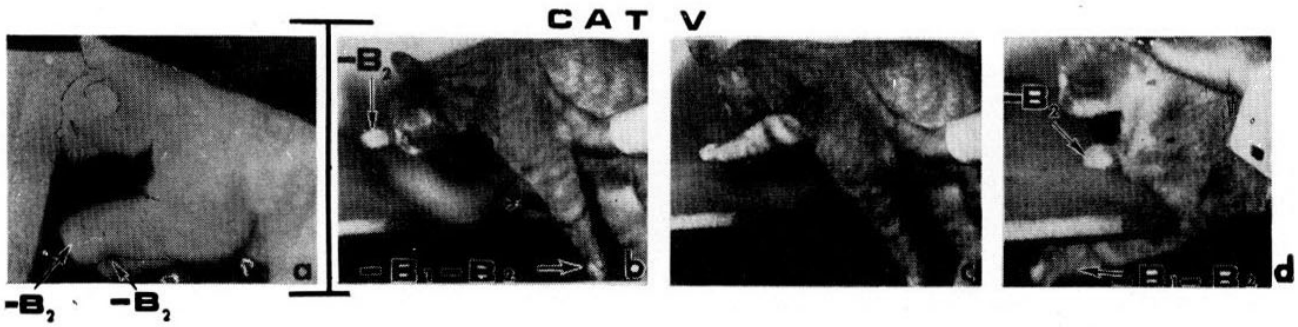


Fig. 12. — *a) Cat V, visual placing after transection of mesencephalic tegmental decussation (fig. 11a). Both legs were used. b and c) Visual placing of cat V after additional pyramidotomy on the right. Left leg did not participate actively. d) Same animal with chest against the table. The right leg achieved placing, the left did not.*

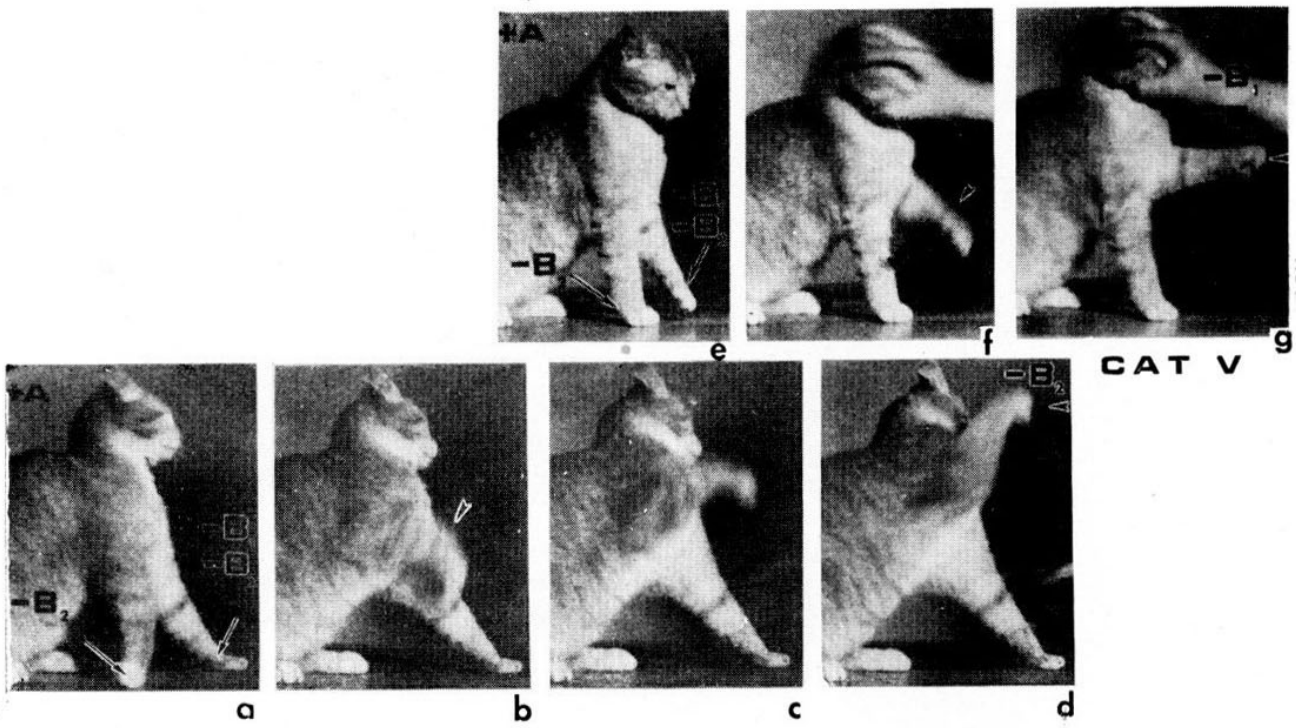


Fig. 13. — *Successive photographic frames of cat V, fighting in anger. Transection of the mesencephalic decussations (System B2) was combined with pyramidotomy (System B1) on the right. Right foreleg (a, b, c and d) had almost normal motility. Note the flexion in wrist and elbow. Left foreleg (e, f, and g) was impaired, it was elevated less than the right one and displayed less wrist flexion.*

to the dorsal and lateral parts of the internuncial zone and no fibers terminate on motor neurons. In the monkey (fig. 14 c) the fibers are distributed more extensively throughout the internuncial zone and terminate in part directly on motor neurons (Bernhard & Bohm, 1954; Chambers & Liu, 1958; Kuypers, 1960). These differences, especially in regard to the corticomotoneuronal connections, probably account for the greater severity of the motor impairments following the destruction of the corticospinal system in the monkey (Tower, 1935, 1940; Walker & Fulton, 1938). This is supported by the fact that in the individual Rhesus monkey such an impairment affects the distal musculature more severely than the proximal (Tower, 1940) and that the motor neurons of the former receive more cortical fibers than those of the latter (fig. 14 c). Moreover, the motor impairment after cortical lesions in the chimpanzee is apparently even more severe (Walker & Fulton, 1938) and the corticomotoneuronal fibers in this animal are considerably more numerous than in the Rhesus monkey (fig. 14 d). This suggests that the motor impairment to some extent is related to a loss of terminal boutons suffered by interneurons and motor neurons, which probably results in an increase in threshold of these affected cells. Such a bouton loss and the resulting increase in threshold seems to jeopardize motor function to a greater extent when affecting motoneurons rather than interneurons. The findings of Goldberger (1963) and those of the present study suggest that the recovery of motor functions after damage to the corticospinal system is dependent upon the subcorticospinal activity. The progressive return of movements (Travis, 1955) may be due to a subsequent progressive decrease in the heightened threshold of the affected spinal cells to a level at which some of the activity in the subcorticospinal pathways again can break through to the motor apparatus. This point of view is somewhat similar to that expressed by Lashley (1924, 1938) and Denny-Brown and Botterell

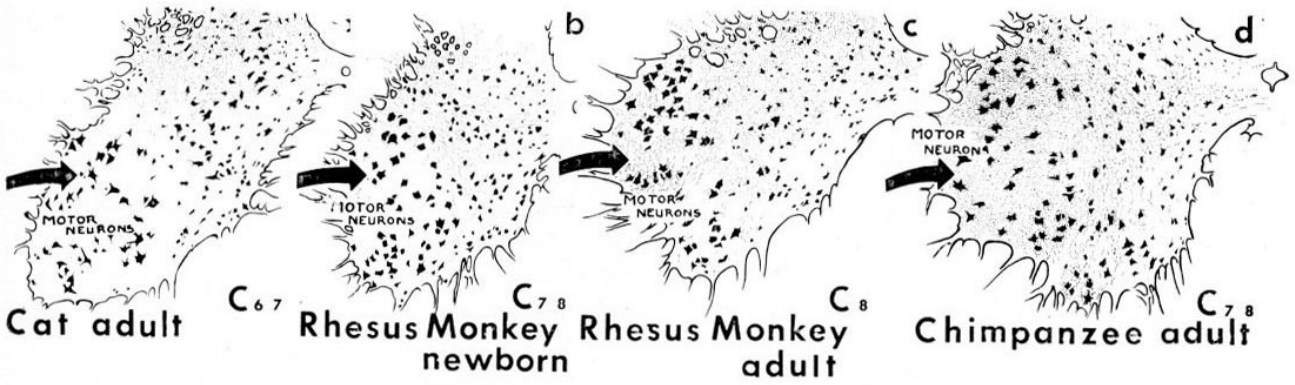


Fig. 14. — *Semidiagrammatic representation of the distribution of the degenerating cortical fibers in the spinal gray of low cervical segments a) in the adult cat after pyramidotomy, b) in the newborn and c) in the adult Rhesus monkey after large pre- and post-central cortical ablations and d) in the adult chimpanzee after a large cortical ablation involving primarily pre-central cortex (in part reprinted from Science, with permission). Note differences in the fiber distribution to the motoneuronal cell groups of the ventral horn.*

(1948). According to Denny-Brown and Botterell (1948) precentral lesions in the Rhesus monkey produce a general depression of motor function (pyramidal shock) involving most prominently distal motor activities. Yet, they observed that this depression can be largely overcome if enough motivation is created. The above interpretation of the substratum of the impairment in motility also agrees with our own findings in the newborn Rhesus monkey (Kuypers, 1962). In these animals (fig. 14 b) the bulk of the direct corticomotoneuronal connections apparently are not yet established, and the corticointerneuronal connections are far less numerous than in the adult. Therefore, large cortical lesions (areas FB, FBA, FA, PB, PC, PEm, PE, PF, Von Bonin & Bailey, 1947) presumably can cause only a limited loss in spinal boutons and an equally limited increase in threshold, largely confined to the interneurons. As a consequence, the subcorticospinal influences upon the motor apparatus presumably are only slightly suppressed. Correspondingly, these animals suffer considerably less motor impairment after such lesions and display a much speedier recovery than the adult, (Kennard, 1938, 1942). This was demonstrated to us most vividly by a four day old Rhesus monkey which climbed onto our hands by

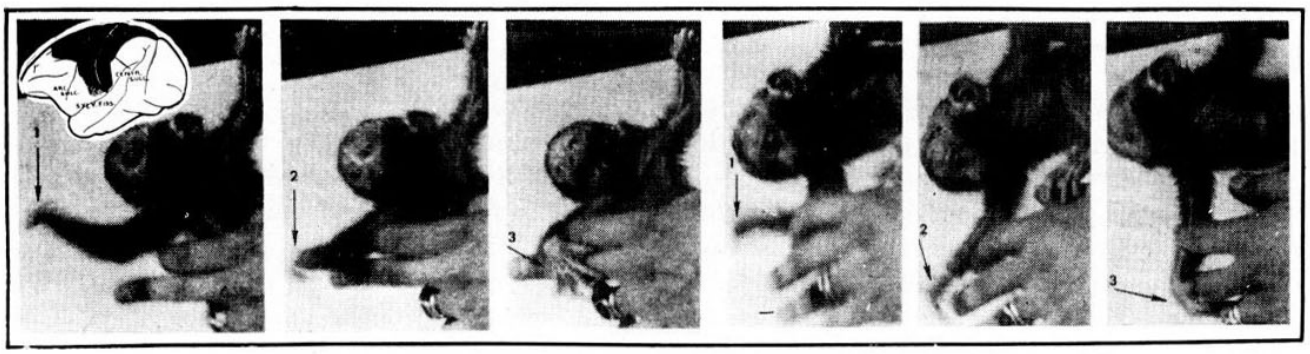


Fig. 15. — *Successive photographic frames of a four day old Rhesus monkey. Extensive ablation of the central cortical areas of the left hemisphere (see insert). Note the activity of the affected extremity.*

means of both its affected and its non-affected extremity (fig. 15) six hours after the removal of the above mentioned area.

The analysis presented here might hold for man as well, as suggested by the findings of Bucy (1957). On the other hand, the impairment resulting from the interruptions of the dorsolateral funiculus (B_1, B_2) in man (Putnam, 1940) seems to be more subject to recovery than in the cat. This might find its explanation in the fact that the lateral corticospinal tract in the cat is almost entirely confined to the dorsolateral funiculus whereas this tract in the chimpanzee of the present study was found to extend into the ventrolateral funiculus as suggested by the earlier findings (fig. 3) of Fulton & Sheehan (1935). Therefore, transection of the human dorsolateral funiculus as in the chimpanzee might interrupt only part of the lateral corticospinal tract. The impairment following interruption of the pyramidal tract or ablation of the motor cortex is presumably more severe in man than in the lower primates (e.g. compare Tower, 1940, Walker, 1952 and Brown and Fang, 1961, and compare Bucy, 1949 and Travis, 1955). This might reflect the presence of an even greater number of cortico-motoneuronal connections in man, a thought suggested by some of our findings of the hypoglossal nucleus (Kuypers, 1958 b, 1957 c). The subsequent recovery, albeit limited, would probably also depend upon subcorticospinal activity. Yet, it should be remarked that the rubrospinal fiber components of the lateral subcorticospinal systems may be less numerous in man than in other primates and their termination may be limited to cervical segments (Sie Pek Giok, 1956). If this is correct, then the lateral system (B_1, B_2) in man would be dominated to a much greater extend by corticospinal components (B_1) than in the cat or the monkey, a condition which also could account for the lesser degree of recovery.

SUMMARY

Two fundamental motor systems were distinguished in the cat, on the basis of the spinal termination of the descending pathways. A) The ventromedial system which arises from the brain stem and presumably influences primarily the trunk and the proximal extremity muscles and B) the lateral system, which arises from the cerebral cortex as well as from the brain stem and presumably influences primarily the distal extremity muscles. Correspondingly, the interruption of these systems appeared to result in disturbances of posture and in disturbances of the distal capacities of the extremities respectively. However, the motor impairment resulting from the destruction of the corticospinal pathway is less severe in the cat than in primates. This is probably related to the destruction of direct cortico-motoneuronal connections which exist only in the latter.

RESUMEN

En base a la terminación medular de las vías descendentes pueden distinguirse dos sistemas motores fundamentales en el gato: A) El sistema ventromedial de origen subcortical y que presumiblemente influye principalmente sobre los músculos del tronco y los músculos proximales de las extremidades, B) el sistema lateral, de origen cortical y subcortical que presumiblemente influye principalmente en los músculos distales de las extremidades. De acuerdo a esto, la interrupción de estos sistemas produce alteraciones de postura y alteraciones de las capacidades distales exploratorias y manipulatorias de las extremidades respectivamente. Sin embargo las alteraciones motoras que resultan de la destrucción de las vías cortico-espinales son menos severas en el gato que en los primates y esto se debe probablemente a la destrucción de las conexiones cortico-motoneuronales directas que sólo estos últimos poseen.

RÉSUMÉ

A cause de la terminaison spinale des voies descendantes, chez le chat deux systèmes moteurs fondamentaux sont distingués: A) le système ventromedial, d'origine souscorticale, qui probablement influence principalement les muscles du tronc et les muscles proximals des extrémités. B) le système lateral, d'origine corticale ainsi que souscorticale, qui probablement influence principalement les muscles distals des extremités. Correspondamment, l'interruption de ces systèmes produit respectivement des dérangements posturals et des dérangements d'activité distale des extremités. D'autre part, les dérangements motoriques suivant la destruction des voies corticospinales sont moins graves chez le chat que chez les primates. C'est probablement dû aux connexions cortico-motoneuronales directes, qui existent seulement chez les derniers.

ZUSAMMENFASSUNG

Auf Grund der Endungsweise der im Rueckenmark absteigenden Bahnen werden bei der Katze zwei fundamentale motorische Systeme unterschieden: A) das ventromediale System, welches aus dem Hirnstamm entspringt und vermutlich den Rumpf und die proximalen Extremitaetenmuskeln hauptsaechlich beeinflusst und B) das laterale System, das sowohl aus der Hirnrinde als auch aus dem Hirnstamm entspringt und wahrscheinlich vorwiegend die distalen Extremitaetenmusklen beeinflusst. Dementsprechend brachte die Unterbrechung dieser Systeme hinsichtlich der Stoerungen in der Haltung und in den distalen Faehigkeiten der Extremitaeten hervor. Die von Unterbrechung der cortico-spinalen Bahnen hervorgerufenen motorischen Stoerungen sind aber bedeutend leichter bei der Katze als bei den Primaten. Das Vorliegen von direkten corticomotoneuronalen Verbindungen in Primaten ist wahrscheinlich dafuer verantwortlich.

REFERENCES

Bernhard, C. G. and E. Bohm. 1954: Cortical representation and functional significance of the cortico-motoneuronal system. Arch. Neurol. Psychiat., Chicago 72: 473.

Bernhard, C. G. and B. Rexed. 1945: The localization of the premotor interneurons discharging through the peroneal nerve. J. Neurophysiol., 8: 387.

Brown, W. J. and H. C. H. Fang. 1961: Spastic hemiplegia in man. Lack of flacidity in lesion of pyramidal tract. Neurol., 11: 829.

Bucy, P. C. 1949: Effects extirpation in man. The precentral motor cortex: 353, University of Illinois Press, Urbana, Illinois.

Bucy, P. C. 1957: Is there a pyramidal tract? Brain, 80: 376.

Cajal, R. S. 1955: Histologie du système nerveux de l'homme et des vertébrés. Instituto Ramón y Cajal, Madrid.

Carpenter, M. B., G. M. Brittin and J. Pines. 1958: Isolated lesions of the fastigial nuclei in the cat. J. Comp. Neurol., 109: 65.

Chambers, W. W. and C. N. Liu. 1957: Cortico-spinal tract of the cat. An attempt to correlate the pattern of degeneration with the deficits in reflex activity folllowing neocortical lesions. J. Comp. Neurol. 108: 23.

Chambers, W. W. and C. N. Liu. 1958: Cortico-spinal tract in monkey. Fed. Proc., 17: 24.

Chambers, W. W. and J. M. Sprague. 1955: Functional localization in the cerebellum. Arch. Neurol. Psychiat., Chicago 74: 653.

Cohen, D., W. W. Chambers and J. M. Sprague. 1958: Experimental study of the efferent projection from the cerebellar nuclei to the brain stem of the cat. J. Comp. Neurol., 109: 233.

Crosby, E. C., T. Humphrey and E. W. Lauer. 1962: Correlative Anatomy of the nervous system. The MacMillan Company, New York.

Denny-Brown, D. and E. H. Botterell. 1948: The motor functions of the agranular frontal cortex. Res. Publ. Ass. nerv. ment. Dis., 27: 235.

Evans, B. H. and W. R. Ingram. 1939: The effect of combined red nucleus and pyramidal lesions in cats. J. Comp. Neurol., 70: 461.

Fulton, J. F. and D. Sheehan. 1935: The uncrossed pyramidal tract in higher primates. J. Anat., Lond., 69: 181.

Goldberger, M. E. 1963: Effects of combined pyramidal and extrapyramidal lesions in the macaque. Anat. Rec., 145: 232.

Jansen, J. and A. Brodal. 1954: Aspects of ecerebellar anatomy. Tanum, Oslo.

Kennard, M. A. 1958: Reorganization of motor function in the cerebral cortex of monkey deprived of motor and premotor areas in infancy. J. Neurophysiol., 1: 477.

Kennard, M. A. 1942: Cortical reorganization of motor function. Studies on series of monkeys of various ages from infancy to maturity. Arch. Neurol. Psychiat., Chicago 48: 227.

Kuypers, H. G. J. M. 1958 a: An anatomical analysis of cortico-bulbar connections to the pons and the lower brain stem in the cat. J. Anat., Lond., 92: 198.

Kuypers, H. G. J. M. 1958 b: Cortico-bulbar connections from the pericentral cortex to the pons and lower brain stem in monkey and chimpanzee. J. Comp. Neurol., 110: 221.

Kuypers, H. G. J. M. 1958 c: Cortico-bulbar connections to the pons and lower brain stem in man. Brain, 81: 364.

Kuypers, H. G. J. M. 1960: Central cortical projections to motor and somato-sensory cell groups. Brain, 83: 161.

Kuypers, H. G. J. M. 1962: Cortico-spinal connections: postnatal development in the Rhesus monkey. Science, 138: 678.

Kuypers, H. G. J. M., W. R. Fleming and J. W. Farinholt. 1962: Subcortico-spinal projections in the Rhesus monkey. J. Comp. Neurol., 118: 107.

Lashley, K. S. 1924: Studies of cerebral function in learning. Arch. Neurol. Psychiat., Chicago, 12: 249.

Lashley, K. S. 1938: Factors limiting recovery after central nervous lesions. J. nerv. ment. Dis., 88: 733.

Lloyd, D. P. C. 1941: Activity in neurons of the bulbo-spinal correlation system. J. Neurophysiol., 4: 115.

Lloyd, D. P. C. 1941: The spinal mechanism of the pyramidal system in cats. J. Neurophysiol., 4: 525.

Lloyd, D. P. C. 1944: Functional organization of the spinal cord. Physiol. Rev., 24: 1.

Marshall, C. 1934: On certain midbrain lesions in cat. Anat. Rec., 58, Suppl.: 26.

Mettler, F. A. 1935: Corticifugal fiber connection of the cortex of the macaca mulatta. The frontal region. J. Comp. Neurol., 61: 509.

Nauta, W. J. H. and P. A. Gygax. 1954: Silver impregnation of degenerating axons in the central nervous system. A modified technic. Stain Tech., 29: 91.

Orioli, F. L. and F. A. Mettler. 1956: The rubro-spinal tract in macaca mulatta. J. Comp. Neurol., 106: 299.

Orioli, F. L. and F. A. Mettler. 1957: Effects of rubro-spinal tract section on ataxia. J. Comp. Neurol., 107: 305.

Petras, J. M. 1963: The descending pathways and terminal distribution of cortical, tectal and tegmental fibers to the spinal cord of the cat. Anat. Rec., 145: 171.

Pompeiano, O. and A. Brodal. 1957: Experimental demonstration of a somatotopic origin of rubro-spinal fibers in the cat. J. Comp. Neurol., 108: 225.

Putnam, T. J. 1940: Treatment of unilateral paralysis agitans by section of the lateral pyramidal tract. Arch. Neurol. Psychiat., Chicago, 44: 950.

Romanes, G. J. 1951: The motor cell columns of the lumbosacral spinal cord of the cat. J. Comp. Neurol., 94: 313.

Schimert, J. 1938: Die Endigungsweise des Tractus vestibulo-spinalis. Z. Anat. Entw-Gesch. 108: 761.

Sie Pek Giok. 1956: Localization of fiber systems within the white matter of the medulla oblongata and the cervical cord in man. (Thesis, University of Leyden) Ydo, Leiden.

Sprague, J. M. 1948: A study of motor cell localization in the spinal cord of the Rhesus monkey. Amer. J. Anat., 82: 1.

Sprague, J. M. 1951: Motor and propriospinal cells in the thoracic and lumbar ventral horn of the Rhesus monkey. J. Comp. Neurol., 95: 103.

Sprague, J. M. and W. W. Chambers. 1953: Regulation of posture in intact and decerebrate cat: I cerebellum, reticular formation, vestibular nuclei. J. Neurophysiol., 16: 451.

Sprague, J. M. and W. W. Chambers. 1954: Control of posture by reticular formation and cerebellum in the intact anesthetized and unanesthetized and in the decerebrated cat. Am. J. Physiol., 176: 52.

Staal, A. 1961: Subcortical projections on the spinal gray matter. (Thesis, University of Leyden), Koninkelijke Drukkerijen Lankhout-Immig N. V., The Hague.

Szentagothai, J. 1941: Die Endigungsweise der absteigenden Rücken markes Bahnen. Z. Anat. EntwGesch., 11: 322.

Tirvik, A. and A. Brodal. 1957: The origin of the reticulo-spinal fibers in the cat. An experimental study. Anat. Rec., 128: 113.

Tower, S. S. 1935: The dissociation of cortical excitation from cortical inhibition by pyramidal section and the syndrome of that lesion in the cat. Brain, 58: 238.

Tower, S. S. 1940: Pyramidal lesions in the monkey. Brain, 63: 36.

Travis, A. M. 1955: Neurological deficiencies after ablation of the precentral motor area in macaca mulatta. Brain, 78: 155.

Travis, A. M. and C. N. Woolsey. 1956: Motor performance of monkeys after bilateral partial and total cerebral decortication. Amer. J. Phys. Med., 35: 273.

Von Bonin, G. and P. Bailey. 1947: The neocortex of macaca mulatta. The University of Illinois Press, Urbana Illinois.

Woolsey, C. N. 1958: Organization of somatic sensory and motor areas of the cerebral cortex. Biological and biochemical basis of behaviors. Wisconsin University Press, Madison.

Walker, A. E. 1952: Cerebral pedunculotomy for relief of involuntary movement. II Parkinson Tremor. J. nerv. ment. Dis., 116: 766.

Walker, A. E. and J. F. Fulton. 1938: Hemidecortication in chimpanzee baboon, macaque, potto, cat and coati. A study in encephalization. J. nerv. ment. Dis., 87: 677.

The Significance of Complex motor Patterns in the Response to Cortical stimulation

J. A. V. BATES

National Hospital - London

Whoever applies a stimulating electrode to the precentral gyrus and other so-called motor regions of the cortex will observe a series of responses which can range from the most discrete and isolated muscular movements to complex movements which must simultaneously involve many different muscle groups. Those responses at one end of the spectrum, so to speak, are highly discrete and include some which, as Sherrington (1906) stressed, are more "fractional" than the animal can and does ever perform, such as a movement of one vocal cord or movement of the anus to one side, and the same is true in man. But on the other hand, the same stimulating electrode on the same cortex at the same strength of stimulus may evoke, from a nearby point, practically simultaneous movements at several joints. Leyton and Sherrington (1917) reported three or four separate movements from many of the points on the anthropoid cortex, although they were stimulating in the most punctate way they could devise and for the shortest time necessary to get some response. They noticed that occasionally the particular combination would form a synergism which was recognizable as one that occurred in the animal. For example, in relation to the finger of Chimpanzee: "A not infrequent response from cortex when the resting posture happened to be one with adducted thumb and semiflexed index was simultaneous abduction of thumb and straightening of index as if to let go an object that had been picked up". Attention has previously been called to the fact (Bates 1957) that if the stimulator is left on for a second or two longer than that required to evoke the first signs of movement, the wrist and finger sometimes assume postures that one can recognize as a feature of the behaviour of the normal hand of the normal adult.

For example one can occasionally recognize the extended thumb and index combined with the flexed outer fingers which is the pointing gesture; or the tightly flexed fingers with the tip of the thumb appearing between the third and fourth fingers which is the 'manus obscena', a posture of the fingers assumed involuntarily by perhaps 5 % of adults, and apparently showing a familiar tendency (Wood Jones, 1941). Ward (1938), in observations on the unrestrained cat, pointed out that when the stimulus to the motor cortex was prolonged beyond the time necessary to produce the first trace of movement, the limb came to assume a posture which seemed to be characteristic of each cortical point, save that to some extent it was modifiable by

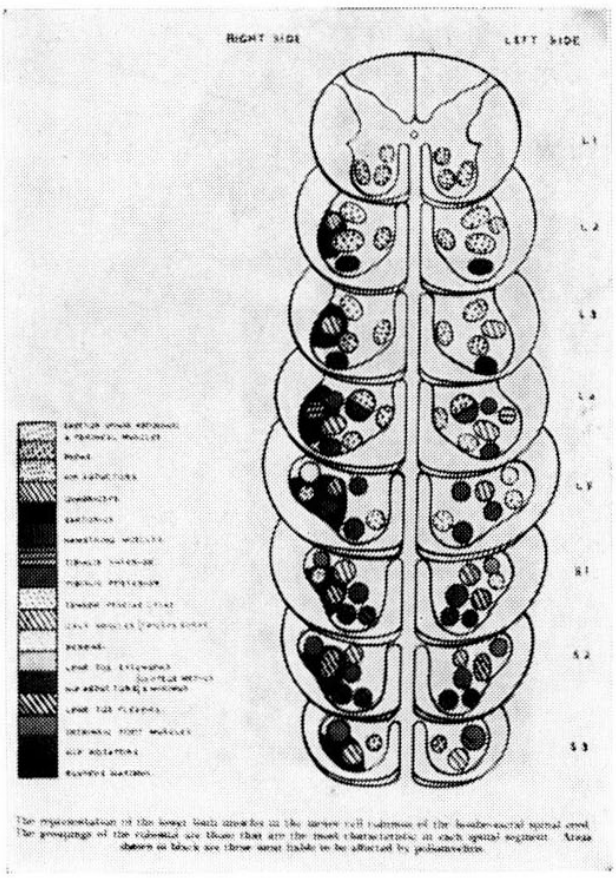


Fig. 1. — *Findings of Sharrard. Location of cell bodies in the lumbar enlargement whose axons run to particular muscles of the lower limb.*

the tonic neck reflex. It would be generally true to say, however, that for most observers and most responses the particular movements forming a complex sequence in time and leading to an involved posture have no obvious correspondence to those we can recognize as occurring in life. And because the combinations of movement are superficially meaningless and difficult to analyse and describe, they have had little attention, compared with the attention given to the topographical arrangement from mouth to toe which is a fact that can be more easily described and appreciated.

In considering the significance and interpretation of the movements evoked by cortical stimulation, Sherrington wrote in 1898: "Flexion adduction of the thumb (in a monkey) though instanced as an action of peculiarly cortical nature, is really the most frequent and facile pure spinal reflex of the upper extremity" and in more general terms in the Integrative Action of the Nervous System (1906) "The local reflex movements obtainable from the bulbospinal animal and the reactions elicitable from the motor cortex of the narcotized animal fall into line on similar series. Both consist of the same group". These observations encouraged one to assemble some of the indirect evidence from man which seemed to lend support to the idea that in interpreting the movements provoked by cortical stimulation there is no necessity to postulate the activation of a motor organisation at a cortical level; rather that one is exciting directly the same arrangements of motor neurones in the bulb and spinal cord that one can excite by stimulation of the periphery. There are three pieces of indirect supporting evidence and one piece of more direct evidence. In summary they are as follows:

1. In the first place, consistent with Sherrington's observations just quoted on isolated movements of the thumb as a facile and pure spinal reflex, it is clear that very discrete thumb and finger movements occur not only in the new born, but also in the anencephalic human infant. Isolated movements of the fingers can also be seen in the famous infant, filmed by Gamper (1926), with no development of the C.N.S. above the red nucleus.

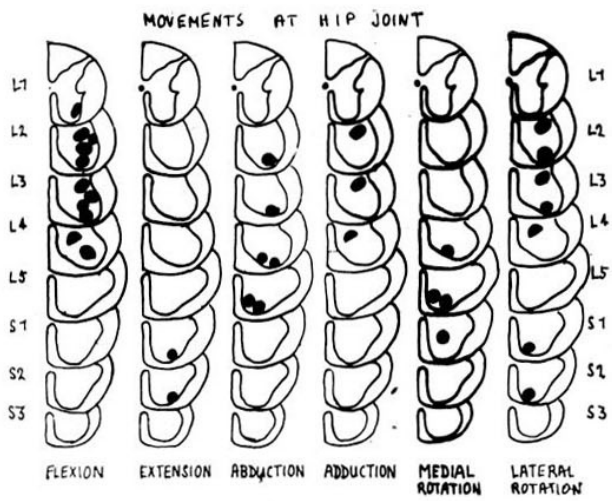


Fig. 2. — *Location of cell bodies in the lumbar enlargement related to movement at hip joint.*

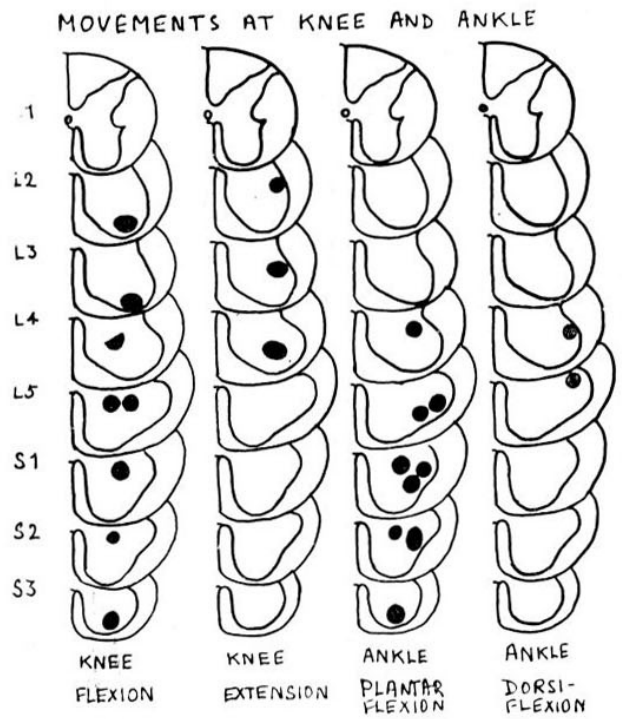


Fig. 3. — *Location of cell bodies related to movements at knee and ankle.*

2. In the second place, complementary to Sherrington's view, when stimulating the medial surface of the normal hemisphere after hemispherectomy, it was possible to identify in two cases a complex movement as a typical crossed extensor response in the ankle and toes (Bates 1953) — this is a response which is commonly regarded as a spinal reflex to the sole.

3. In the third place, as first pointed out by Burdon Sanderson (1874) in the cat, it was apparent (Bates 1953) that movements evoked by stimulating the descending fibres in the corona radiata and internal capsule after hemispherectomy are quite as discrete as those evoked by stimulation of the cortex.

The last and more direct piece of evidence has been obtained during stereotactic operations when a movement of the toes and inversion of the ankle from stimulation of the capsule was identical to that obtained by stroking the sole of the foot.

These then are the kind of observations which can be made on man and suggest that from the evidence of stimulation there is no necessity to postulate a motor organisation at a cortical level. Whether the evidence of ablation should lead to belief in cortical motor centres is another matter, but I prefer to view the consequences of ablation as a manifestation of Sherrington's 'Isolation Dystrophy'; a depression of spinal motor function, to use Denny Brown's phrase; or as perhaps Bremer would agree, a state of altered spinal motor function consequent on the loss of the 'Tonus corticale'.

Now although one may postulate spinal motor mechanisms that are excited by stimulation or depressed by denervation, the general notion has always seemed rather vague in terms of the known facts of anatomy and physiology of the spinal grey matter. From experimental neurophysiology we know that the motoneurones of the spinal cord can be made to fire rhythmically by the application of direct currents to the soma; and we know that during reflex activity there are slow potential changes in the grey matter associated with rhythmic firing. We know also that electrical fields can be recorded at points of convergence of inflow from the sensory roots and from the higher centres, and it is reasonable to presume that the detailed shape of these fields in three dimensions will determine the de-

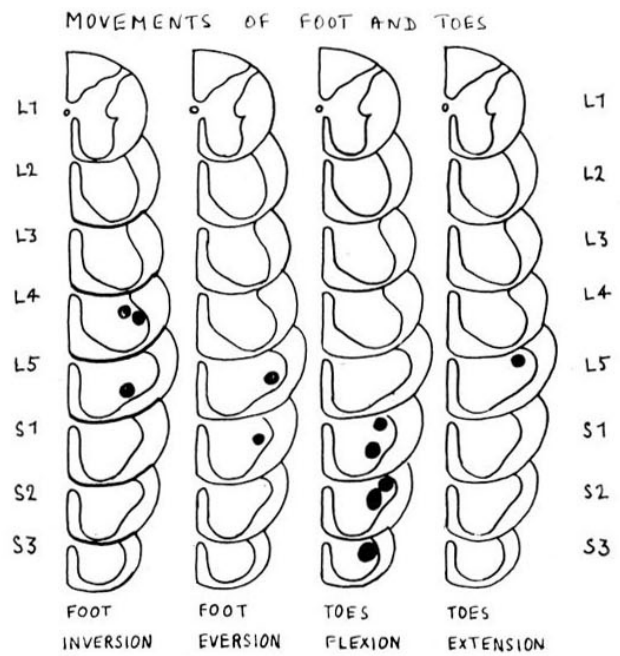


Fig. 4. — *Location of cell bodies related to movements of foot and toes.*

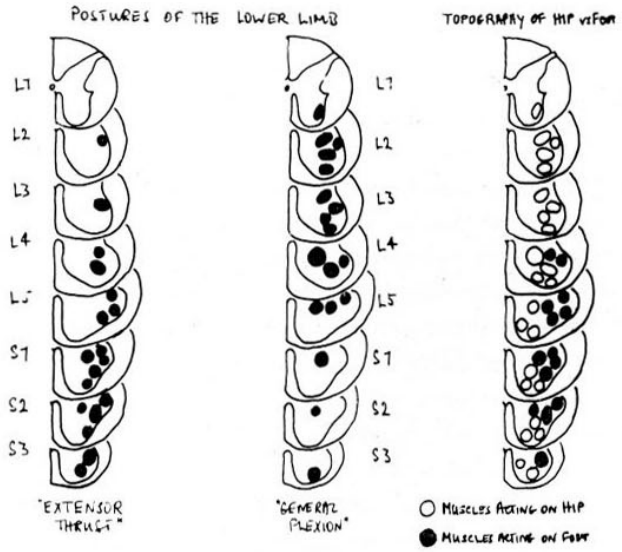


Fig. 5. — *For opposing postures of the lower limb, namely flexion and full extension at the leg, two separate regions of the cord are involved.*

tails of excitation and inhibition in motor units by the way these potential fields impinge on the patterned arrangement of motor cell groupings. It is, however, on the face of it unlikely that the study of cell body groupings will fully reveal the functional pattern. The cell body itself provides only a small part of the total area of the neurone — Lorente de No (1953) puts it at only 6% and it is likely that the full clue to the organisation lies in the relation of the axonal and dendritic plexuses. These are, however, technically beyond our powers to unravel at the moment.

However, it has seemed worthwhile to examine a simple hypothesis. A pattern of movement implies an ordered sequence of activity in different motor cell groups in the grey matter of the spinal cord. One asks — is the spread of activity from one muscle to another or the practically simultaneous involvement of a number of muscles consistent with the view that activity in the cord in spreading outwards from a focus like ripples from a stone in water. In other words, is the particular sequence which goes to form the complex response a sequence which you would expect from the known facts of grouping of cell bodies whose axons innervate the muscles responsible for the particular sequence. For example, if one were to observe medial rotation at the hip followed by flexion at the knee, one would ask are the cell bodies whose axons innervate respectively the medial rotator muscles and knee flexor muscles grouped in directly adjacent masses in the cord — if they were not adjacent, if the anatomical data showed cells in a group between them which were apparently not active, (say for example causing flexion of the hip which did not occur), this hypothesis would be defeated.

To examine this one can call on three independent sets of data already published quite independently. The first group of data contains a set of responses to stimulation of the medial surface of the intact normal hemisphere exposed by cutting a window in the falx after hemispherectomy. This data was published nine years ago (Bates 1953) and fourteen responses from seven subjects were filmed and analysed with more detail-assurance than is usually possible by continuous projections of a loop. The separate movements were indicated by a code, thus: $T_{(1-5)}.F \rightarrow A.PF \rightarrow A.DF \rightarrow K.E$ means that there was flexion of all the toes, followed by plantar flexion of the ankle joint, followed by dorsiflexion of the ankle joint; followed by extension of the knee joint.

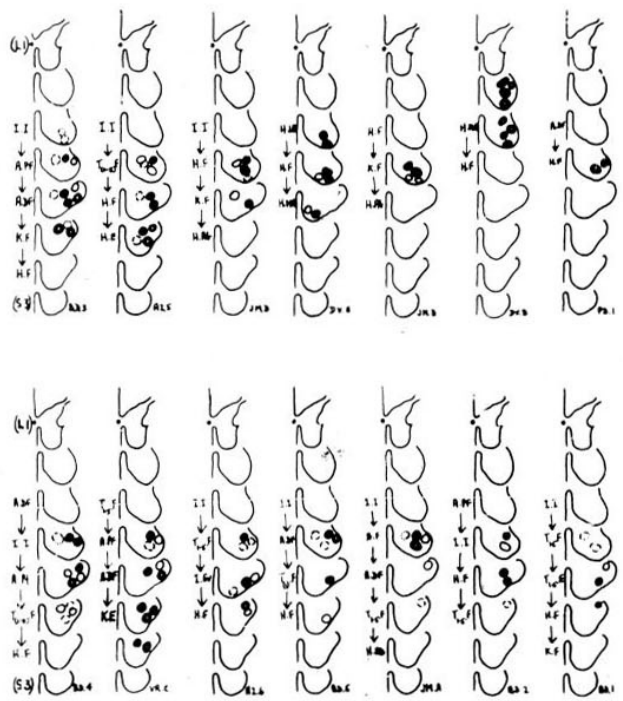


Fig. 6. — *The zones of activity appropriate to each movement in the order in which movements have been observed are superimposed in this picture.*

The second set is the data in Grey's Anatomy from which we can list all the muscles which cause the different movements about the joints of the lower limb. Thus the above sequence would read:

$T_{(1\text{-}5)}.F \rightarrow$ Flexor hallucis longus and Flexor Digitorum longus

$A.Pf \rightarrow$ Gastrocnemius and Soleus

$A.Df \rightarrow$ Tibialis Anterior and Extensor Digitorum longus

$K.E \rightarrow$ Quadriceps and Rectus Femorus

The third set of data comes from the painstaking work of Sharrard (1955). He published some definite information of the location of certain motor cells in the lumbar enlargement whose axons run to particular muscles of the lower limb. Sharrard obtained the spinal cords of seven selected individuals who died between three months and eight years after the onset of poliomyelitis and in whom the level of paralysis in different muscles had been estimated by clinical examination. The sites of cell loss in the diseased cords were found by comparison with the groupings in normal cords which he prepared, and using the clinical data he could reconstruct the location of the cell bodies whose axons run to particular muscles. His findings are summarized in fig. 1. The next step was to combine the data from Sharrard's work with that in Grey's Anatomy to produce figures illustrating the regions of the grey matter in the cord that must contain active motor neurones when the particular movements observed in response to cortical stimulation are occurring. The results for fourteen separate movements of the lower limb are shown in figs. 2, 3, 4.

Before applying this data to examine the hypothesis it is interesting to observe that if the individual movements at all the joints are combined for each of the two characteristic and opposing postures of the lower limb, namely flexion, and full extension of the leg as it prepares to take the full weight of the body, then one can see that two separate regions of the cord are involved (fig. 5). The functional grouping implies a wave-like ascending and descending field of excitation in time with each step.

All that now remains to test the hypothesis is to superimpose the zones of activity appropriate to each movement in the order in which the movements have been observed to occur, and to indicate them appropriately. This has been done in fig. 6. For this figure the active zones in L 3, 4, 5, and S. 1. only are considered as they are in all cases sufficient to account for the movements. In view of the arrangement of many of the groups in longitudinal columns, the information from L 1. and 2. and S.2 and 3 does not help to examine the hypothesis and makes the figures more confusing. Reading the figure from left to right, the sequences in the top row and the first three of the bottom row could be in accord with the hypothesis. In the case of the remaining four in the bottom row, the hypothesis by itself would appear to be insufficient to account for all the sequences though in three of the four it accounts well enough for the first three movements.

It is necessary to ask whether the observations could fit the hypothesis equally well by the operation of a random process. In other words, do the facts from Grey's Anatomy and Sherrard's data allow such latitude that *any* sequence of movements could be accounted for by assuming a tendency for active cells to activate their neighbours. This possibility can be examined by a statistical test in the following way. The fourteen observed responses to stimulation (containing a total of 44 sequences of movement) have been matched with an imaginary series of 100 responses (containing 320 sequences of movement) by the use of a table of random numbers and a list of the movements observed and their relative frequencies. The details are given in the appendix. Ten of the fourteen observed responses could have occurred according to the hypothesis of adjacent interaction. Twenty of the hundred in the imaginary series could be accounted for. A χ^2 test shows this difference to be significant. In other words a purely random process is very unlikely to give the actual amount of agreement that was observed.

To go further along the same lines in man seems at the moment difficult because although there is a far richer variety of

movement complexes in the upper limb, we have no corresponding reliable data for the positions in each segment of the motor cells supplying the different muscles. This hypothesis, however, may have predictive value. Because, for example, we have observed the posture outstretched thumb and index and flexed outer fingers, one would predict that the cells for the extensor indicis and the flexor digitorum sublimis occupy adjacent stations in the grey matter of C.7., perhaps one day it will be possible to discover if this is so. It is probable, however, that the quickest way to proceed will be on lower animals. In the case of the cat, we have some definite data on motor cell grouping from the study of Romanes (1951), who discussed its significance in conjunction with data from other mammals (1953). He concludes that the location "represents, in part, the morphological division of the muscles, but also is of topographical significance in relation to the joints moved... The functional significance of this plan remains obscure in the absence of detailed information concerning... the patterns of muscular activity which take place during normal movements of a limb". Unfortunately the repertoire of movements of the lower limb from cortical stimulation in the cat is somewhat limited, but it would seem worth while to explore, with electromyography, the sequence of muscular activity in the response in detail in this mammal in the light of Romanes' data in the belief that the sequence of movements from activation of a single point will be consistent with the topographical arrangement of cells.

SUMMARY

It is argued that in considering the significance of movements produced by cortical stimulation, it seems unnecessary to postulate the existence of motor centres in the cortical grey matter in the sense of neuronal organisations similar to, but distinct from, those which must be present in the spinal grey matter. The reasons are, in the first place there is a close similarity between the movements in response to stimulation of the cortex and stimulation of the internal capsule after hemispherectomy; secondly, although stimulation can produce discrete movement of a single finger, similar discrete movements are present in new-born and anencephalic infants, which implies that the activity of spinal centres can produce most discrete movements; thirdly, there is similarity between some cortical responses and certain presumably spinal reflex movements to peripheral stimulation; fourthly, one can consider the distribution of motor neurones in the lumbo-sacral grey matter in relation to the destination of their axons, and of the known action of each of the muscles they supply. From this it appears that some complex and apparently meaningless sequences of movement of the leg in response to cortical stimulation could be accounted for by the hypothesis of a simple process of local spread of excitatory effect in the spinal grey matter.

RESUMEN

Se discute la importancia de los movimientos producidos por la estimulación cortical; innecesario postular la existencia de centros motores en la materia gris cortical en el sentido de organizaciones neuronales similares aunque diferenciadas de las que caracterizan la materia gris medular. Las razones que conducen a esta conclusión son: En primer lugar hay una gran similitud entre los movimientos que aparecen como reacción a la estimulación de la corteza y a la de la cápsula interna luego de la hemisferectomia; segundo: aunque la estimulación es capaz de provocar movimientos discretos de un dedo, se observa la presencia de movimientos similares tanto en recién nacidos como en niños anencefálicos, lo que implica la capacidad de los centros medulares para provocar aún los movimientos más discretos; tercero: hay similitud en-

tre algunas respuestas corticales a la estimulación periférica y algunos movimientos reflejos presumiblemente medulares al mismo tipo de estimulación; cuarto: se puede considerar la distribución de las neuronas motoras de la materia gris lumbo-sacral de acuerdo a la ubicación de su respectivo axon y de la acción conocida de cada una sobre los músculos en que actúa. De aquí se desprende que algunas secuencias complejas y aparentemente sin importancia del movimiento de la pierna como respuesta a la estimulación cortical serían explicadas por la hipótesis de que se trata de un simple proceso de propagación local del efecto excitante en la materia gris medular.

RESUME

L'argument va, que, considérant l'importance des mouvements produits par stimulation corticale, il ne semble pas nécessaire de postuler l'existence de centres moteurs dans la matière grise corticale dans le sens l'organisation neuronales similaires, bien que distinctes, à celles qui doivent exister dans la matière grise spinale. Les raisons etant que premièrement il y a une similarité étroite entre les mouvements en réponse d'une stimulation corticale et d'une stimulation de la c. interne après l'hémisphérectomie; deuxièmement, bien que la stimulation puisse produire le mouvement discret d'un seul doigt, des mouvements discrets pareils sont présents chez le nouveau-né et l'anencéphalique, ce qui implique que l'activité des centres spinaux peut produire les mouvements les plus discrets; troisièmement, il y a similarité entre quelques réponses corticales et certains mouvements réflexes probalement spinaux par stimulation périphérique; quatrièmement, on peut considérer la distribution des neurones moteurs dans la matière grise lumbo-sacrale en relation avec la destination de leurs axones et de l'action connue de chaque muscle relié à eux. De ceci il apparaît que quelques séquences complexes et apparemment dépourvues de sens, de mouvement dans la jambe en réponse à une stimulation corticale peuvent être expliquées par l'hypothèse d'une simple extension d'un effet excitatoire dans la matière grise spinale.

ZUSAMMENFASSUNG

Es besteht die Ansicht, betrachtet man die Bedeutung der Bewegungen, die durch corticale Stimulation provoziert werden, es unnötig sei die Existenz eines motorischen Zentrum in der corticalen grauen Substanz im Sinne einer neuronalen Organisation ähnlich-aber doch verschieden von der, die in der spinalen grauen Substanz angenommen werden muss, zu postulieren.

Folgende Gründe werden daher angeführt; Erstens: Es besteht eine grosse Ahnlichkeit zwischen den Bewegungen als Antwort auf die Stimulation des cortex und der capsula interna nach Hemissphärektomie. Zweitens: obwohl die Stimulation diskrete Bewegungen eines einzelnen Fingers hervorrufen kann-sind ähnliche diskrete Bewegungen beim Neugeborenen un Anencephalen auch vorhanden, welches beweist, dass die Aktivität der spinalen Zentren solche diskrete Bewegungen hervorrufen kann. Drittens: Es besteht eine Ähnlickeit zwischen corticaler Antwort und gewissen, wahrscheinlich spinalen Reflexbewegungen bei peripherer Stimulation. Viertens: Man kann die Verteilung der Moto-Neuronen in der lumbo-sacral grauen Substanz, in Beziehung zur Destination ihres Axons und der bekannten Aktivität jedes Muskels zu dem sie gehören-betrachten. Aus diesen Gründen geht hervor, dass manche komplexe und offenbar sinnlose Bewegungsabläufe des Beines, als Antwort auf corticale Stimulation, erklärt werden können, durch die Annahme eines einfachen Prozesses, lokaler Ausbreitung, durch Reizung der spinalen grauen Substanz.

REFERENCES

Bates, J. A. V. (1953): Stimulation of the Medial Surface of the Human Cerebral Hemisphere after Hemispherectomy. Brain, *76*, 405-447.

Bates, J. A.V. (1953): A Comparison between Movements Produced by Stimulation of the Motor Cortex and the Internal Capsule in the Same Individual. J. Physiol. *123*, 49-50P.

Bates, J. A. V. (1957): Observations on the Excitable Cortex in Man in Lectures on the Scientific Basis of Medicine. London: Athlone Press.

Burdon Sanderson, J. (1874): Note on the Excitation of the Surface of the Cerebral Hemispheres by Induced Currents. Proc. Roy. Soc. *22*, 368.

Gamper, E. (1926): Bau und Leistungen eines Menschlichen Mittelhirnwesens. Ztschr. ges. Neurol. Psychiat. (Springer, Berlin) *102*, 154.

Leyton, A. S. F. and *Sherrington, C. S.* (1917): Observations on the Excitable Cortex of the Chimpanzee, Orang-utang, and Gorilla. Quart. J. exp. Physiol., *11*, 135-222.

Lorente de *No, R.* (1953): in The Spinal Cord. Ciba Foundation Symposium (p. 40) Churchill, London.

Romanes, G. J. (1951): The Motor Cell Columns of the Lumbo-Sacral Spinal Cord of the Cat. J. Comp. Neurol. *94*, 313-358.

Romanes, G. J. (1953): in The Spinal Cord. Ciba Foundation Symposium (p. 37) Churchill, London.

Sharrard, W. J. W. (1955): The Distribution of Permanent Paralysis in the Lower Limb in Poliomyelitis. J. Bone Surg. *37*, 540-558.

Sherrington, C. S. (1898): Experiments in Examinations of the Peripheral Distribution of the Fibres of the Posterior Roots of some Spinal Nerves. Phil. Trans., B, *190*, 45-186.

Sherrington, C. S. (1906): The Integrative Action of the Nervous System. Cambridge University Press (2nd edn., 1947).

Ward, J. W. (1938): The Influence of Posture on Responses Elicitable from the Cortex Cerebri of Cats. J. Neurophysiol. *1*, 463-475.

Wood Jones, F. (1941): The Principles of Anatomy as seen in the Hand. Bailliere, Tindall and Cox, London.

• News

SYMPOSIUM ON THALAMIC REGULATION OF SENSORIMOTOR ACTIVITIES

A Symposium on "Thalamic Regulation of Sensorimotor Activities" will be held at Columbia University, College of Physicians and Surgeons, New York City, on November 30 to December 2, 1964. This Conference is the first of a series of Symposia to be presented by the Parkinson's Disease Research and Information Center and is co-sponsored by the National Institutes of Health and the Parkinson's Disease Foundation. Information concerning this meeting may be obtained from the program co-director, Dr. Melvin D. Yahr, New York Neurological Institute, 710 West 168th Street, New York 32, New York.

INTERNATIONAL NEUROLOGICAL CONGRESS

The eighth International Neurological Congress, will be held in Vienna September 5 through 10, 1965. The officers are as follows: president, Prof. Hans Hoff; secretary-general, Dr. Heimut Tschabitscher; scientific secretaries, Drs. K. Gloning and F. Gerstenbrand; and treasurer, Dr. K. H. Spitzy. The mayor themes will be: "Neuromuscular Diseases," with Prof. Raymond Garcin of Paris as chairman; "Disturbances of the Occipital Lobe," with Prof. Hoff as chairman; and "Late Manifestations of Head Injury," with Prof. E. Herman of Lodz, Poland, as chairman. The first 2 subjects will be discussed as joint symposia with the sixth International Congress of Electroencephalography and Clinical Neurophysiology. Free papers also may be read. The deadline for the submitting of contributions to the scientific program is December 30, 1964, and summaries should not exceed 500 words. Completed papers must be submitted by May 15, 1965. All correspondence, titles, summaries, and papers should be directed to the secretary-general, Dr. H. Tschabitscher, Kongressbüro, Wiener Medizinische Akademie, Alserstrasse 4, Vienna IX, Austria.

2nd SYMPOSIUM OF THE FULTON SOCIETY

Vienna — September 4 — 1965

One day before the International Congress of Neurology

FRONTAL LOBE - Program

"L'Aire Oculogyre Frontale du Singe". - Dr. J. M. Brucher (Louvain). — "Frontal Lobe and Basal Ganglia: Behavioral Effects of Lesions in Rodent, Carnivore and Primate". - Dr. H. Lukas Teuber (Boston). — "L'Apraxie dans les Lesions du Lobe Frontal". - Dr. H. Hécaen (Paris). — "Contributions of the Subcortical Structures to Frontal Lobe Function". - Dr. R. Heath (New Orleans). — "The Architecture of the Human Frontal Lobe and the Relation to its Functionoal Differentiation". - Dr. Von F. Sanides (Frankfurt). — "L'Aire Suplementaire Motrice (Enseignement Apporte par la S.E.E.G. dans L'Epilepsie)". - Dr. J. Talairach (Paris). — "Comparative Studies on Psychopathology of Frontal Cortex". - Dr. R. Messimy (Paris). — "Frontal Lobe Epilepsy". - Dr. A. Earl Walker (Baltimore). — "Psychological Deficit Associated with Frontal Lobe Injury in Man". - Dr. O. L. Zangwill (London).

INTERNATIONAL FEDERATION OF SOCIETIES FOR ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY

The International Federation of Societies for Electroencephalography and Clinical Neurophysiology has announced that the sixth international congress will be held in Vienna September 5 through 12, 1965. Three symposia will be held, which are as follows: "Modern Trends in the Neurophysiological Investigation of Brain Diseases," "The electroencephalogram in Stress: Physiological and Psychological Aspects," and "Spinal Cord Function: Mechanisms of Physiological and Pathological Reflex Activity." The meeting date will coincide with those of the eighth International Congress of Neurology, and joint symposia will be held on the subjects "Disorders of the Occipital Lobe" and "Neuromuscular Diseases." Free communications on electroencephalography and electromyography also are planned. Further information may be obtained from the secretary, Dr. K. Pateisky, Kongressbüro, Wiener Medizinische Akademie, Alserstrasse 4, Vienna IX, Austria.

Book Reviews

"Electromyographie by François Isch". — Editions Doin - Deren & Cie. 8 Place de l'Odeon. Paris, 1963. pp. 258.

Throughout the pages of this very valuable book Dr. Isch provides the readers, a considerable amount of information on electromyography and its clinical uses.

The book comprises 4 parts. — *The first one* is of general and technical character; it contains the physiological basic principles indispensable for the comprehension of bioelectric activity in muscles and problems related to tracing, amplifying and recording of muscle potentials. Phenomena observed in normal and pathological muscles are pointed out.

In the second part the diseases of peripheral motor neuron and muscle are studied. Neurogenic and myogenic atrophies; irritative states of the peripheral motor neuron; disturbances in neuromuscular transmission and disturbances of metabolic origin.

The third part of the book is concerned about motor disorders of central origin and treats all the syndromes in which these may appear.

The fourth part presents us the contribution of electromyography to the study of children's diseases and of several special fields, traumatology, orthopedia; ophtalmology and otorhinolaryngology.

In the final chapter of the book, the author briefly and clearly exposes all the benefits that can be drawn out from electromyography for diagnosis as well as for stating the importance of the lesion and following its evolution.

The author's purposes of writing this fine book giving the reader, on one side knowledge concerning both the technical aspect of electromyography and records interpretation, and on the other side the description of its clinical usefulness and importance, have been perfectly achieved. All the subjects are treated along different chapters in a complete, methodical and didactic way.

The author is undoubtedly a electromyographist as well as an expert clinician.

The book's print and presentation are excellent.

The reviewer would emphasize that this book can be of great value for electromyographists as well as for clinical neurologists.

V. S.