Further Notes on the Centre Median Nucleus of Luys

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"The number of thalamic nuclei has increased in the last few years. To the four classical centers that were described by Burdach one should add the Centre median of Luys and the semi-lunar of Flechsig. Finally, von Monakow and Nissl have subdivided each principal nucleus into a large number of secondary pleiades on the basis of the relative volume of cells and the number of myelinated fibers of the interstitial plexus. It is our belief that the individuality of the thalamic centers should not be based upon the aspect of such centers in Nissl or Weigert sections, but upon the study of their fine anatomy as it appears in silver chromate stained sections and, above all, upon the specificity of their connections. A nucleus should only be considered as an individual center when besides having peculiar structural features, its afferent and efferent fibers are specific."

Santiago Raymon y Cajal 1904
14th International Medical Congress
Translation by Enrique Ramon-Moliner

In 1931 Rioch added a "note" to his then definitive studies on the diencephalon of the Carnivora (Rioch, 1929 a,b, 1931a). This addendum attempted to rectify his earlier inclusion of the nucleus centre median of Luys in the region he identified as the nucleus parafascicularis. Following supplemental studies in a series of primate brains and a re-evaluation of the parafascicular region in Carnivora, Rioch delineated a rather limited centre median nucleus in the cat and dog as a nuclear entity homologous with the centre median exhibited in higher species. He described this small-celled centre median as contiguous with, but separable from, the parafascicularis caudally and continuous with the pericentral and central lateral intralaminar nuclei at its rostral pole. His observations in lower monkeys (Rioch, 1931b), together with Le Gros Clarks (1929, 1930) corresponding comparative studies, presented data suggesting a progressive phylogenetic development of the nucleus centre median.
Because of its limited development and small, pale staining cellular composition in sub-primate species, the centre median has frequently been overlooked, mislabelled or included within the limits of adjacent nuclei. More recently, there has been a reverse tendency on the part of authors to erroneously label a number of nuclei in the caudal intralaminal region as centre median (Jasper and Ajmone-Marsan, 1954). In studies in man and other higher primate species, involving nuclei in the region of the centre median, the author and other investigators have frequently called attention to the fact that the centre median and adjacent cell groups in the region of the internal medullary lamina have rather vague boundaries with each other as well as with the surrounding principal thalamic nuclei (Mehler, Feferman and Nauta, 1960, Mehler, 1962, 1965a).

In his sub-primate studies, Rioch (1931 a,b) called attention to the great numbers of fibers of passage which course through the internal medullary lamina region. The present author and others (e.g., Le Gros Clark, 1936, Walker, 1938a, Nauta and Kuypers, 1958, Stewart and King, 1963) have demonstrated such fibers of passage in the caudal intralaminar region that some authors have interpreted as connections with the centre median (Wallenberg, 1900, Bowsher 1957, Anderson and Berry 1959). It appears, therefore, that in many cases the limited cytological development or fibrous obscuration of the centre median has frequently led some investigators to improper identification of the centre median or to interpret ascending fibers of passage as connections with the nucleus.
Cytoarchitectural studies of the thalamus led Le Gros Clark (1929, 1930) to suggest that the centre median (CM) represents a caudal elaboration of intralaminar cell groups because its cells resemble certain small-celled elements intercalated in the internal medullary lamina. Similarly, its phylogenetically constant relationship with the nucleus parafascicularis prompted Papez (1932) to conclude that CM in lower mammals is comparable with the lateral part of parafascicularis. In 1954, Kulhenbeck in concurrence with Papez's notion suggested that phylogenetically CM represents a lateral differentiation of the parafascicular nucleus which is prominent in all lower mammalian species.

In respect to a cytoarchitectural definition of CM, the author and his associates basically subscribe to Cecile and Oskar Vogt's (1941) subdivision of the nucleus in man. In extensive studies of normal and pathological material the Vogts demonstrated that CM can be subdivided into a small-celled ventrolateral region and a larger-celled dorsomedial region adjacent to the internal medullary lamina. Niimi, Katayama, Kanaseki and Morimoto (1960), on the basis of comparative cytoarchitectural studies in a number of lower mammalian forms, suggest that the larger-celled dorsomedial paralaminar region of the Vogts' classification probably corresponds to the pars lateralis of the parafascicular nucleus which appears to predominate in subprimate species. Contrasting these observations with ontogenetic studies of CM in the human fetus, Niimi and associates then concluded that probably only the small-celled ventrolateral CM region corresponds to the CM proper. In the same year we (Kehler et al., 1960) arrived at a similar conclusion following comparable observations (Mehler, 1957, 1959, 1960) which suggested that only the ventrolateral small-celled region that exhibits relatively disproportionate phylogenetic increases in volume, in contrast with the dorsomedial paralaminar region, should be identified as the nucleus centre median.
Comparative anatomical studies of the afferent and efferent connections of the CM-Pf complex and adjacent intralaminar region are being continued. Data suggested by current studies indicate that experimental means are now available to objectively establish the homologues of these and other thalamic nuclei by delineating them from adjacent cell groups on the basis corticofugal or other afferent projections recently established (see, recent findings). We believe that judicious use of the selective silver impregnation method of Nauta (1957) should—like the prudent application of the Marchi method by LeGros Clark (1936) and Walker (1938a, b)—allow for significant advancement in our understanding of the neural organization of the thalamus and lead us to a more realistic knowledge of its function. In as much as the identification of the true nuclear homologue of CM in many subprimate species is still a matter of speculation, the present report will be limited principally to those species in which sufficient experimental data have accumulated to meet Cajal's (1904) criteria for the confirmation of the individuality of a nucleus.
The efferent projections of the centre median (CM) were the topic of much controversy during the past three decades. During the 1930's, however, experimental studies of thalamo-cortical relationships in monkeys revealed that CM was not a cortically dependent nucleus (Walker, 1935, 1938a). Le Gros Clark and Russell's (1939) study appeared to experimentally rule out the insular cortex, suspected by Dejerine (1901), which was not studied in detail by Walker. Their conclusion was confirmed in Roberts and Akert's (1963) recent re-evaluation of the thalamic projections to the insular region. Included in Le Gros Clark and Russell's report were observations on a human case with a large lesion in the ventrolateral part of the putamen. They noted that CM in this case was almost totally degenerated except for a number of cells in its dorsomedial extremity.

In 1941, Cecile and Oskar Vogt demonstrated that the ventrolateral, small-celled subdivision of CM preferentially projects to the putamen and the larger-celled dorsomedial region of their subdivision of CM projects mainly to the caudate. Their analyses of cases of status marmoratus clearly established these connections in the human brain and subsequent neuropathological studies by McLardy (1948) and Simma (1951) confirmed the CM-striatal relationships. Experimental confirmation of the Vogts findings appear in Powell and Cowan's (1956) studies in the monkey. For a more comprehensive review of the literature dealing with retrograde cell degeneration studies, the reader is referred to Powell and Cowan's narrative or to Powell's (1958) subsequent review.
Antegrade degeneration studies in the monkey confirm the predominantly putaminal distribution of the CM projections demonstrated by Nauta and Whitlock (1954) in studies in the cat. The lesion shown in figures 1 and 2 was produced by a stereotaxically directed electrode introduced from behind, 4 mm lateral to the midline, at an angle of 30 degrees from the horizontal plane.

Figures 1 and 2 --- about here 2/3 - 3/4 page

The fiber degeneration issuing from the lesion is tripartite in distribution. Ventrolaterally, the intranuclear neuropil of CM is disrupted by fine-fibered degeneration which is confined within the limits of the nucleus up to its dense fibrous border with the pulvinar and ventral posterior medial complex. Ventromedially degenerating fine-fibered fascicles course caudomedially and terminate primarily within the limits of the parafascicular nucleus (Pf). The fiber caliber and fasciculation of this degeneration projecting upon Pf suggests interruption of prescentral corticofugal connections which descend through the region of the lesion (see; corticofugal projections). Dorsad to the lesion, dense fiber degeneration courses dorsolaterally in the internal medullary lamina upon which the lesion impinges. Terminal degeneration, dorsal and lateral to the lesion, according to Olszewski's (1952) terminology, distributes to the pars multiformis of the medialis dorsalis (MDmf), nucleus centralis lateralis (Cl) and adjacent regions of the ventral lateral (VL) nucleus. The pattern and density of these intra-thalamic connections primarily duplicate the distribution of medially coursing (i.e., via an intralaminar route) cerebello-fugal fiber projections repeatedly observed by us in other studies in the monkey (Mehler et al 1958, Mehler 1965). Scattered degeneration observed in the pulvinar and lateral
posterior nuclei relates to incidental occipito- or tecto-thalamic projections interrupted by the insertion of the lesion electrode.

The regional distribution of projections to the striatum exhibited in this case, (fig. 3) corresponds remarkably well with basic topological organization demonstrated in the monkey by Powell and Cowan (1956). The trans-thalamic passage exhibited by these projections similarly confirms and extends both Nauta and Whitlock's (1954) and Powell's (1958) observations that the CM-striatal projections describe a wide path through the ventral anterior (VA) and rostro-lateral reticular nuclei of the thalamus. With the exception of the intra-thalamic connections that resemble the distribution of ascending fiber degenerations which pass through the lesion region, the dense fine-fibered fascicles of the CM efferent projections do not appear to distribute terminal connections in their passage through the lateral region of the nucleus ventralis anterior. As these degenerating fascicles interdigitate with the normal fibers of the external medullary lamina, some terminal-like axo-dendritic ramifications appear in relation to reticularis thalami cells encountered in the region of passage. In other words, these connections with the reticularis thalami do not present the more obvious axo-somatic type of termination and their distribution appears limited to the region of passage of the projections to the striatum similar to that described in the cat by Nauta and Whitlock (1954).

Figure 3 ---- about here 1/2 page
In their migration across the internal capsule (CI) to connect with the striatum the degenerating CM projections describe an even wider swath than is encountered in their trans-thalamic trajectory. Such a distribution might explain Nashold, Hanbery and Olszewski's (1955) finding that striatal lesions involving adjacent portions of the internal capsule resulted in more extensive intralaminar cell changes than partial lesions of the caudate or putamen. Most of the lesions in Powell and Cowan's (1956) cases also impinged on the internal capsule presumably enhancing the completeness of their CM and intralaminar nuclei cell changes. The topology of the CM changes in their "experiment 4" corresponds reasonably well with the findings in the present case and best illustrate the point in question.

As depicted, the majority of the afferent projections to the striatum course dorsal to the pallidal segments. Some projections to the putamen, however, traverse the lamella pallidi internus to distribute to the more ventral and oral regions of the striatum. No connections with the pallidal segments are evident. Degeneration in the fornix and a small number of degenerating fibers, distributing to the dorsal part of the claustrum and the transitional zone between the insular and parietal corticies, relates to incidental damage. The lesion electrode, stereotaxically directed from behind, entered the occipital cortex, passed through the ipsilateral splenium of the corpus callosum, the fornix, and the nucleus pulvinaris medialis and entered the caudal pole of the nucleus centrum medianum.
Ascending afferent connections with CM were conceived before the turn of the century by von Monakow (1895), Dejerine (1897) and others. In 1901 Dejerine concurred with von Monakow in the notion that CM receives a certain number of fibers of the medial lemniscus and represented a kind of relay ganglion between the dorsal funicular nuclei and the cortex. The literature reveals practically no experimental data supporting the existence of medial lemniscal connections with CM (LeGros Clark, 1936, Bowsher, 1958). Dejerine considered that some fibers of the superior cerebellar peduncle and reticular formation also enter CM. Wallenberg (1900) was among the first to describe ascending Marchi degeneration from the spinal trigeminal nucleus in the rabbit which disappeared into the lateral region of the medial nucleus, identifying the area as "Centre median de Luys." Subsequent studies led Wallenberg (1905) to conclude that fibers issuing from the principal sensory nucleus of the trigeminus represent a dorsal trigeminal pathway that projects into the internal medullary lamina and CM. Other investigators employing the Marchi method in experimental studies in rabbits and cats reported similar findings. The majority, like Wallenberg, followed von Monakow's (1895) assumption that his nucleus "medialis b" in the cat is homologous to the CM of higher forms. Rioch (1931 b) called attention to the fact that such a contention is only partially correct since only a small circumscribed caudal portion of von Monakow's nucleus medialis b appears to correspond to the simian CM. However, Dejerine's (1926) later affirmation of "trigemino - CM" connections in man led to an almost doctrinal acceptance of Jacob's (1923) view that CM serves as a center for automatisms of the head region.
One of the most significant papers which maintained support of the contention that CM receives ascending connections is Papez and Rundles (1937) study of the so-called dorsal trigeminal tract. This report contains a concise review of the older literature bearing on the afferent and efferent connections of CM which limitations prevent us from discussing in detail. On the basis of studies of normal horse and human brain material, Papez and Rundles concur with Dejerine's later idea that the majority of dorsal trigeminal tract fibers originating from the main sensory nucleus of the trigeminus appear to end chiefly in the CM of the contralateral side. In an experimental re-evaluation of the dorsal trigeminal tract employing the Marchi method, Russell (1954) demonstrates that the myelinated axons of the main sensory nucleus do not contribute to the so-called dorsal trigeminal tract but ascend through the ventral tegmentum just internal to the medial leminiscus and distribute to the ventral posterior medial (VPM) nucleus of the thalamus and the centre median.

Comparable studies employing the selective silver impregnation method of Nauta (1957), which will be reported in detail in a later communication, confirm Russell's observations with respect to the ascending course of these projections and their connections with the pars magnocellularis of the VPM. However, these data reveal that the degeneration in the periphery of CM, often misconstrued as endings in Marchi preparations, actually constitute fibers of passage which turn ventro-laterally and terminate on VPM cells. Similarly, Stewart and King's (1963) recent Nauta method study of projections from the pars caudalis of the spinal trigeminal nucleus reveals that the "trigemino - CM" projections described by Wallenberg (1900) and others actually constitute
"trigemino-intralaminar" connections which course through the CM-Pf complex.

We have repeatedly demonstrated identical findings in respect to spinal projections en passage dorsal to the CM-Pf complex which connect with central lateral, para- and intralaminar cell groups (Mehler, et al., 1956, 1957, 1960, 1962, 1969a). Sagittal sections (Figs. 4 and 5) best illustrate the predominantly dorsal course and "C1" distribution found in such cases.

Figures 4 and 5 ---- about here, full page

Concurrently, however, comparable studies in man (Bowsher, 1957) and cat (Anderson and Berry, 1959) indicated positive data with respect to spinal connections with CM but our findings in carefully controlled chimpanzee experiments (Mehler, 1957, 1959, 1960) and human cordotomy cases (Mehler, 1962) clearly indicated to us that these reported differences in man were more apparent than real. Our contention that reports of positive findings in the cat also represented erroneous interpretation of the limits of the small feline CM, or fibers of passage, was supported by Getz's (1952) earlier negation of CM connections in studies employing both the Glees (1946) and the original Nauta (1950) methods. The presence of primarily "C1" intralaminar terminations, first identified by Getz, was confirmed by Nauta and Kuypers (1958) who, in a qualified manner, described spinal, trigeminal and reticular projections as only entering the CM-Pf complex en passage into rostrally adjacent "C1" nuclei. Nauta and Kuypers' report is frequently misquoted as supporting evidence for the existence of ascending afferent connections with the centre median.

The cerebellar afferents to CM, suggested by Dejerine (1901), have reportedly been demonstrated in the cat (Thomas, Kaufman, Sprague and Chambers, 1957, Cohen, Chambers and Sprague, 1958) monkey (Carpenter, 1959) and man (Hassler, 1949). Le Gros Clark (1936) and Walker (1938a), however, both concluded that the
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degenerating cerebellar fibers they observed penetrating the CM, in the monkey and chimpanzee respectively, coursed through the nucleus and distributed to nuclei in or adjacent to the internal medullary lamina. In 1958 we (Mehler, Vernier and Nauta, 1958) confirmed such a course and terminal distribution of projections from the dentate and interpositus nuclei in the monkey and chimpanzee. These data have been supplemented and extended and will be reported elsewhere (Mehler, 1965b). The basic course and distribution of the cerebello-thalamic projections, abstracted from frontal, horizontal and sagittal brain series, is diagrammatically summarized in figure 6. It should be pointed out that the density of the cerebello-intralaminar connections greatly exceed the density of the spinal projections to the "Cl" intralaminar region.

Gigantocellular reticular formation projections have reportedly been demonstrated by Mallart and Bowsher (Bowsher, personal communication) in support of electrophysiological reports suggesting reticulo-CM connections (Magoun and McKinley, 1942, Starzl, Taylor and Magoun, 1951, Albe-Fessard and Rougeul, 1958).

On the basis of repeated analysis of CM in over 50 reticular formation lesion cases in cats (C. Teg. series, e.g., Nauta and Kuypers, 1958, Mehler, 1965a) and monkeys (Mehler, unpublished) we have never observed significant terminal connections with the nucleus as defined in the present study. In many cases fibers of passage comparable with the spinal, trigeminal or cerebellar projections to the "Cl" intralaminar region can be observed, in or adjacent to CM, but terminal connections comparable with those exhibited in pallidal or precentral cortical lesions cannot be demonstrated in lesions of the reticular formation (see recent findings).
The possibility that the parvocellular portion of the nucleus ruber might project to the CM was considered by Powell (1958). Recognizing the concomitant increases in the size of both nuclei in the primate brain, Powell suggested that this relationship should be investigated. Subsequently, Hinman and Carpenter (1959) reported dense pre-terminal degeneration in CM in their study of rubral efferent projections in the cat. The latter authors state that the lesions in these cases were produced by electrodes introduced in a vertical plane on the ipsilateral side. In experiments in which lesions of the nucleus ruber are produced by electrodes introduced from the contralateral side (e.g., case CT.4 in Nauta and Kuypers, 1958), no significant terminal degeneration is exhibited in the feline CM as defined in the present study. Lesions extending into the pre-rubral region, or H-Fields, however, often elicit degeneration in CM by interrupting pallidal or cortical projections traversing the respective diencephalic regions. Glees and Wall's (1946) report of CM fiber connections originating from the substantia nigra appears to be an example of such lesion contamination resulting from the insertion of the lesion electrodes through critical diencephalic regions of passage (see; Nauta and Mehler, 1965). Recent studies of nigral efferent projections in the monkey utilizing stereotaxic approaches from the side and behind the diencephalon do not reveal nigral connections with the CM (Cole, Nauta and Mehler, 1964).

In the absence of positive data in our material we have repeatedly contended that such "positive" claims in anatomical and electrophysiological studies stem mainly from problems of interpretation resulting from errors in delimiting CM from adjacent principal and intralaminar nuclei (see, Mehler, et al., 1960, 1962, 1965). Positive findings in subsequent studies have borne out our claims.

Figures 6 and 7 ---- about here, full page
Recent findings. In attempts to provide data on the organization of the subthalamic region lesions were placed in the H-field of Forel in the cat (e.g., cases CTeg. 143 and 147, Nauta, unpublished). Observations in such cases suggested to us that a system of afferent projections to the CM must originate from regions ventral or rostral to the dorsal thalamus. Glees and Wall (1946) had described such connections in the monkey following Marchi analysis of comparable ipsilateral, perpendicularly oriented, stereotaxic lesions of the subthalamic nucleus or the substantia nigra. However, a number of similar lesion cases produced by introducing the lesion electrode from various angles or from the contra-lateral side (e.g., Nauta and Kuypers, 1958, Cole, Nauta and Mehler, 1964) could not confirm either our own findings or the previously derived conclusions of Glees and Wall. Finally on attaining an uncomplicated lesion of the internal segment of the globus pallidus in the monkey and lesions of the nucleus entopeduncularis in the cat, an explanation for the above mentioned findings was obvious (Nauta and Mehler, 1961). It became apparent then that any vertically directed lesion electrode passing through the ipsilateral thalamus en route to the subthalamic region could easily interrupt the pallido-CM collateral-like offsets passing through the ventral thalamic nuclei or destroy the parent fibers in the fasciculus thalamicus as they reflect through the H-fields (Fig. 7).

The complete description of pallido-fugal projections in the monkey, upon which figure 7 is based, is detailed elsewhere (Nauta and Mehler, 1964, 1965). Figures 8 to 11 illustrate the corresponding CM distributions of projections from the entopeduncular nucleus in the cat, the long suspected subprimate homologue of the internal pallidal segment. Dense terminal degeneration in these cases
selectively enmeshes only the ventrolateral small-celled region we have repeatedly depicted as the CM in previous studies (Mehler, 1957, 1960, 1965a). The distribution of these terminal connections objectively delineates the small feline CM from adjacent "C1" intralaminar cell groups and from the nucleus parafascicularis.

Figures 8-11 --- about here, 2/3 page

Data suggesting that the afferent connections with the CM might originate from other forebrain structures has been extant for a number of years (Dejerine, 1901, McLardy, 1931). In his description of the myeloarchitecture of the CM, Olszewski (1952) pointed out that the fiber bundles present in the oral region of the CM decrease in number as one examines successively more caudal levels, decrease in such a manner that the more caudal portions of the nucleus show no fiber bundles. We can find no specific mention in the literature of myeloarchitectural changes in the neuropil of the CM in investigations of damage to the internal segment of the globus pallidus but several authors have described such changes in the human thalamus in cases with cortical damage. For instance, on the basis of such findings Papez and Rundles (1937) ascribed to the possibility of Dejerine's (1901) belief that fibers to the CM originate from a cortical area generally included in the region of the quadrangle of Marie.

In 1956 Auer demonstrated such cortico-fugal fibers to the intralaminar region that could also be traced into the CM-Pf complex in the cat following partial ablations of the coronal and proreus gyri. Recent studies by Niimi, Kishi, Miki and Fujita (1963) confirm and extend Auer's observations in the cat. They state that corticofugal degeneration is found primarily in the ventrolateral
CM region while the dorsomedial "CM" region (i.e., Niimi et al., 1960) is almost free from degeneration. They indicate that no cortical projections terminate in the entopeduncular nucleus and that these bundles in the internal capsule traverse mainly the dorsomedial portion of the nucleus. It should be mentioned that in all our entopeduncular lesion cases, approached from the lateral side only the ventrolateral portion of the nucleus and the immediately adjacent optic tract was destroyed. These data strongly suggest that separate pallidal (entopeduncular) and cortical projections to CM exist in the cat similar to those demonstrated in the monkey.

Cortical ablation studies in the monkey involving the pre-motor region (i.e., Areas 6 and 8 of Broadmann) reveal, in addition to other distributions, corticofugal projections whose terminal distributions are essentially limited to the "Cl" intralaminar region and the nucleus parafascicularis (Astruc, 1964). Conversely, corresponding studies of the "motor" cortex (i.e., Area 4) by Petras (1964) demonstrate massive fine-fibered terminations that distribute throughout CM. More recently, Petras (1965) has confirmed similar "precentral-CM" connections in the chimpanzee. Figure 7 depicts (heavy interrupted line) confirmatory data abstracted from a precentral ablation case cut in the sagittal plane of section (Mehler, unpublished). These latter patterns, although significantly more dense in appearance than the pallido-fugal connections, strikingly complement the areal distribution of the pallido-centre median connections previously demonstrated. In both of the cortico-fugal fiber studies cited, the initial findings indicate that the projections to both the principal and the intralaminar nuclear groups are quite specific. Continuing studies may yet reveal further details in regard to the specificity of these and other connections with the diencephalon.
Summary and Conclusion

Neuropathological evidence supported by consistent experimental findings in both retrograde cell, and antegrade fiber degeneration studies establishes that the centre median projects primarily upon the putamen. Although the presence of ascending afferent connections with the centre median has been reported, the author and his associates have repeatedly refuted these claims. We contend that such positive claims in both anatomical (e.g., Wallenberg, 1900, Bowsher, 1957) and electrophysiological (e.g., Kruger and Albe-Fessard, 1960, Collins, 1964) studies involving the caudal region of the internal medullary lamina represent fallacious identifications or erroneous localizations of "CI" intralaminar connections, or reflect neural events reflecting from the globus pallidus or the precentral cortex. The latencies in many of the neurophysiological studies strongly suggest that this is the case (Bernstein and King, 1964).

Our contention that the only significant afferent connections with the centre median originate from forebrain structures lying rostral to this nucleus is supported by recent demonstrations that both the globus pallidus and the precentral "motor" cortex project in a convergent manner upon the nucleus centre median. In the cat and monkey, terminal connections originating from the internal segment of the globus pallidus (Nauta and Mehler, 1961, 1964, 1965) and/or the precentral "motor" cortex (Petras, 1964, 1965) literally outline homologous nuclear regions, which, in both species, exhibit corresponding efferent projections to the putamen. These experimental data confirm findings originally demonstrated in human neuropathological material and lend support to Schulman's (1957) hypothesis that the centre median might play a critical role in mechanisms that mediate certain dyskiniasias.
It is now a century since Luys (1865) first identified the large pale nucleus, in the "center towards the middle" of each side of the human thalamus, and proposed the name centre median. In the light of recent experimental evidence, however, it appears that Luys' original conjecture that the centre median was a convergence center for all peripheral impulses; supported by Dejerine's views and perpetuated by many contemporary neurological investigators, has obscured the determination of the nucleus' true functional status in the organization of the thalamus and delayed our understanding of its actual neurological relationship with the forebrain.
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