Movement skill and its disorder

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Introduction

There are three neuronal control fmechanisms or movement ; reflex, automatic and voluntary movement. Reflex occurs at the spinal cord level (Figure 93). Automatic movement is induced at the level of the brain stem (Figure 94). Voluntary movement is mainly controlled by the cerebral cortex (Figure 95). Expression of voluntary movement is an integrate system with complex mechanisms (Figure 96) organised as follows:

After informatic processing (initiation, planing and programing)in the brain, the 'out put' information is transmitted to the muscle via the spinal cord (extrapiramidal tract) and motor neuron (Figure 95).

Mechanism of muscle contraction

Motor neuron excitation induces a release of acetylcholine from the nerve terminal. After binding to acetylcholine receptor, excitation of plasma membrane of muscle fiber causes mobilization of Ca^{2+} from the sacoplasmic reticulum. Ca^{2+} binds to the troponin receptor on the actin filament, and then the head of the myosin filament touchs the actin molecule. The myosin head is moved by hydrolysis reaction from ATP to ADP (Figure 97). This sliding phenomenon causes muscle contraction.

ATP originates mainly from glucose ($C_6H_{12}O_6 + 6O_2$--- $6CO_2 + 6H_2O$ + 38 ATP) and fatty acid. (Figure 98) Accordingly, nutritional intake is important for athletes, especially in endurance sports. A carbohydrate meal taken 2 or 3 days before the race contributes to glycogen accumulation (store) in the muscle (Table 10).

Dysfunction diseases of the motor system

Parkinson's disease: Parkinson's disease causes dysfunction of motor behavior, by depletion of dopamine neurotransmitter in the striatum. Another possibility could arise from the observation that pharmacological destruction of the striatum by 5,7 DHT (5-HT neurotoxin), results in abnormal rolling movement in animals (Figure 99), from which they can recover by 5-HTP (5-hydroxytryptophan; 5-HT precursor). This suggests the significance of the serotonergic system of basal ganglia, as well as dopaminergic system.

Motor neuron disease: This disease causes muscle shrinkage and body paralysis. The reason may be induced by point mutant of SOD 1 (superoxide dismutase 1) gene. As a result, an increase of free radical is a possible mechanism for muscle defect.

Progressive muscular dystrophy: There is a high ratio of male children patients who have Duchenne, rather than Becker type muscular dystrophy. The reason for this is deficiency of dystrophin gene in the X chromosome (Figure 100). It causes cell death of heart and intercostal muscle until twenty years old.

Myotonia: This disease prevents muscle reverting back to relaxation after contraction; a prolonged abnormal phenomenon during muscle contraction. This is abnormal function is of the plasma membrane in the muscle fiber cell.
The abnormal gene is known to be 19q13.2.

Myasthenia gravis: Deficiency of muscle strength causing early fatigue. This is due to abnormal transmission in the neuro-muscular junction. The reason being abnormal mechanisms of acetylcholine release and its binding to the receptor.

Summary

The control of motor skill consist of three systems: reflexive, autonomic and voluntary. Walking movement has an automatic motor function performed by three main control systems in brain stem.

Voluntary movement is caused by the pathway from the cerebral cortex (motor area) to the spinal cord (anterior gray horn) inputting the motor neuron cell body which is the final common path to the muscle. The motor area is governed by the cerebelum and basal ganglia. Its output is controlled by feed back systems from the sensory area.

Acetylcholine (neuro-muscular junction), Ca^{2+}(endoplasmic retriculum) and ATP (hydrolysis by ATPase) play an important role as chemical mediaters for muscle contraction. The dysfunction of each in any level of the motor control system causes motor disorder such as motor neuron disease, myasthenia gravis (acethylcholine receptor), progressive muscular dystrophy and Parkinson's disease (dopamine neuron in basal ganglia).

Table10 : Effect of a meal on glycogen concentration in the muscle

Meal	Glycogen concentration (g/100g muscle)	Time(min) to exhaustive fatigue(bicycle ergometer)
Combined	1.75	113.6
High fat, high protein	0.63	56.9
High carbohydrate	3.31	166.5

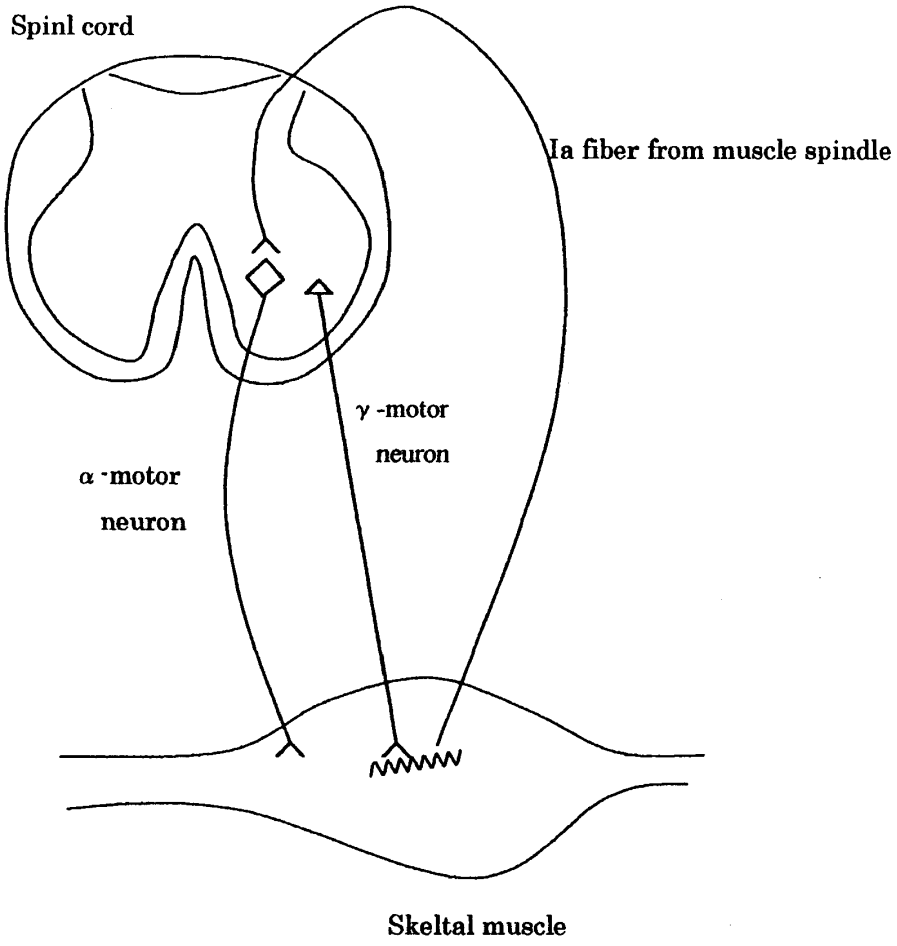

Figure93 : The pathway of the stretch reflex

Muscle stretch (tension) stimulates the muscle spindle, and the inpulse ascends onto the cell body of the motor neuron, stimulating action.

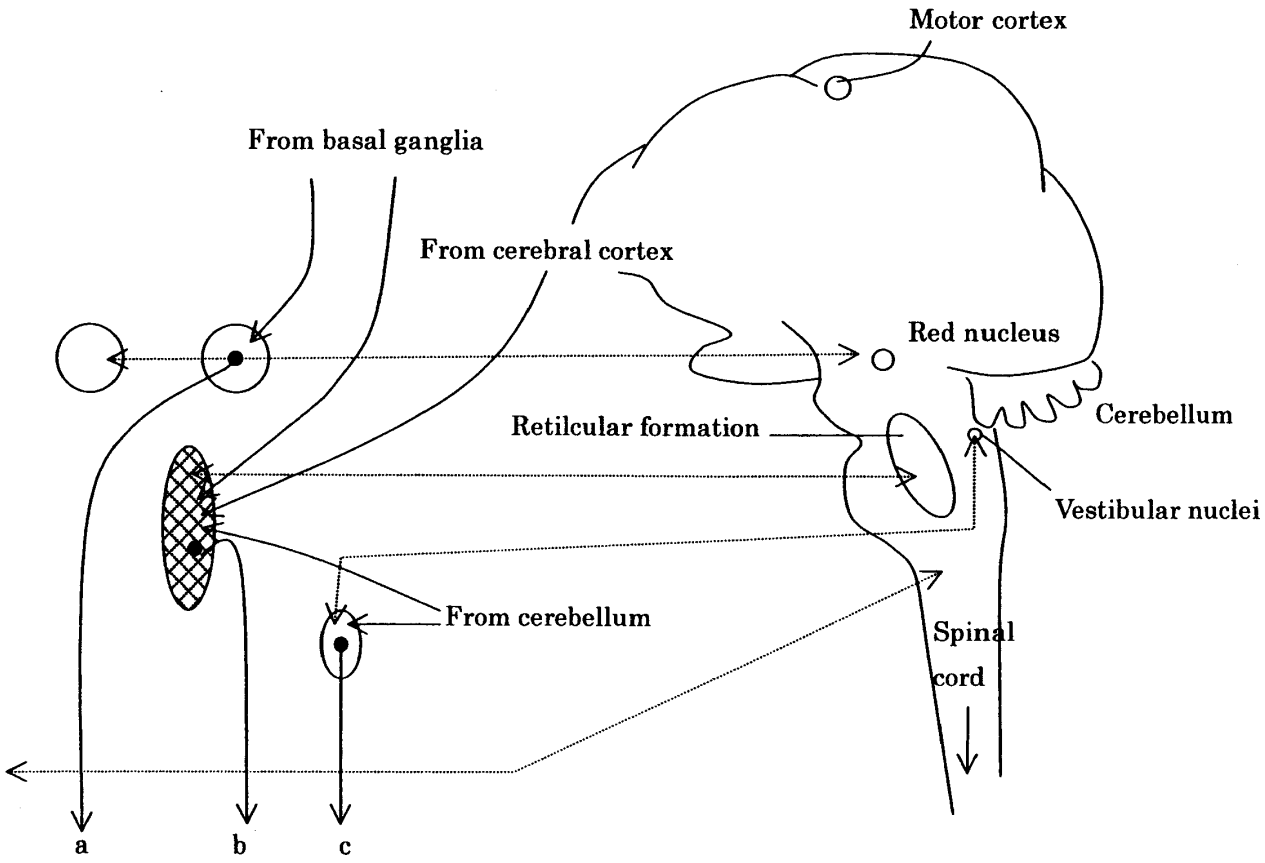

Figure94 : The extrapyramidal tract :

The brain regions which project movement down the spinal cord.

– related neural output

a : Rubrospinal Tract ; It relates to the regulation of muscle tonus for flexor(walking).

b : Reticulospinal Tracts ; It has the function of facilitation for flexor neuron and inhibition for extensor neuron(sitting behaviour).

c : Lateral Vestibulospinal Tract ; It has the function of monosynaptic facilitation for α -motor neuron which governs extensor of foot and knee(walking and standing).

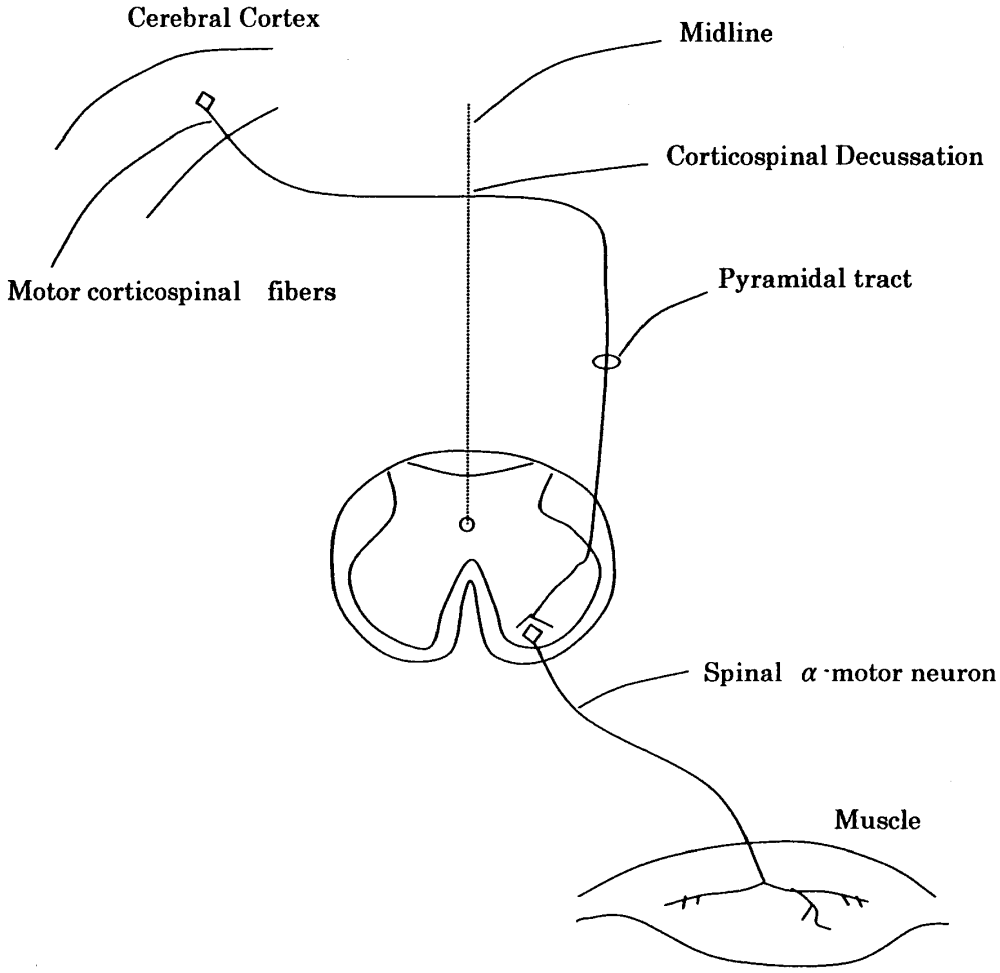

Figure95 : Corticospinal tract(pyramidal tract)

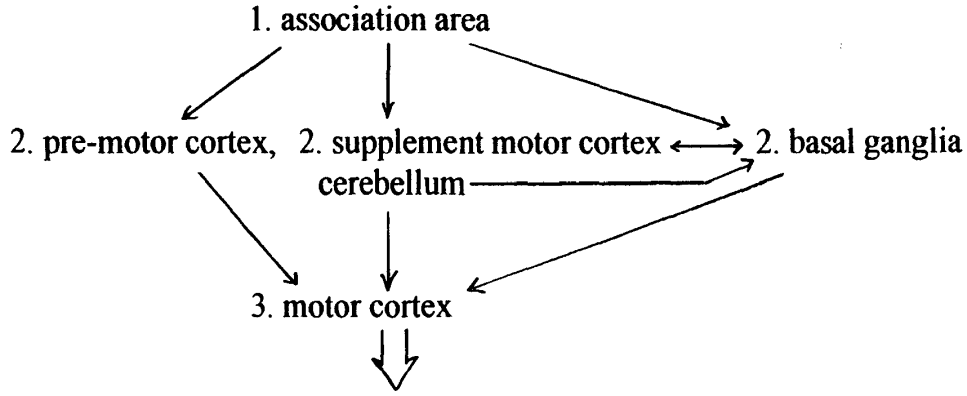

Figure 96. Organization of the motor system

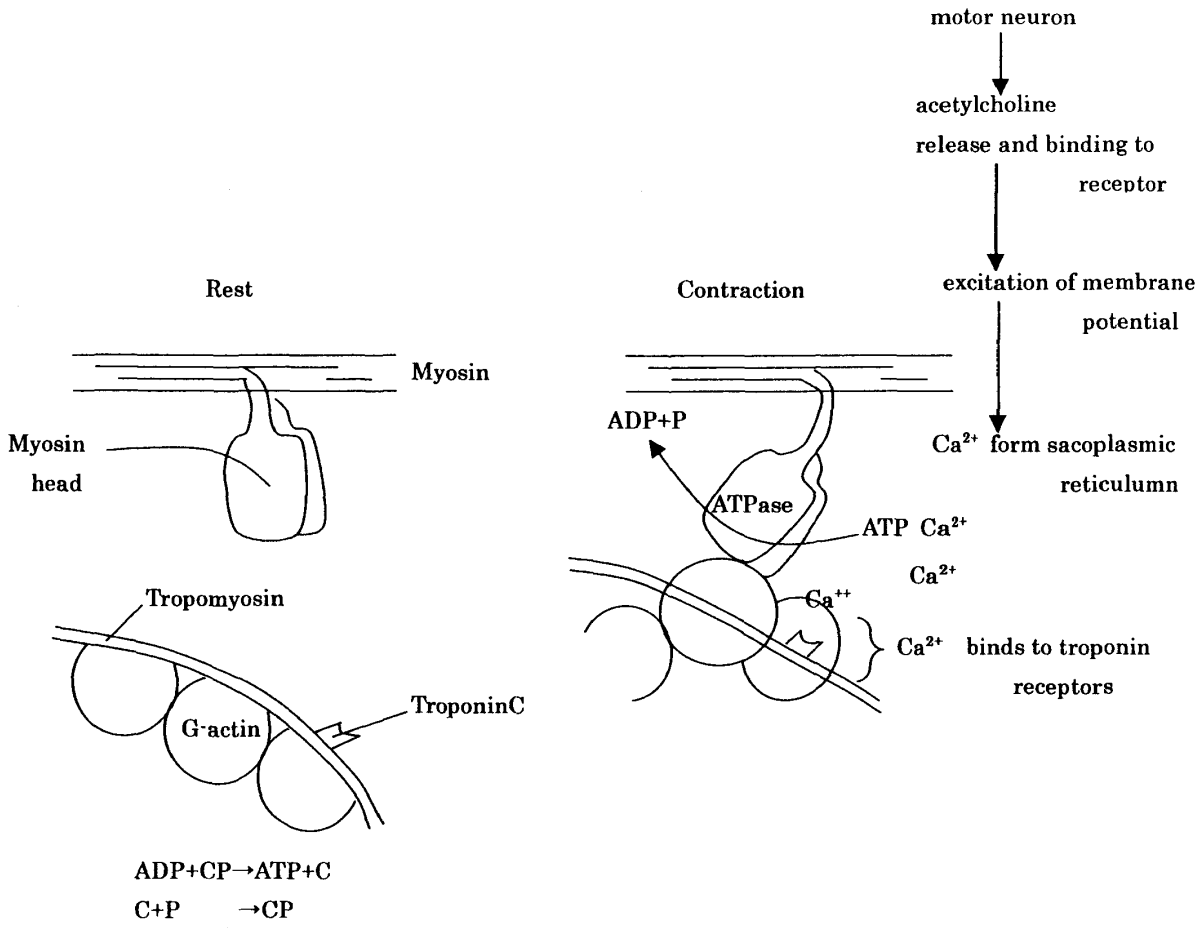

Figure97 : ATP hydrolysis by ATPase following skeltal muscle contraction

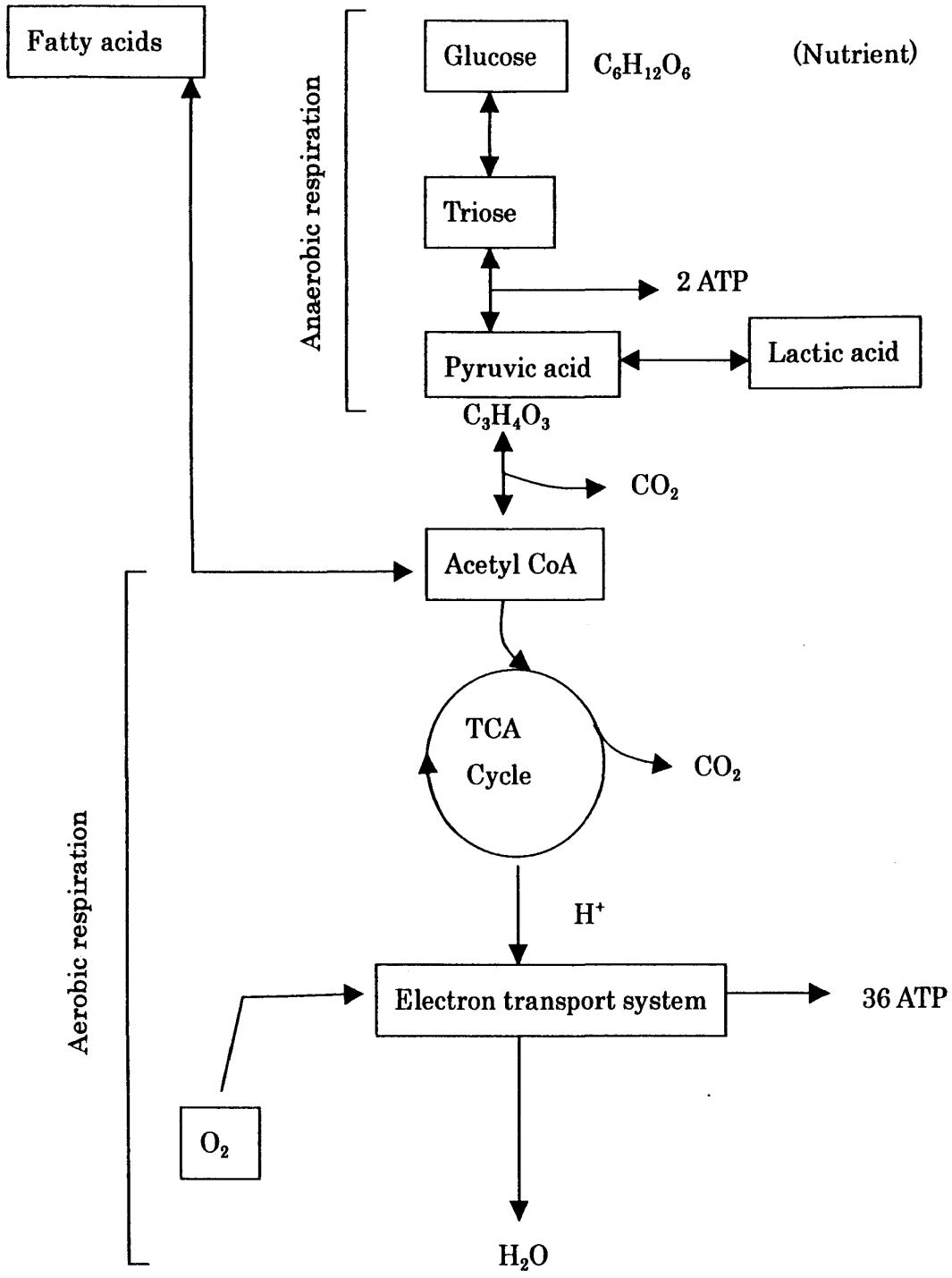

Figure98 : Production process of energy source(ATP) in muscle

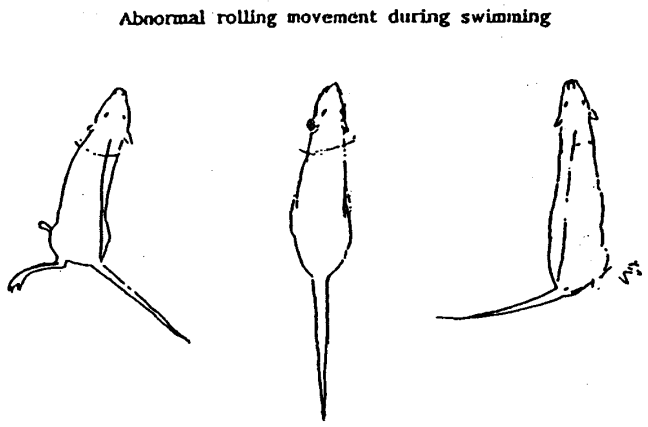

Figure99 : Rolling movement by 5,7-DHT treatment(10 μ g) into bilateral striatum in rats

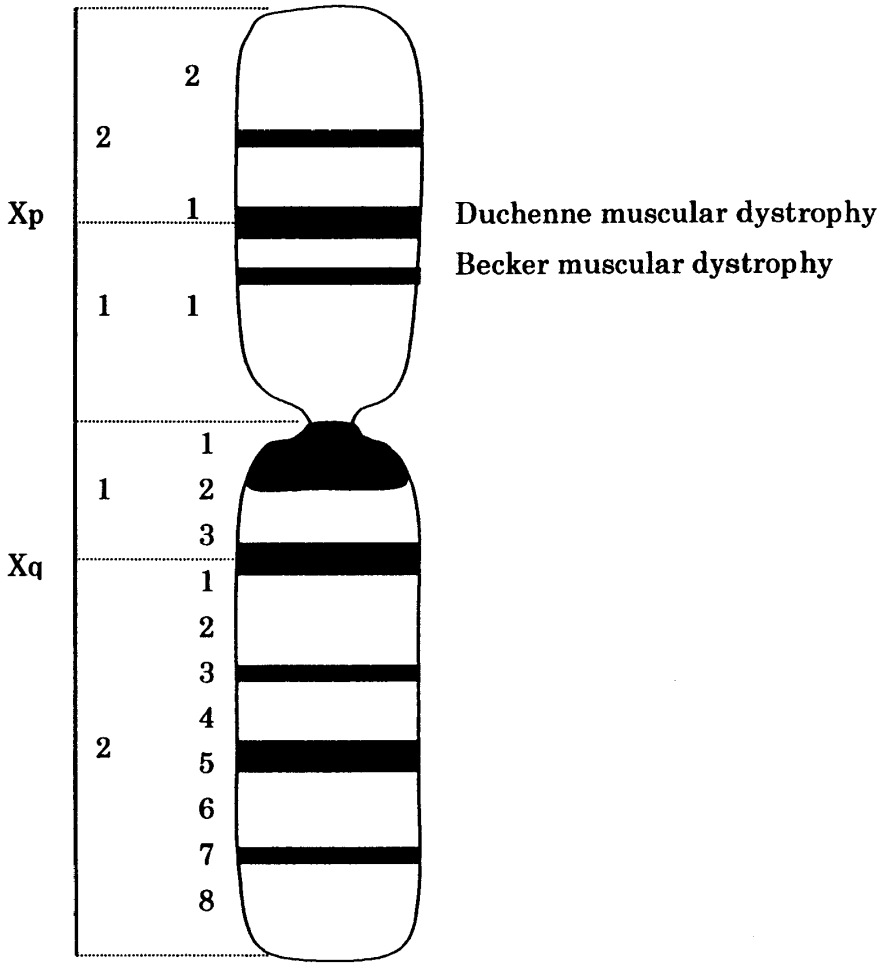

Figure100 : Gene map of the human X chromosome.

Muscular dystrophy gene code for huge dystrophin ; cytoskeletal protein.