Rabi Datta
A Treatise on Human Physiology

Reading excerpt

A Treatise on Human Physiology
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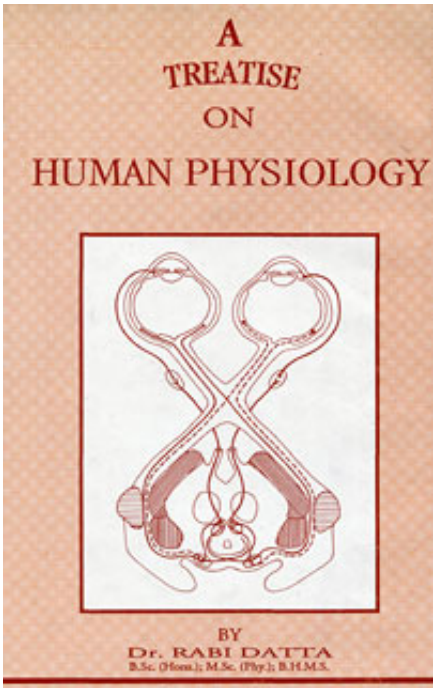

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(11). Describe the connections and functions of cerebellum.

Connections of Cerebellum:

The cerebellum is connected with the rest of the nervous system by the superior, middle and inferior peduncles, each of which contains both afferent and efferent fibres.

Afferent Connections: Afferent fibres to the cerebellum convey impulses primarily to the cerebellar cortex. These tracts enter the cerebellum mainly via the inferior and middle cerebellar peduncles and include spinocerebellar. cuneocerebellar, olivocerebellar, vestibulocerebellar and pontocerebellar tracts as well as numerous smaller bundles. Within the cerebellar cortex these fibres lose their myelin sheath and end either as mossy fibres or climbing fibres.

Mossy fibres bifurcate repeatedly in the white matter, enter the granular layer and often provide branches to adjacent folia.

Climbing fibres pass from the white matter through the granular and Purkinje cells. These non-myelinated fibres divide into numerous branches and climb the dendritic tree of the Purkinje cell. Climbing fibres contact only the smooth branches of there dendrites, collaterals of climbing fibers make contacts with almost all cell in the cerebellar cortex. (Fig. 36A.).

Efferent conections:

Each region of the cortex sends the axons of its Purkinje cells to a definite part of the cerebellar nuclei. This corticonuclear system is organised in an orderly fashion. The vermis projects to the medial or fastigial nucleus and is represented there in the same rostrocaudal order as the folia. The most lateral portions of the cortex, except the flocculus, project to the lateral or dentate nucleus. Intermediate areas send fibres to the nucleus interpositus (n. globosus and n. emboliformis of man).

Probably all of the efferent fibres of the cerebellum arise in its nuclei, except for some running directly from the nodulofloccular lobe to the vestibular nuclei. The fastigial nucleus sends fibres to

the vestibular nuclei and to the reticular formation (RF) of medulla. The dentate & interposed nuclei project through the superior peduncle to the red nucleus and thalamus. The influence of the cerebellum is therefore exerted on motor neurons through the vestibulospinal, reticulospinal and rubrospinal pathways and on the precentral motor cortex through the ventrolateral nucleus of thalamus.

Functions of the Cerebellum :The cerebellum is concerned with the co-ordination of somatic motor activity., the regulation of muscle tone and mechanisms that influence and maintain equilibrium.

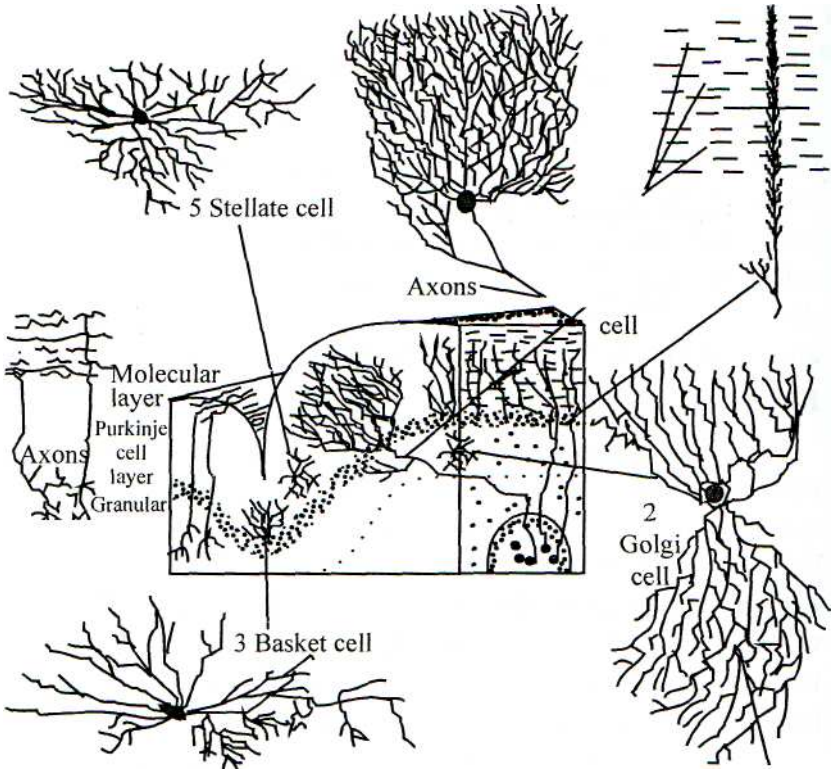


Fig.: 36A. Location and structure of neurons in the cerebellar cortex.

Afferent pathways convey impulses from wide variety of different receptors, including the organs of special sense. Among these afferent systems, the input from stretch receptors (i.e., muscle spindle and Golgi tendon organ) is especially large. These impulses are conveyed by the spinocerebellar and cuneocerebellar tracts. The principal function of stretch receptors appears to be unconscious neural control of muscle tone.

Neocerebellar Lesions:

Lesions involving the cerebellar hemispheres and the dendate nucleus affect primarily skilled voluntary and associated movement.

The muscles become hypotonic (flabby) & tired easily. The deep tendon reflexes tend to be sluggish and often have a pendular quality.

There are severe disturbances of co-ordinated movement referred to as asynergia in which the range, direction and force of muscle contractions are inappropriate. Distances frequently are improperly gauged (dystmetria) and fall short of the mark or exceed it (past-pointing).

Rapid successive movements,e.g., alternately, supinating and pronating the hands and forearms are poorly performed (dysdiadochokinesis).

The tremor seen in association with neocerebellar lesions occurs primarily during voluntary and associated movements. This is referred to as "intention tremor"' as it is not present at rest.

Ataxia is an asynergic disturbance associated with neocerebellar lesions which results in a bizarre distortion of voluntary and associated movements. It includes particularly the axial muscles, and groups of muscle around the shoulder and pelvic girdles. This disturbance is evident during walking and is characterized by muscle contractions which are highly irregular in force, amplitude & direction, & which occur asynchronously in different parts of the body.

Nystagmus commonly is seen in association with cerebellar disease; it is most pronounced when the patient deviates the eyes laterally toward the side of lesion. This disturbance consists of an oscillatory pattern in which the eyes slowly drift in one direction and then rapidly move in the opposite direction to correct the drift.

Speech disturbances are common in association with cerebellar lesion of long standing, speech often is slow, monotonous and some syllables are unnaturally separated. There is a slurring of speech and some words are uttered in an explosive manner.

Archicerebellar Lesions : Lesions of this type produce disturbances of locomotion & equilibrium bilaterally. The patient is unsteady in the standing position and shows considerable swaying of the body. When attempts are made to walk there is staggering & a tendency to fall to one side or backwards. The gait is jerky, unco - ordinated & resembles that of a drunken man.

Anterior lobe of Cerebellum : There is no established paleocerebellar syndrome in man, but lesions of the anterior lobe of the cerebellum in the dog produce severe disturbances of posture and greatly increased extensor muscle tone.

Sherrington in a classic experiment demonstrated that electrical stimulation of the anterior lobe of the cerebellum could inhibit extensor muscle tone in a decerebrate aminal.

Computer Functions: Recent physiological studies of the functional organisation of the cerebellar cortex suggest that the cerebellum may function as a kind of computer in the regulation and control of movement.

The cerebellum appears to organize & integrate information flowing to it via numerous neural pathways. The cerebellar output participates in the control of motor function by transmitting impulses to: l)certain brain stem nuclei (i.e.,the lateral vestibular & red nuclei) that in turn project to spinal levels, and 2) thalamic relay nuclei which can modify the activity of cortical neurons directly concerned with motor function.

The fact that the cerebellar cortex transforms all input into inhibition, suggests that there is probably no dynamic storage of information in the neuronal circuitry, of the cerebellar cortex. Thus, the cerebellum processes its input information rapidly, conveys its output indirectly to specific brain stem nuclei, & has virtually no short- term dynamic memory. These qualities enhance the performance of the cerebellum as a special kind of computer in that it can provide a quick and clear response to the input of any particular set of information.

(12). **Describe the connections and functions of hypothalamus.**

Connections of the hypothalamus :

The hypothalamus, in spite of its small size, has extensive and complex connections. Some fibres are organized into definite bundles, while others are diffuse and difficult to trace.

AFFERENTS: The afferent connections of hypothalamus which have been established are:

1) The medial forebrain bundle (MFB) - a complex group of fibres arising from the basal olfactory regions, the periamygdaloid region, and the septal nuclei, that pass to & via, the lateral parts of the hypothalamus. This tract is well developed in lower vertebrates, but in man it is small.

2) Hippocampo-hypothalamic fibres originate from the hippocampus & the fornix, to the medial mammillary nucleus, lateral preoptic nuclei, and the dorsal hypothalamic area.

3) Amygdalo-hypothalamic fibres follow two pathways to the hypothalamus : (a) The stria terminalis and (b) the course ventral to the lentiform nucleus. The former fibres are distributed to the medial preoptic nucleus, the anteror hypothalamic nucleus, and the ventromedial & arcuate nuclei. The later passes to the lateral hypothalamic nucleus.

4) Thalamo - hypothalamic fibres — arise chiefly from the

midline thalamic nuclei.

5) Brain stem reticular afferents—ascend to the hypothalamus via the mammillary peduncle and the dorsal longitudinal fasciculus. Fibres in this bundle spread out over caudal & dorsal regions of the hypothalamus where they become part of the periventricular system.

6) Retinohypothalamic fibres arise from the ganglion cells of the retina, and project via the optic nerves & chiasma to the suprachiasmatic nuclei.

EFFERENTS :

The efferent connections of the hypothalamus appear, in part, to be reciprocal to the afferent systems. There are several efferent hypothalamic pathways which have no counterpart among afferent systems.

1) The MFB conveys impulses from the lateral hypothalamus anteriorly to the nuclei of the diagonal band & to the medial septal nuclei which in turn send fibres to the hippocampal formation via the fimbria of the fornix.

2) The dorsal longitudinal fasciculus contains descending fibres from medial and periventricular parts of the hypothalamus distributed to the central gray matter of the midbrain & the tectum.

3) The pathways by which impulses originating in the hypothalamus are relayed to the nuclei of medulla & spinal cord are poorly understood. It is presumed that it occurs via reticular formation.

4) Mammillary efferent fibres, arising from the medial mammillary nucleus, and to a lesser extent from the lateral and intermediate mammillary nuclei, form a well-defined bundle which divides into two : 1) The mammillothalamic tract and the mammillotegmental tract.

The former projects to anteroventral and anteromedial thalamic nuclei of the same side. The second are terminate in the dorsal and ventral tegmental nuclei.

utilized for egg-shell formation. In women, deficiency in bone matrix formation, rise in mineral reabsorptions from bones, osteoporosis, delayed recovery from fractures and negative calcium balance are sometimes observed at or after menopause. Oestrogen & testosterone bring relief.

Clinical conditions :Rickets and osteomalacia, produced by the dietary defficiency of calcium, phosphorus or vitamin D, are characterized by reduced intestinal absorptions of calcium & phosphates, low serum Pi, rise in urinary phosphates, demineralization of bones, and Ca^{++} balance serum calcium is, however, almost normal in rickets although sometimes it falls in osteomalacia. Hypervitaminosis D, caused by excessive vitamin D intake, may raise the serum calcium. Advanced renal failure in chronic nephritis shows low serum Ca^{++} and high serum Pi, the phosphate retention contributing to acidosis. Excessive PTH secretion (hyperparathyroidism) causes demineralisation of bones, rise of serum -Ca^{++}, fall in serum-Pi, rise in Ca^{++} & phosphates excretions & metastatic calcifications of soft tissues, failure of PTH secretion (hypoparathyroidism) reduces the serum -Ca^{++}, urincalcium, and urinary Pi, raises the serum Pi moderately and produces hypocalcemic tetany.

(17). **Describe sources, functions and deficiency signs of Vit. D.**

Vit. D is soluble in fat-solvent and insoluble in water. About six varieties of vit. D have been discovered, among them D2 (calciferol) and D3 (cholecalciferol) are important. It can stand ordinary cooking process as it is heat stable.

Sources : The important sources of vit. D are fish liver oils, e.g. codliver oil, halibut liver oil. Moderate amount of Vit. D is present in butter, eggs & milk. It is almost nil in liver of mammals. Vit. D3 is formed in the skin by UV-rays (ultraviolet rays).

Deficiency signs : Vit D deficiency produces rickets in children & osteomalacia in adults. In tropical countries with plentiful

sunlight, these diseases are prevalent, only in such sections of the population as are deprived of the opportunity of sufficient exposure of the sun-young children employed in cottage industries, regular night shift workers in factories & newspaper press, women (particularly pregnant).

In Rickets, fall in intestinal absorptions of Ca^{++} & Phosphate rise in Urinary $PO_4^{=}$, loss of bone-Ca^{++}, softeness & deformities of bones (like bow-legs & pigeon chest) and delay in the closer of fontanelle are observed. The cartilage cells at the ends of lung bows continue to grow & proliferate, formation of new bone-matrix also continues. But ossification of the cartilage is delayed & the large cartilage tissue at the end. of long bones produces swellings & pain at the wrist, ankle, & sternal ends of ribs. Enamel of teeth becomes thin & shows pits & other defects. Negative Ca^{++} balance results, but serum- Ca level may often be prevented by the parathyroid hormone from falling low.

Osteomalacia is characterized by a fall in intestinal absorptions of Ca^{++} & $PO_4^{=}$ rise in urinary phosphate-ve-Ca^{++} balance, muscular weakness loss of bone-Ca^{++}, softness, pain, deformities & easy fracture of bones, pelvic deformation, bending vertebral column & bowed legs,

Symptoms of both the diseases may be cured by vit. D administration except when there is a genetic failure of the renal formation of 1,25 DHCC (1,25 - $(OH)_2$ - D_3)

Both vitamins D_2 & D_3 are antirachitic for man. Dihydrotachysterol & irrediated 7 dehydrositosteril (Vit. D5) also have anti-rachitic activities.

Chronic renal failure may also cause a failure of 1,25 DHCC formation leading to a non-inherited vitamin D resistant rickets or Osteomalacia. Functions: Vit. D regulates Ca & P metabolism by influencing bones, Kidneys, intestinal mucosa, avian shell glands.

1) Vit. D Promotes intestinal absorption of Ca^{++} & $Po_4^{=}$. 1,25 DHCC is merely thrice as effective as 25 HCC & the latter is about

twice as potent as cholecalciferol in this respect. A full in blood-Ca^{++} may stimulate parathyroids to secrete more parathormone (PTH) which may influence the kidney increasing the conversion of 25 HCC to 1,25 DHCC. The latter is carried by blood to the intestinal mucosal cells where it probably combines with a specific citosol protein (mobile receptor). This protein DHCC complex is to probably translocate to nucleus, there 1,25 DHCC may induce the transcription of such mRNA as may synthesize a specific Ca - binding protein then helps in the active Ca^{++} transport across the microvillus border of the cells against an electro chemical gradient, each molecule of the protein combining probably with one Ca^{r+} during the process. Thus, Ca^{++} absorption is increased.

2) Vit. D also promotes resorption of bone & mobilization of calcium from bones. 1,25DHCC is more than 30 times as effective as 25HCC in this respect. Here also, 1,25 DHCC may stimulate the transcription of such mRNA in osteoclasts as may synthesize calcium transport proteins, thus calcuim transport from the bone cell is increased. Vitamin D metabolites augment the effect of parathormone on bone resorption - the hyper calcaemic effect of parathormone is subnormal in rickets or Osteomalacia, The bone resorptive effect of Vit. D causes a rise in serum Ca^{++} & Pi levels.

3) When the blood Ca" is low, 1,25 DHCC is formed in the kidney from 25 HCC and reduces the urinary excretions of calcium & Phosphates by promoting their reabsorptions by the proximal tubule cells of kidney. But when blood Ca^{++} is normal or high, kidney produces 21,25 DHCC instead of 1,25 DHCC; 21,25 DHCC raise urinary Ca^{++}, but has little effect on intestinal absorption of Ca^{++} 24-25 DHCC also influences tubular reabsorptions of Ca^{++} & $PO"_4$.

4) Active metabolites of Vit. D increase the activity of alkaline phosphatase in bone cells &, microvillus borders of intestinal epithelial cells, probably through the induction of synthesis of

such mRNA as synthesizes that enzyme. This enzyme increases phosphate ion concentration in bones & facilitates mineral deposition there.

5) Vit. D increases incorporation of sulphur in chondroitin sulfate molecules and thus enhances mucopolysaccharide synthesis in bone & tooth matrices, as mucopolysaccharides participate in calcification by first concentrating & then liberating calcium in those matrices, Vit. D facilitates mineralization of these tissues.

6) Vit. D may enhance the activities of phytase and a Ca^{++} dependent ATP-ase in the intestinal mucosa of rats & chicks.

According to a modern concept of Deluce and others, Vit D3 is a unique prohormone which is not only synthesized in the body but also obtained from food; 1,25 DHCC is considered in its turn as the active hormone formed from that prohormone in a specific organ (kidney) under the stimulating action of parathormone which may be considered as a trophic hormone in this respect. Line other hormones, 1,25 DHCC is also carried by blood to diverse target organs like bones, intestine & kidneys, In many target organs 1,25 DHCC behaves almost like steroid hormones in influencing the transcription of mRNA in cell nuclei by acting according to the mobile receptor model.

Hypervitaminosis-D: When excessive amounts of Vit. D taken for long time, have produced hypercalcemia in infants and nephrocalcinosis in both infants & adults. Continued excessive intake of Vit. D may cause sudden loss of appetite nausea, intense thirst, irritability, depression excessive mobilization of bone-$Ca''^{1}\wedge$ into blood, hypercalcemia, hypotonicity of muscles, abnormal bone development, poor body growth, renal calculi in the kidney tubules, etc. It may produce serious & sometimes fatal results.

(18). Describe the sources, deficiency signs, and functions of i vitamin C.

Vitamin - C (Ascorbic Acid)

Sources : Vegetable sources : Fresh fruits, mainly citrus

fruits, e.g., emblic myrobalan (amlaki), orange, lemon, tomato, pine-apple, papaya, etc. fresh vegetables, e.g., cabbage, cauliflower, lettuce, spinach, green peppes beans, etc., properly sprouted pulses, germinating grains, etc. are also rich sources. Potato & seeds are poor, but rich during germination.

Animal sources : Generally poor. In animal body the adrenal cortex containing a good amount. Cow's milk, meat, & fish contain a little. Blood serum less. It is not much stored in the body. Most of the ingested vit. Cis promptly excreted although vit. C is high threshold substance. Daily excretion in urine is 30-50 mgm. The aqueous & vitreous humors are very rich in it.

Deficiency signs : scurvy 1) Increased fragility of capillaries causing haemorrhages - under the skin, periosteum, intestine, kidneys, etc. The gums show erosion of the mucous memb. at their margins & due to increased fragility of the capillaries there is frequent bleeding 2) Malformations of bones and teeth. The osteoblasts remain functionless. Many of them are reverted to fibroblasts. There is irregular deposition of bone salts & decreases in density of long bones. Teeth also show similar changes. The functions of the odontoblasts are impaired. New dentine is not formed & the tissue becomes spongy and porous. 3) Increased brittleness of bones leading to fracture. 4) Anaemia-number of R.B.C. & platelets are reduced. 5) Delayed bl. clotting & clot retraction. 6) skin erruptions. 7) Increased susceptibility to infections. 8) Impaired healing of wounds. 9) Disturbance of carbohydrate metabolism. 10) Reproductive failure both in males & in females.

Functions of Vit. C:

1. Ascorbic acid helps in the deposition and maintenance of intercelluar substances in mesenchymal tissues and thus prevents the symptoms of scurvy (antiscorbutic vitamin).

2. Vit. C may be related to the synthesis of hydroxyproline & hydroxy lysine, from proline & lysine respectively, in collagen molecules. In scurvy, hydroxyproline content of collagen may

become subnormal. It is believed that protocollagen proline hydroxylase and protocollagen lysine hydroxylase may catalyze with the help of ascorbic acid, Alfa-ketogluterate, Fe++ & molecular oxygen. Some authorities believe that monodehydroascorbic acid, produced by oxydation of ascorbic acid, may be the active vit. C metabolite participating in hydroxylations. In scurvy, failure of conversion of procollagen to collagen due to the failure of hydroxylation may lead to a rapid destruction of the collagen interme diate this causes defects of intercellular substances & produces may symptom of scurvy.

3) Formation of carnitine in the liver by hydroxylation of Gama-butyrobetaine is also helped by ascorbic acid, Gama-Ketoglutarate, Fe^{+H} and a deoxygenase.

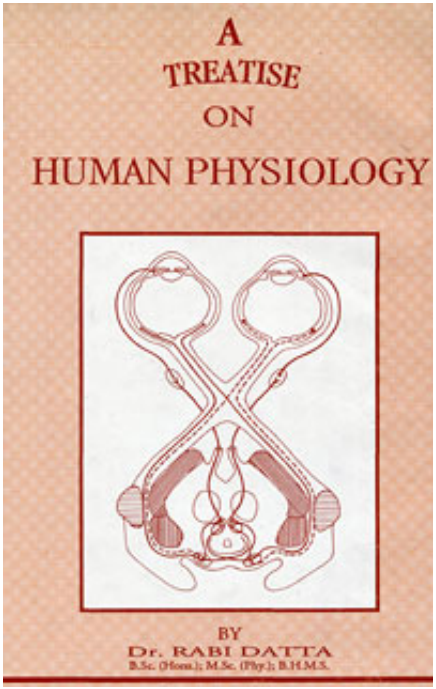
4) Vit. C helps in the action of Alfa -hydroxylase or monoxygenase which catalyzes the Alfa-oxidation of long chain fatty acids to form alfa-hydroxy fatty acids.

5) Vit. C may have some role in steroids hormone synthesis probably in some hydroxylase reactions. Adrenal cortex & corpus luteum both of which secrete steroid hormones, contain much vit. C. Moreover, the ascorbate level of adrenal cortex falls when steroid secretion rises, during stress and fatigue & on injecting ACTH or bacterial toxins.

6) The vitamin may help in the normal metabolism of phenylalanine & tyrosin, as a co-factor for p-hydroxy phenylpyruvate hydroxylase, thus ascorbate participates in hydroxylation & conversion of p-hydroxyphenylpyruvate to homogentisic acid. Vit. C may also be a co-factor for homogentisate oxidase which oxidizes homogentisic acid. Ascorbate deficiency sometimes causes abnormal urinary excretions of homogentisic acid, p-hydroxyphenylpyruvate & other intermidiates of tyrosin metabolism.

7) Vit. C serves as a co-factor for dopamine Beta-hydroxylase and thus, helps in the hydroxylation of dopamine to norepinephrine.

8) Vit. C may take part in oxidation reductions in tissues



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