THE STRETCH REFLEX AND SKELETAL MUSCLE TONE

The stretch reflex is the contraction of a skeletal muscle in response to passive stretch. It is also called *the myotatic or muscle spindle reflex (MSR)*





Structure of the muscle spindle

Muscle spindles are fusiform stretch receptors present in the *fleshy parts* of skeletal muscles parallel to the muscle fibres which are called extrafusal fibres (figure 36). Each spindle consists of several small muscle fibres called intrafusal fibres enclosed in a connective tissue capsule that is attached to the sides of the extrafusal fibres. The central parts of these fibres are non-contractile and constitute the receptor areas of the spindles. On the other hand, their peripheral parts are contractile and when they contract, they stretch the central receptor areas. There are 2 types of intrafusal muscle fibres, which are the following (figure 37):

(1) Nuclear bag fibres : These have a *dilated central area* filled with nuclei and there are typically 2 of these fibres per spindle.

(2) Nuclear chain fibres : These also have multiple nuclei but they are arranged as a *chain in the receptor area*. They are attached to the sides of the other type, and there are 4-8 of these fibres per spindle.



Innervation (nerve supply) of the muscle spindles

(A) Afferent nerves arising from the spindles

(1) Type la nerve fibres : These are type *A alpha fibres* that are thick (average diameter 17 microns) and rapidly-conducting (velocity of conduction 70-120 meters / second). They arise from the receptor areas of *both the nuclear bag and nuclear chain muscle fibres*, where their endings wrap round the fibres forming *primary* (= *annulo-spiral*) *endings*.

(2) Type II nerve fibres : These are type *A beta fibres* that are *thinner* and slower in conduction than the Ia fibres (average diameter 8 microns). They arise from secondary (= flower-spray) endings at the sides of the primary endings in the nuclear chain fibres only.

(B) Efferent nerves supplying the spindles (gamma efferents)

The peripheral (contractile) parts of the intrafusal fibres are supplied by *thin myelinated* motor nerve fibres called *gamma efferent nerves* (= Leksell nerves). These are type *A gamma fibres* (having diameter 3-6 microns) that are the *axons of small anterior horn cells* called the *gamma motor neurons* (figure 36). They form about 30 % of the efferent nerve fibres in the ventral roots (so they are called the *small motor nerve system*), and are 2 types :

(1) Gamma-d (dynamic) fibres : These supply the *nuclear bag fibres*, where they end by *plate endings* (figure 37).

(2) Gamma-s (static) fibres : These supply the *nuclear chain fibres*, where they end by *trail endings* (figure 37).

** Large A beta efferent nerves also supply the intrafusal fibres (figure 37).

Nervous pathway of the stretch reflex

Impulses from the muscle spindles are transmitted to the CNS by its fastconducting afferent nerve fibres. These proceed *directly without intervening interneurons* to the ventral horns (figure 36) where they excite the *alpha motor neurons* that supply the stretched muscle (by releasing *glutamate*). Impulses are then transmitted by the alpha motor neurons to the stretched muscle leading to contraction of its extrafusal fibres.

Therefore, the stretch reflex arc contains only *one synapse*, and it is *probably the only monosynaptic reflex* in the body. Its *reaction time* (*or total reflex time*) is short (19- 24 milliseconds) and its central delay does not exceed 0.9 millisecond (proving that it is monosynaptic).

Mechanism of stimulation of the muscle spindles

The adequate stimulus for excitation of the muscle spindles is *stretch*, and this can be produced by either *passive stretch of the whole muscle* or stimulation of the *gamma efferent fibres*. The latter cause contraction of the peripheral parts of the intrafusal fibres, which stretches their central parts and the resulting muscle contraction is said to occur via a **gamma-spindle loop**.

Function of the muscle spindles

The muscle spindles constitute a feedback mechanism that *maintains* the muscle length constant. Elongation (stretch) of the muscle excites the muscle spindles, which leads to contraction and shortening of the muscle. On the other hand, if the muscle is shortened, the discharge of the muscle spindles decreases, which leads to relaxation and elongation of the muscle. The latter response is sometimes called *negative stretch reflex* (see below).

Responses of the muscle spindles to stretch

(1) Dynamic response : This occurs while the muscle length is *increasing*, and it informs the CNS about the *rate of change of muscle length*. It is produced mainly as a result of stretch of *the nuclear bag fibres*. The response is an increase of the rate of discharge from the *primary endings* in these fibres, which is followed by a marked decrease when the new length is maintained (because these receptors are *rapidly-adapting*). (2) Static response : This occurs while muscle stretch is *maintained*, and it informs the CNS about *changes of the muscle length*. It is produced mainly as a result of stretch of *the nuclear chain fibres*, and the response is an increase of the rate of discharge from *the primary and secondary endings* in these fibres, which continues as long as the new muscle length is maintained (because these receptors are *almost non-adapting*).

TYPES OF THE STRETCH REFLEX

(1) Dynamic stretch reflex : This is initiated by sudden stretch of the muscle and the response is a *brief strong contraction* that ends rapidly because it occurs *as a result of the dynamic response* of the muscle spindles (see above). It is the basis of the *tendon jerks* (page 66).

(2) Static stretch reflex : This is initiated by steady stretch of the muscle and the response is a *continuous contraction as long as the stretch is maintained* because it occurs *as a result of the static response* of the muscle spindles. It is the basis of the *skeletal muscle tone* (page 66).

(3) Negative stretch reflex : This is initiated by *muscle shortening*, and the response is *muscle relaxation and its elongation to its resting length* due to reduction of discharge from the muscle spindles.

(4) Inverse stretch reflex : This is initiated by *overstretch* of the muscle, and the response is *muscle relaxation through activity of the Golgi tendon organs* (page 65).

(5) Cerebellar stretch reflex (page 109).

THE GAMMA EFFERENT SYSTEM

FUNCTIONS OF THE GAMMA EFFERENT NERVES

Stimulation of these nerves leads to stretch of the central parts of the muscle spindles, which *increases the sensitivity of the muscles to stretch and may result in reflex muscle contraction*.

CONTROL OF GAMMA EFFERENT DISCHARGE

The gamma motor neurons are controlled by signals discharged from : (1) Certain supraspinal areas (page 71) : These discharge *facilitatory and inhibitory signals through the descending tracts* (figure 36). Such discharge adjusts the stretch reflex in skeletal muscles, which is important for appropriate control of movements and posture. Also, *anxiety is often associated with increased gamma efferent discharge* (by supraspinal facilitatory signals), which causes exaggerated tendon jerks in anxious persons (page 69)... Chapter 5 Alpha- gamma linkage & functions of the stretch reflex

(2) The skin : Noxious stimulation of the skin increases the gamma efferent discharge to the flexor muscles, which potentiates the withdrawal reflex.

(3) The skeletal muscles : Signals from skeletal muscles also increase the gamma efferent discharge as shown in the *Jendrassik maneuver* (page 68).

Alpha gamma linkage (or coactivation)

Whenever the alpha motor neurons are activated (whether by supraspinal signals or by impulses discharged from skeletal muscles) the gamma motor neurons are activated at the same time. The *role of gamma efferent coactivation is to prevent relaxation of the muscle spindles during extrafusal muscle contraction, and to maintain them capable of adjusting the alpha motor neuron discharge throughout the movement.*

FUNCTIONS OF THE STRETCH REFLEX

(1) Maintenance of the erect posture against the force of gravity : This occurs through producing a *strong muscle tone in the antigravity muscles*.

(2) Damping (smoothing) function : The signals discharged to a muscle usually have varying intensities, and this would result in incoordinated movements. However, the signals are adjusted through the *alpha-gamma linkage* so that smooth movements are produced (= *signal averaging*).

(3) Increasing the power of muscle contraction : As a result of the *alpha-gamma linkage*, both the extrafusal and intrafusal fibres contract when a muscle is stimulated. The intrafusal fibres elicit a stretch reflex by the *gamma-spindle loop mechanism* (page 61), which results in a more powerful contraction of the extrafusal fibres (= servo-assist function).

H REFLEX (= HOFFMANN'S REFLEX)

This is a *stretch reflex that is produced experimentally* by electric stimulation of the Ia afferent fibres from a muscle. It is usually elicited by electric stimulation of the popliteal nerve, and the response is recorced from the calf muscles by the electromyograph.





THE SKELETAL MUSCLE TONE

DEFINITION

The skeletal muscle tone is a state of continuous mild or partial (or subtetanic) contraction of skeletal muscles *during rest*.

MECHANISM

It is a *static type of the stretch reflex* (page 63) that is produced as a result of continuous mild stretch of skeletal muscles during rest by the *series elastic elements* present in the tendons (refer to muscle and nerve)..

DISTRIBUTION

It is present in *all skeletal muscles*, but specially in the *antigravity muscles* (because they are subjected to more stretch by the force of gravity). These muscles include (1) *Extensors of the lower limbs* (2) *Flexors of the upper limbs* (3) The *muscles of the back and back of neck* (4) The *elevators of the lower jaw*.

FUNCTIONS OF THE SKELETAL MUSCLE TONE

(1) It is essential for maintenance of the *erect posture*.

(2) It helps both the *venous return and lymph flow* from the lower limbs (against the force of gravity).

(3) The abdominal muscles' tone prevents visceral ptosis.

(4) It is an *important source of heat production*, so it is markedly increased on exposure to cold (refer to energy metabolism).

THE TENDON JERKS (TENDON REFLEXES)

A tendon jerk is the response of a skeletal muscle to sudden stretch produced by tapping its tendon *sharply and strongly by a medical hammer*. It is a *dynamic type of the stretch reflex* (page 63), and it consists of rapid contraction of the muscle followed by rapid relaxation.

Examination of tendon jerks

The limb is placed in a position at which the muscle under test is mildly stretched, and its tendon is struck briskly with the hammer. The commonly tested jerks include the following :

(1) Knee (or patellar) jerk : The leg is *semiflexed* at the knee joint by asking the subject to place the leg to be tested crossing over the other leg (figure 41) or by placing the leg in the dependent position (figure 44). The *patellar tendon is palpated and struck sharply just below the patella*. The response is contraction of the *quadriceps femoris muscle* resulting in extension of the knee and jerking of the leg forward. Its centre is the 2nd, 3rd and 4th lumbar segments.

THE GOLGI TENDON ORGANS (GTOs)

These are the receptors present in the tendons of skeletal muscles (figure 38). Each GTO consists of *a netlike collection of knobby nerve endings* that give rise to thick myelinated type **Ib afferent nerve fibres** (which are a type of A alpha fibres) that have a diameter of about 16 microns.

A small bundle of muscle fibres is *connected in series with each GTO*, and the GTO is stimulated by the tension developed in that bundle. Therefore, the GTOs are *tension receptors* (i.e. they *detect muscle tension*), and they are stimulated by *both passive stretch as well as by active contraction of skeletal muscles*. They are *slowly-adapting* and are *not under nervous control because they do not receive efferent nerve supply*.

Effect of stimulation of the GTOs

Signals from the GTOs excite inhibitory interneurons called *Golgi bottle neurons* (page 51) which produce IPSPs at both the alpha and gamma motor neurons. Therefore, such *Golgi tendon reflex is disynaptic* (figure 39) and leads to *relaxation of the muscle from which it originates*.



Figure 39 : The nervous pathways of the stretch and inverse stretch reflexes.

Responses and functions of the GTOs

As in muscle spindles, the GTOs have *dynamic and static responses*. The former occurs when the muscle tension suddenly increases and it terminates rapidly while the latter occurs when the increased tension is maintained. The

main function of the GTOs is *maintenance of a constant muscle tension* by a *negative feedback mechanism* (i.e. if the muscle tension increases, the GTOs are stimulated resulting in muscle relaxation and reduction of its tension, and vice versa).

The inverse stretch reflex (autogenic inhibition)

This is *reflex relaxation of a muscle in response to excessive stretch*. It is an inhibitory reflex that occurs if the muscle tension markedly increases. It is initiated by *excitation of the GTOs*, and *is a protective reaction against tearing of the muscle or avulsion of its tendon from its bony attachment*.

The lengthening reaction (= clasp knife effect)

This reaction is obtained in *spastic (or hypertonic) muscles e.g. in upper motor neuron lesions.* It is demonstrated by flexion of a patient's limb at its main joint by means of the examiner e.g. *the lower limb at the knee joint* (figure 40). The reaction consists of *muscle contraction upon moderate stretch of the quadriceps muscle followed by sudden muscle relaxation upon overstretch*, and it occurs as follows : As the limb is flexed, the *quadriceps femoris muscle is lengthened* (so it is called the lengthening reaction) and a resistance is encountered due to contraction of this muscle as a result of the stretch reflex. However, with sustained flexion, *the inverse stretch reflex is initiated*, so the initial resistance suddenly disappears and the limb gives up and flexes easily, as *occurs during closing a pocket knife* (so it is also called the *clasp knife effect*).

<u>**</u> When the examiner tries to extend the limb after its flexion, he will also initially find resistance then the limb suddenly gives up and extends easily. This is due to a *sequence of stretch and inverse stretch reflexes that occurs in the hamstring (flexor) muscles*. This effect is sometimes called the **shortening reaction** (referring to shortening of the extensor muscles).





(11) The *static type is inhibited by excessive stretch* via the inverse stretch reflex (see above).

(12) The contraction is jerky in the dynamic type while it is smooth in the static type (because in the latter, the spinal motor neurons discharge at a *low-frequency and also alternate their activity*). In addition, the O_2 consumption during the reflex is low, so the static type is also not rapidly fatigued.

The following table shows the differences between the static and dynamic types of the stretch reflex :

	DYNAMIC STRETCH REFLEX	STATIC STRETCH REFLEX
Stimulus	Sudden stretch (by tapping on tendons)	Maintained stretch (e.g. by gravity)
Afferent	Type la fibres	Type II fibres
Response	Brisk contraction and rapid relaxation	Maintained smooth contraction
Function	Has clinical significance only	Production of muscle tone
Adaptation	Rapidly-adapting	Slowly-adapting
Fatiguability	Rapidly-fatigued	Slowly-fatigued
Enhancement	Enhanced by Jendrassik's and similar maneuver	Not affected by Jendrassik's and similar maneuvers
Existence	only elicited clinically (by a hammer)	exists normally in all muscles specially antigravity muscles

HIGHER CONTROL OF THE STRETCH REFLEX

(A) SUPRASPINAL FACILITATORY AREAS

(1) The facilitatory reticular formation : This is a wide *active area* that *discharges spontaneously by an intrinsic activity*. It is present mainly in the *pons* and its signals reach the spinal cord through the *ventral reticulospinal tract*. It facilitates the stretch reflex *mainly by activating the gamma motor neurons*, and *almost all other facilitatory areas stimulate it*.

(2) The primary cortical motor area (area 4) : This discharges facilitatory signals to the *alpha motor neurons through the corticospinal tract*.

(3) The vestibular and inferior olivary nuclei : These stimulate the facilitatory reticular formation and *also discharge direct facilitatory signals to the alpha motor neurons* through the vestibulospinal and olivospinal tracts.

(4) The caudate nucleus and neocerebellum : These stimulate the facilitatory reticular formation as well as the vestibular and inferior olivary nuclei.

(B) SUPRASPINAL INHIBITORY AREAS

(1) The inhibitory reticular formation : This is a small *inactive area* (i.e. having *no intrinsic activity*) present mainly in the *medulla oblongata*. It is *activated by signals from the other inhibitory areas*, and its signals reach the spinal cord through the *lateral reticulospinal tract*, where they *inhibit mainly the gamma motor neurons*.

(2) Certain cortical areas : These include mainly the *premotor area* $(= area \ 6)$ and area 4 S $(= main \ cortical \ suppressor \ area$). These areas activate the inhibitory reticular formation both directly and through stimulating the lenticular nucleus of the basal ganglia (see below).

(3) The red nucleus (in the midbrain) : This nucleus discharges *inhibitory signals to the alpha motor neurons through the rubrospinal tract*.

(4) The lenticular (or lentiform) nucleus and paleocerebellum : These activate the inhibitory reticular formation and *inhibit the vestibular nucleus*.

** The main facilitatory tracts are the ventral reticulospinal, the vestibulospinal and the corticospinal tracts, while the main inhibitory tracts are the lateral reticulospinal and the rubrospinal tracts. The reticulospinal tracts terminate at the gamma motor neurons while the other tracts terminate at the alpha motor neurons.

<u>**</u>. Normally, the net effect on the alpha motor neurons is facilitatory while the net effect on the gamma motor neurons is inhibitory. The latter effect is largely due to the inhibitory effect of cortical areas 6 and 4 S (which is much greater than the facilitatory effect of area 4 on the alpha neurons).

DECEREBRATE RIGIDITY (DR)

This is a state of hypertonia that occurs in decerebrate animals (page 55). In these animals, the midcollicular transection *abolishes the inhibitory effects of both the cortical areas 6 & 4 S and the lenticular nucleus*. Accordingly, *the net effect at the gamma motor neurons becomes facilitatory* (which is a *release phenomenon*). This *increases the discharge of these neurons*, resulting in a type of rigidity called *gamma rigidity* (see below). Such rigidity is produced through the *gamma spindle loop mechanism* (page 61), so *muscle deafferentation in this case will decrease the rigidity*.

Gamma rigidity and alpha rigidity

Gamma rigidity (= spasticity clinically) refers to muscle stiffness that occurs as a result of increased spinal motor activity with *relative overexcitation of the gamma motor neurons* e.g. in DR and UMNL (page 80). These

(B) CENTRAL REFLEXES

(1) **Conditioned reflexes :** These are acquired (i.e. develop by learning) and are integrated in the cerebral cortex.

(2) Unconditioned reflexes : These are inherent (or inborn) i.e. occur without learning, and include (a) *Hypothalamic reflexes* (regulate many functions e.g. body temperature and water balance) (b) *Midbrain reflexes* (mediate postural reflexes and most visual reflexes) (c) *Medullary reflexes* (mediate cardiovascular, respiratory and digestive reflexes) (d) *Spinal reflexes* which include superficial, deep and visceral reflexes.



Figure 32 : Method of eliciting the plantar reflex (left) and the abdominal and cremasteric reflexes (right).

THE SUPERFICIAL (CUTANEOUS) REFLEXES

These are polysynaptic reflexes that *require facilitation by the pyramidal system*. They include the following reflexes :

(1) The plantar reflex : Scratching the outer (lateral) edge of the sole (figure 32) by a blunt object (e.g. a key) causes plantar flexion of all toes in *normal awake adults and infants more than one year of age*. Such response is changed in many conditions into the *Babinski's sign* (page 82) and its centre lies in L_5 , S_1 and S_2 segments of the spinal cord (mainly the first sacral segment).

(2) The abdominal reflexes : Striking the abdominal skin lightly (e.g. by a pin) leads to contraction of the underlying muscles, as indicated by movement of the umbilicus (figure 32). They are a type of the withdrawal reflex (see below), and their centres lie in *the* 7^{th} *to the* 12^{th} *thoracic segments of the spinal cord* (depending on the site of stimulation).

(3) The cremasteric reflex : Striking the skin at the medial side of the upper part of the thigh *in males* causes contraction of the cremasteric muscle and upward retraction of the testis on the same side (figure 32). It is a type of the withdrawal reflex, and its centre lies in the *first and second lumbar segments* of the spinal cord.

(4) The withdrawal (flexor) reflex : This is a protective powerful reflex (because it inhibits other reflexes occurring at the same time). Noxious stimulation of the skin (e.g. at a limb) leads to contraction of the flexor muscles of that limb and its withdrawal away from the stimulus (figure 33).

<u>**</u> The **pin is used** for eliciting the abdominal, cremasteric and flexor reflexes, in addition to *testing of pain sensation* and demonstration of the *triple response* (refer to circulation).

(5) The crossed extensor reflex : This is reflex extension of a limb during flexion of the other limb as a result of a withdrawal reflex (figure 33). It occurs with strong noxious stimuli, and is *supportive in function*.

(6) The anal reflex : Scratching the skin around the anus leads to contraction of the external anal sphincter. Its centre lies in *the third and fourth sacral segments of the spinal cord*.

(7) The positive supporting reflex (reaction) : Applying pressure to the *sole* (e.g. the pressure exerted by the body weight during standing) leads to contraction of *both the flexor and extensor muscles* of the lower limbs. It is the *only reflex that does not obey the principle of reciprocal innervation* (page 56). Its centre extends *from the first lumbar segment to the first sacral segment of the spinal cord*, and during standing, it renders the lower limbs to act as 2 solid pillars that support the body against gravity.

Extension of the limb occurs in the direction of pressure applied to the sole i.e. if pressure is applied to the lateral side of the sole, it leads to extension and abduction of the lower limb, while if it is applied to the medial side, it leads to extension and adduction of the lower limb. This effect has been called **the magnet reaction**.

(8) The scratch reflex : This is initiated by the sensation of itch particularly when caused by *multiple tactile stimuli* (e.g. the reflex initiated by a crawling insect). It can also be produced experimentally by stimulating the skin with a weak faradic current, and it results in rhythmic scratching movements to remove the irritant stimulus (and sometimes production of pain which also relieves the effect of the irritant stimulus).

<u>**</u> The corneal reflex is a protective superficial (but not a cutaneous) reflex. Touching the cornea of one eye (by *a piece of cotton*) causes contraction of the *orbicularis oculi muscles on both sides*, resulting in *bilateral blinking*. The afferent impulses are conducted by the *trigeminal nerve*, while efferent impulses are conducted by the *facial nerves*. Its examination is important to check the *integrity of the trigeminal nerve* (page 28).

rhythm is replaced by rapid irregular low-voltage *beta waves* (figure 78). It represents *breaking up of the synchronized neuronal alpha activity*, so it is also called *alpha block or desynchronization*.

Such response is *reversible* (so if the eyes are closed, the alpha rhythm is resumed). It is due to stimulation of the **ascending reticular activating system** (= ARAS, see below).Such system can also be stimulated by cortical signals discharged via *corticofugal fibres* (providing a pathway through which certain cortical events can initiate arousal e.g. *during emotions*).



Figure 78 : Arousal or alerting response (alpha block or desynchronization).

CLINICAL SIGNIFICANCE OF THE EEG

(1) It helps in determining the sites of focal pathological processes in the brain e.g. the *sites of tumours* (in which the EEG waves are distorted) or the sites of fluid collection e.g. *a subdural hematoma* (in which the EEG waves are damped).

(2) It helps in diagnosis of certain brain diseases, particularly *grand mal and petit mal epilepsy* (each of which causes characteristic EEG changes).





THE RETICULAR FORMATION

This is a network of neurons located in the brain stem, extending *upwards* to the diencephalon (thalamus, hypothalamus and subthalamus) and *downwards to the upper part of the spinal cord* (figure 79), where it merges with

its intemeurons. Many nuclei and centres are present within its meshes (e.g. *the respiratory and cardiac centres, the substantia nigra, and the red, vestibular and raphe nuclei*). It is divided into **sensory and motor parts**.

(A) THE SENSORY PART OF THE RETICULAR FORMATION

This consists of small neurons that have multiple interconnections with each other (which allows for convergence, divergence and after discharge). It receives a *rich sensory input (afferent fibres)* from (1) All ascending lemnisci (2) The visual, auditory and olfactory nervous pathways (3) The basal ganglia (4) The cerebellum (5) The cerebral cortex (via *corticofugal fibres*) (6) The hypothalamus (7) The vestibular apparatus.

(B) THE MOTOR PART OF THE RETICULAR FORMATION

This consists of large neurons which receive signals from the sensory part and *their axons constitute the output (efferent) fibres* from the reticular formation. It contains **facilitatory and inhibitory parts**:

(1) Facilitatory (excitatory) reticular formation :

This is located mainly in the *pons*. It has an *inherent activity* and the axons of its neurons divide into 2 branches :

(a) An ascending branch, which excites thre cerebral cortex, and is called the *Ascending Reticular Activating System or ARAS* (see below).

(b) A descending branch (= *Ventral reticulospinal tract*) which exerts a facilitatory effect on the spinal gamma motor neurons (page 71).

(2) Inhibitory reticular formation :

This is located mainly in the *medulla oblongata*. It has *no inherent activity*, and its axons descend as *the lateral reticulospinal tract*, which inhibits the spinal gamma motor neurons (page 72)

FUNCTIONS OF THE RETICULAR FORMATION

(1) Control of the *level of consciousness* through the ascending reticular activating system (see below).

(2) Regulation of the *stretch reflex and muscle tone* through the reticulospinal tracts.

(3) Pain inhibition by the raphe magnus nucleus (page 35).

(4) Control of sleep by 2 specific centres in its meshes (see below)

(5) *Control of visceral functions* (e.g. cardiac activity) by controlling the spinal lateral horn cells.

ASCENDING RETICULAR ACTIVATING SYSTEM (= ARAS or RAS)

This is a *multineuronal polysynaptic system* of nerve fibres that originate at the *facilitatory reticular formation*. Its fibres extend upwards,

then some project directly to the cerebral cortex, while the majority relay first at the *nonspecific thalamic nuclei*, from which other fibres arise and project diffusely to *almost all parts of the cerebral cortex* (figure 79). The latter pathway is called the *reticulo-thalamo-cortical pathway*.

FUNCTIONS OF THE ARAS

The ARAS *controls the electric activity of the cerebral cortex*, and is concerned with *consciousness and production of the alert response*, so reduction of its activity leads to sleep (see below).

FACTORS THAT AFFECT THE ACTIVITY OF THE ARAS

(A) Factors that increase the ARAS activity

(1) Sensory signals (specially pain).

(2) Signals from the cerebral cortex (via the *corticofugal fibres*) which increase alertness and resist the desire to sleep (e.g. during emotions and voluntary movements).

(3) Certain drugs called the *analeptic drugs* e.g. catecholamines, amphetamine and caffeine.

(B) Factors that decrease the ARAS activity

(1) Reduction of signals from the sensory pathways or the cerebral cortex.

- (2) Stimulation of the sleep centres (see below).
- (3) Extensive damage of the ARAS (e.g. by tumours).

(4) General anesthetic drugs : These drugs lead to unconsciousness through *depressing the ARAS activity* (by inhibiting the synaptic transmission between its neurons).

SLEEP

Sleep is *a physiological state of temporary unconsciousness*. Its duration varies *inversely with age* (average 18 hours in infants, 8 hours in adults and 6 hours in old persons). The sleep / wakefulness 24-hours rhythm is determined mainly by *synchronization with the 24-hours light / dark cycle* (see below). However, it is also affected by various habits and conditioned reflexes, as well as by many psychological and physical factors.

PHYSIOLOGICAL CHANGES DURING SLEEP

(1) Circulatory system : The heart rate, cardiac output, vasomotor tone and arterial blood pressure are all decreased and the circulation time is prolonged
(2) Respiratory system : The rate and depth of respiration are decreased (so pulmonary ventilation is decreased with a tendency to *acidosis*) and *periodic breathing may also occur* (refer to respiration).