Classificaton of nerve fibers

Table 11.2.1: Classification of Mammalian Nerve Fibres

Group						
Erlanger and Gasser	*Lloyd & Hunt**	*Myelination***	*Diameter (microns)*	*Conduction velocity (m/s)*	*Function*	*Agent to which conduction is most susceptible*
A alpha	I	M	13-20	70-120	Proprioception Motor supply to skeletal muscles	Pressure
A beta	II	M	4-13	25-70	Touch, kinesthetic sense, pressure	Pressure
A gamma	-	M	3-6	15-30	Motor supply to intrafusal muscle fibres	Pressure
A delta	III	M	1-5	5-30	Pain, temperature, pressure, touch	Pressure
B	-	M	1-3	3-14	Preganglionic autonomic fibres	Hypoxia
C	IV	UM	0.2-1.0	0.2-2	Pain, temperature, pressure Postganglionic autonomic fibres	Local anaesthetics

CLASSIFICATION DEPENDING ON DIAMETER AND CONDUCTION OF IMPULSES

- Three major types depending on the basis of diameter and rate of conduction:
- Type A nerve fibers
- Type B nerve fibers
- Type C nerve fibers

CLASSIFICATION OF NERVE FIBER BY SPEED OF CONDUCTION AND SIZE

Fiber type	Conduction velocity(m/s)	Fiber diameter(μm)	functions	myelin	Sen. to local anes.
A FIBERS					
alpha	70-120	12-20	Motor, skeletal muscle	Yes	least
beta	40-70	5-12	Sensory, touch pressure, vibration	Yes	
gamma	10-50	3-6	Muscle spindle	Yes	
delta	6-30	2-5	Pain(sharp, localized)temperature ,touch	Yes	
B FIBER	3-15	< 3	Preganglionic autonomic	Yes	
CFIBER	0.5-2.0	0.4-1.2	Pain (diffuse, deep), temperature, postganglionic autonomic	No	most

Responses to injury to nerve

Objectives

Should be able to describe,

I. Types of injuries

II. Responses of nerve injury in

CNS and PNS

End Organs (e.g. Muscles)

III. Factors that effect nerve regeneration

Nerve injury may occur due to :

- Obstruction of the blood flow
- Toxic substances
- Pressure over the fibre- crushing of the fibre
- Transection of the fibre

Classification of nerve injuries

(Seddon Classification)

1. Neuropraxia
2. Axonotmesis
3. Neurotmesis

Neuropraxia

- Injuries due to pressure
- Local conduction block only
- This is the mildest for of nerve injury. Mild, blunt blows, including some low-velocity missile injuries close to the nerve
- Recovery takes place without wallerian degeneration.

- Pressure over the fibre- short period - Obstruction of the blood flow, hypoxia
- The axon is not destroyed mild demyelination looses the function for a short period ,conduction block.returns within few hrs – few wks

Axonotmesis

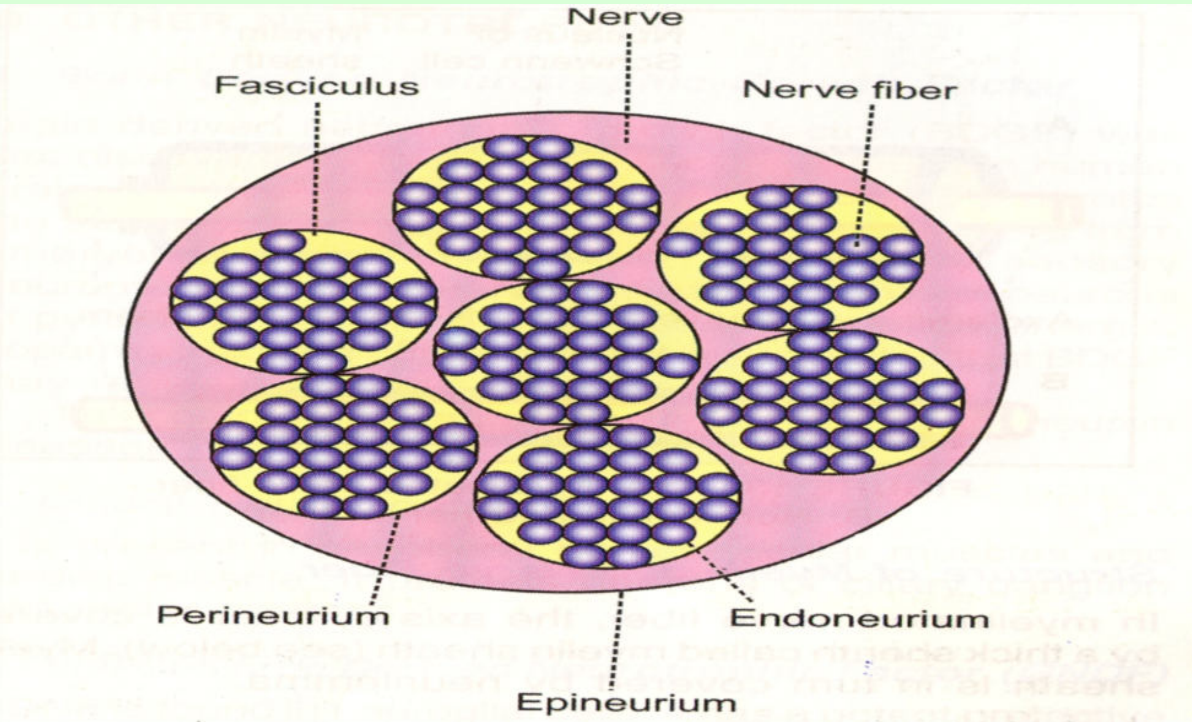
- Usually traction injury
- Endoneurial tubes are intact

Axonotmesis

- Severe pressure over the fibre- long period – endoneurium is intact
- Repair of function 18 months

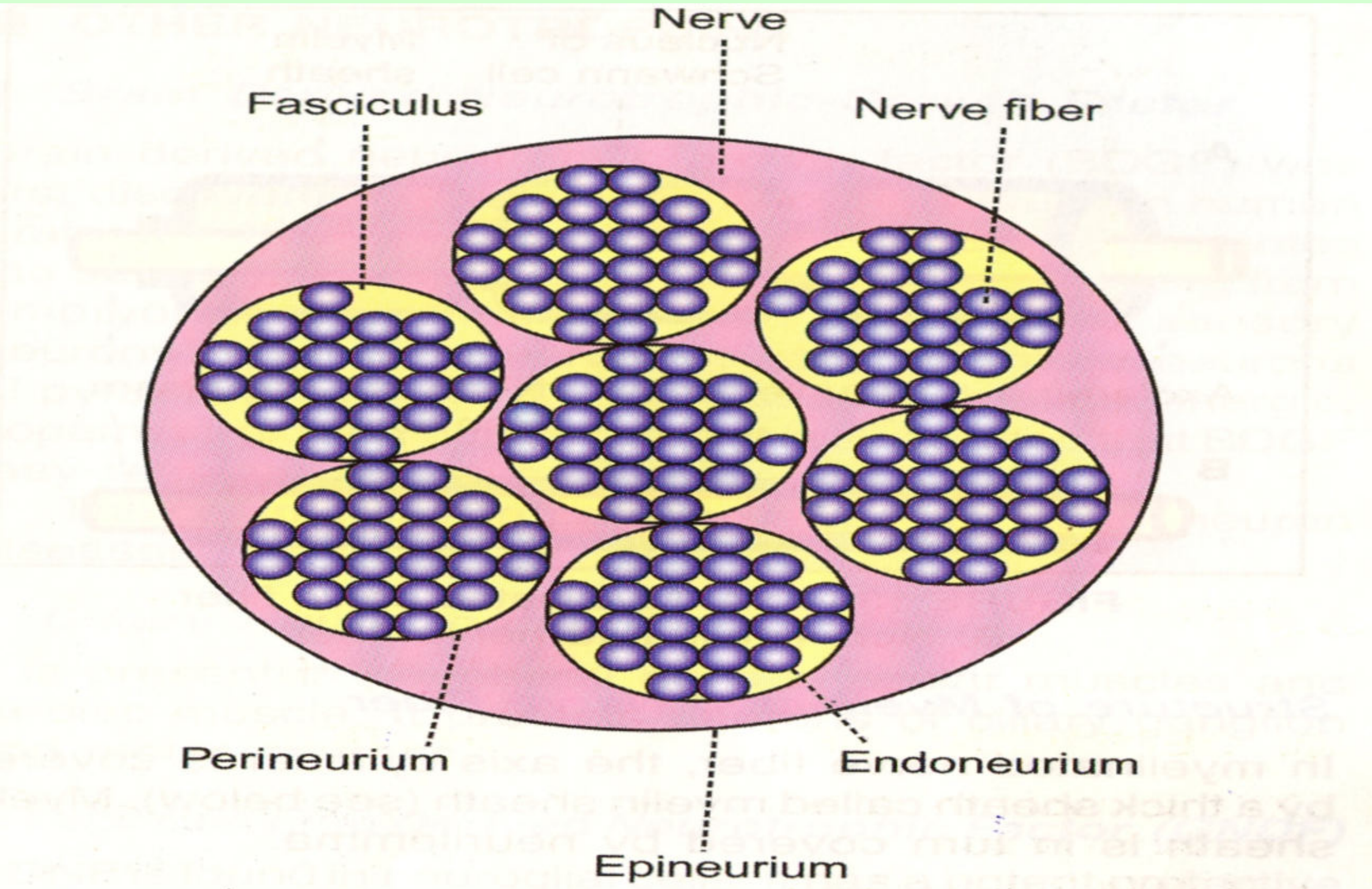
Endoneurium

- Each nerve fibre is covered by endoneurium

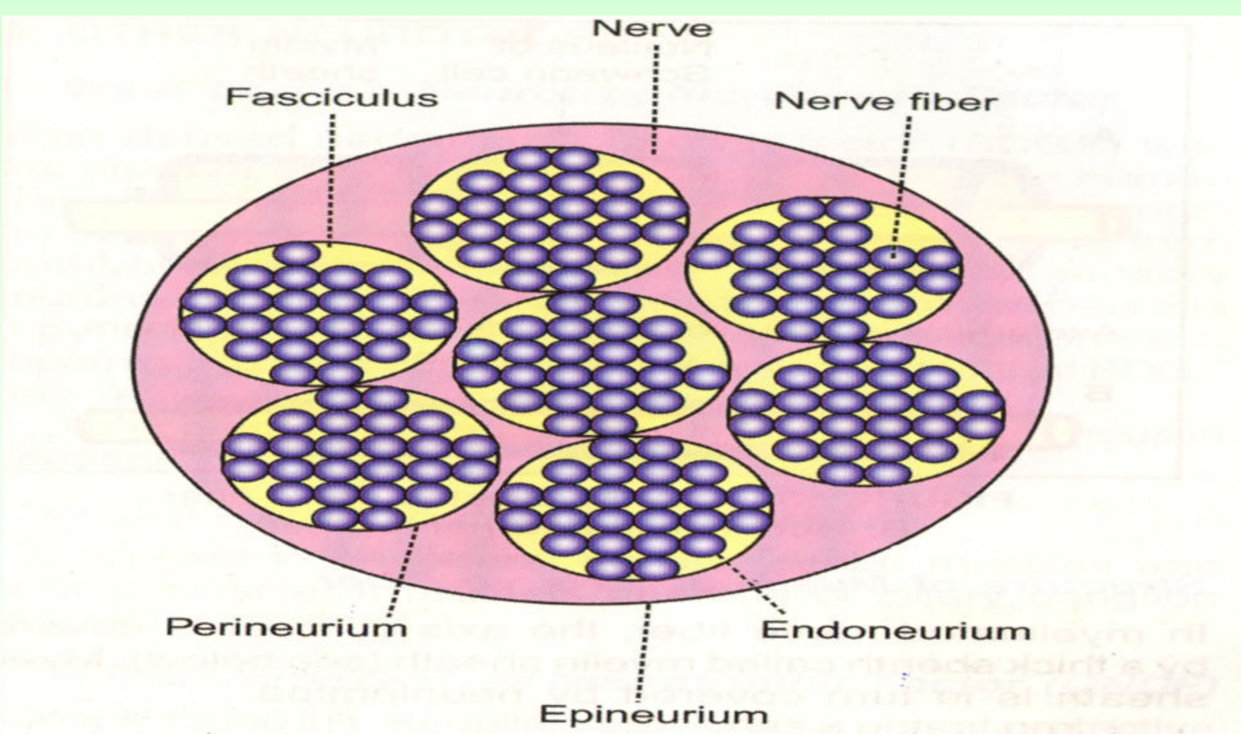


- Endoneurium is interrupted
- Epineurium and perineurium are intact
- Recovery is slow
- Neurotmesis

Each fasciculus is covered by Perineurium



Epineurium
Whole nerve is covered by a sheath



Degenerative changes
- axonotmesis- Walleriandegeneration

- *Changes in cell body*
- *Changes in axon*

Changes in cell body

1. Cellular edema
2. Chromatolysis starts near axon hillock. (Dispersion of Nissl fine granules -Cytoplasmic RNA)
3. Moving of nucleus to periphery

Changes in axon

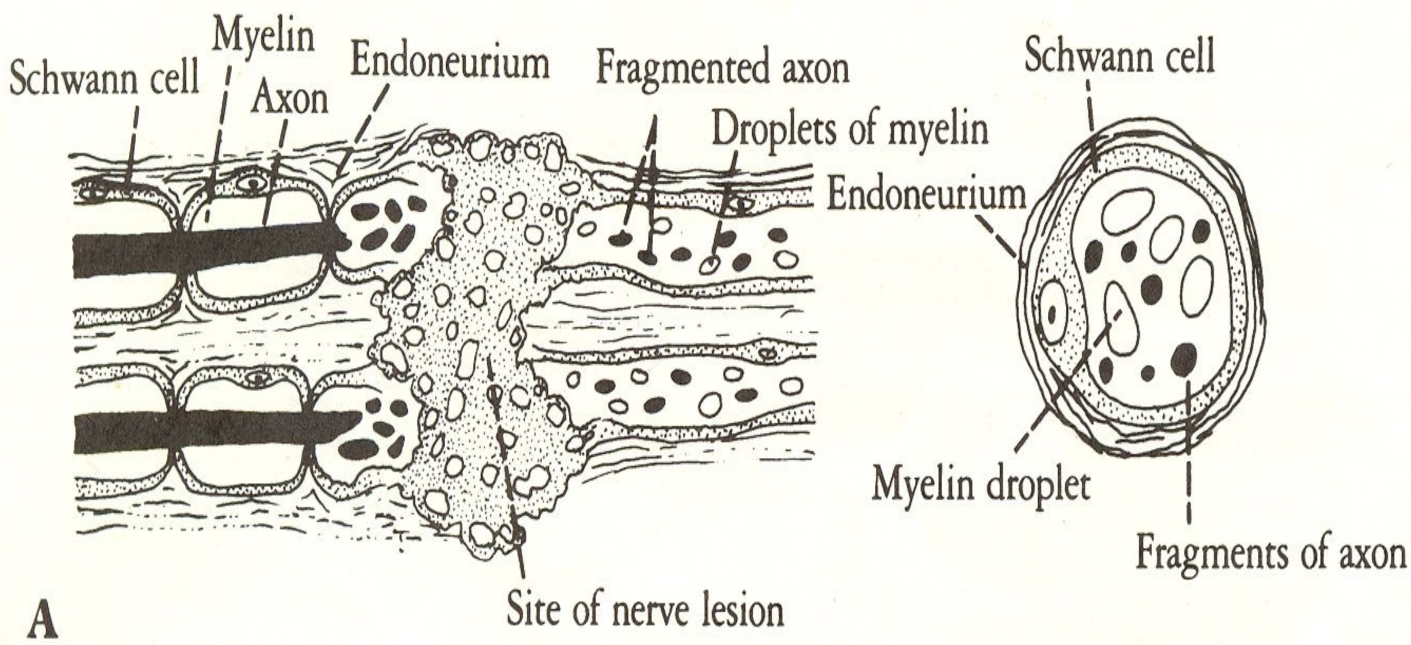
1. Degeneration process

a. Distal segment

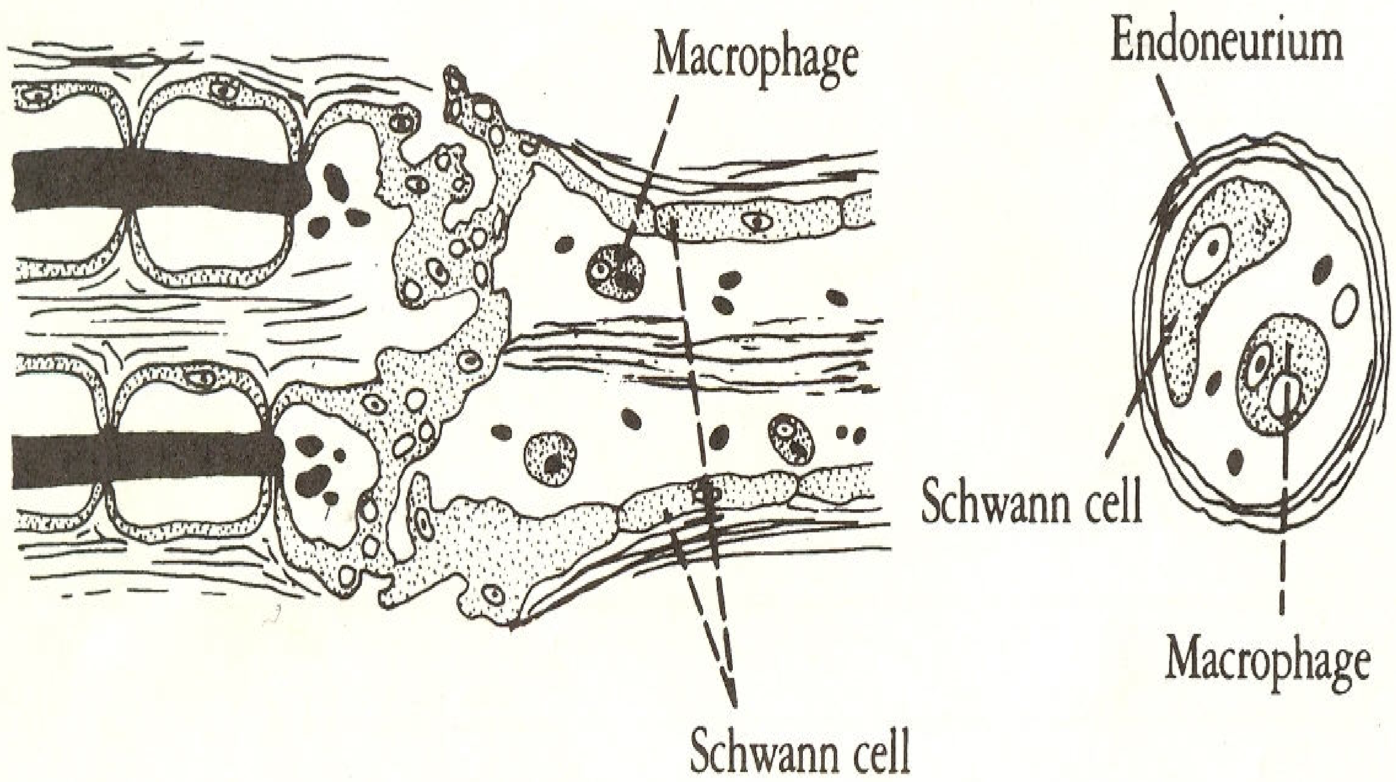
- **Swelling and fragmentation of axon & branches called *walleriandegeneration***
- **Debris digested by Schwann cells and tissue macrophages**

b. Proximal segment

- **Degenerate till first node of Ranvier**


Proximal end
Distal end
Myelin
Schwann cell
Axon
Endoneurium
Fragmented axon
Droplets of myelin
Endoneurium
Schwann cell
Myelin droplet
Fragments of axon
Site of nerve lesion

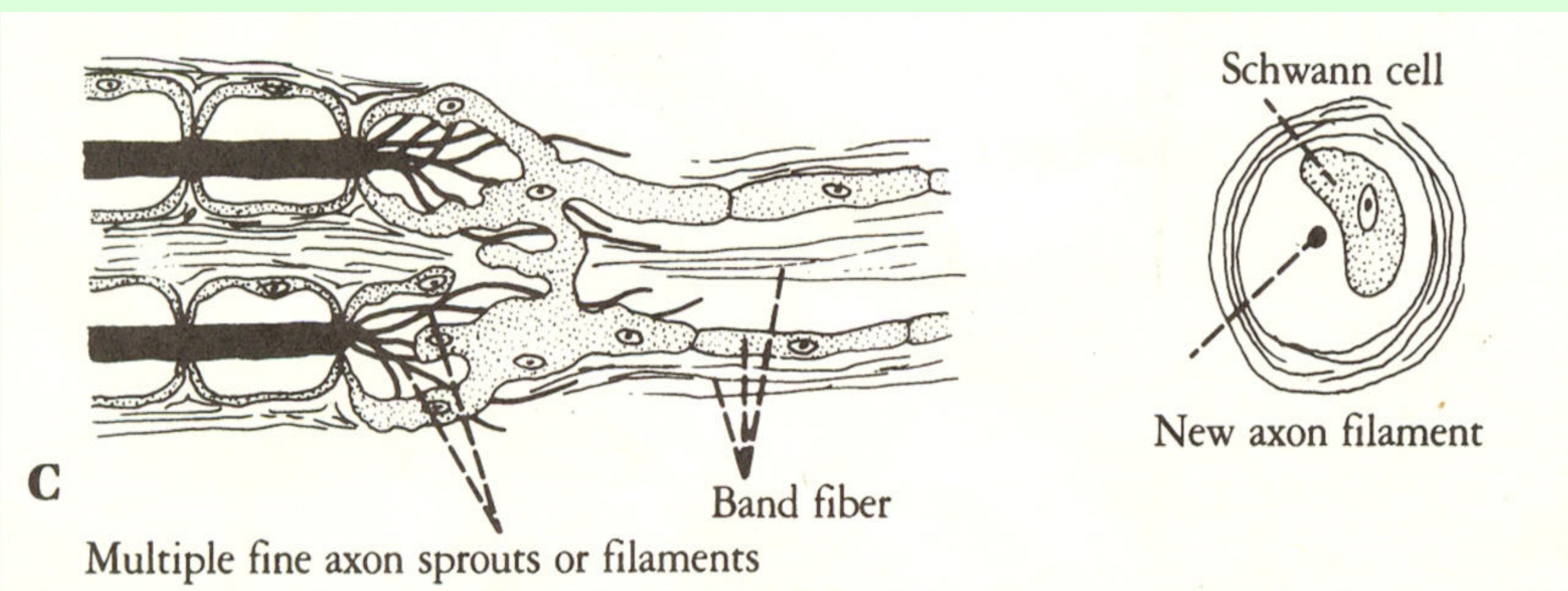
A


Macrophage
Endoneurium
Schwann cell
Macrophage
Schwann cell

B

2.Regeneration process

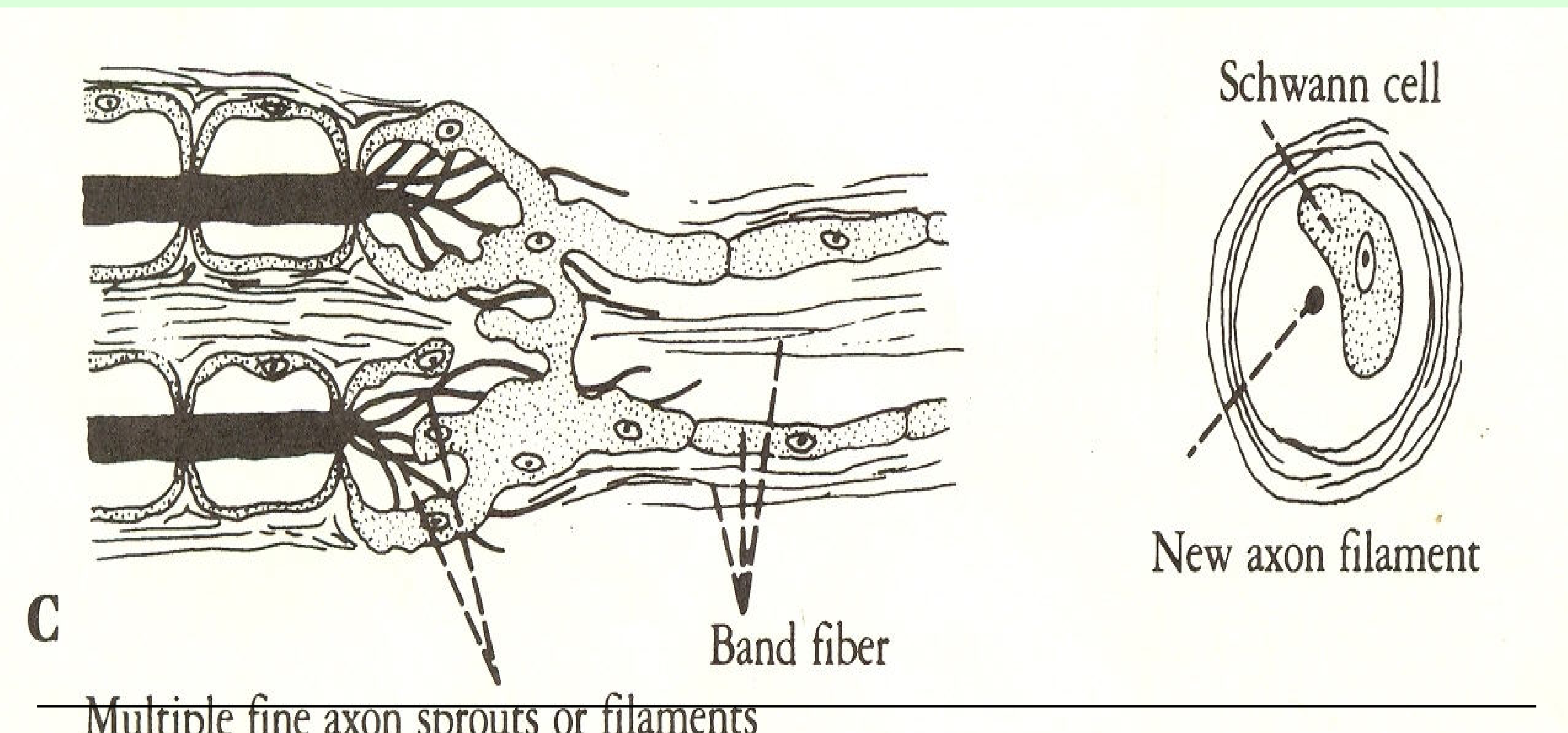
- Schwann cells rapidly proliferate and forms parallel cords within basement membrane
- Endoneurial sheath and contained cords of Schwann cells called *band fiber*
- The band fiber extends from first node of Ranvier in proximal segment up to end organ.



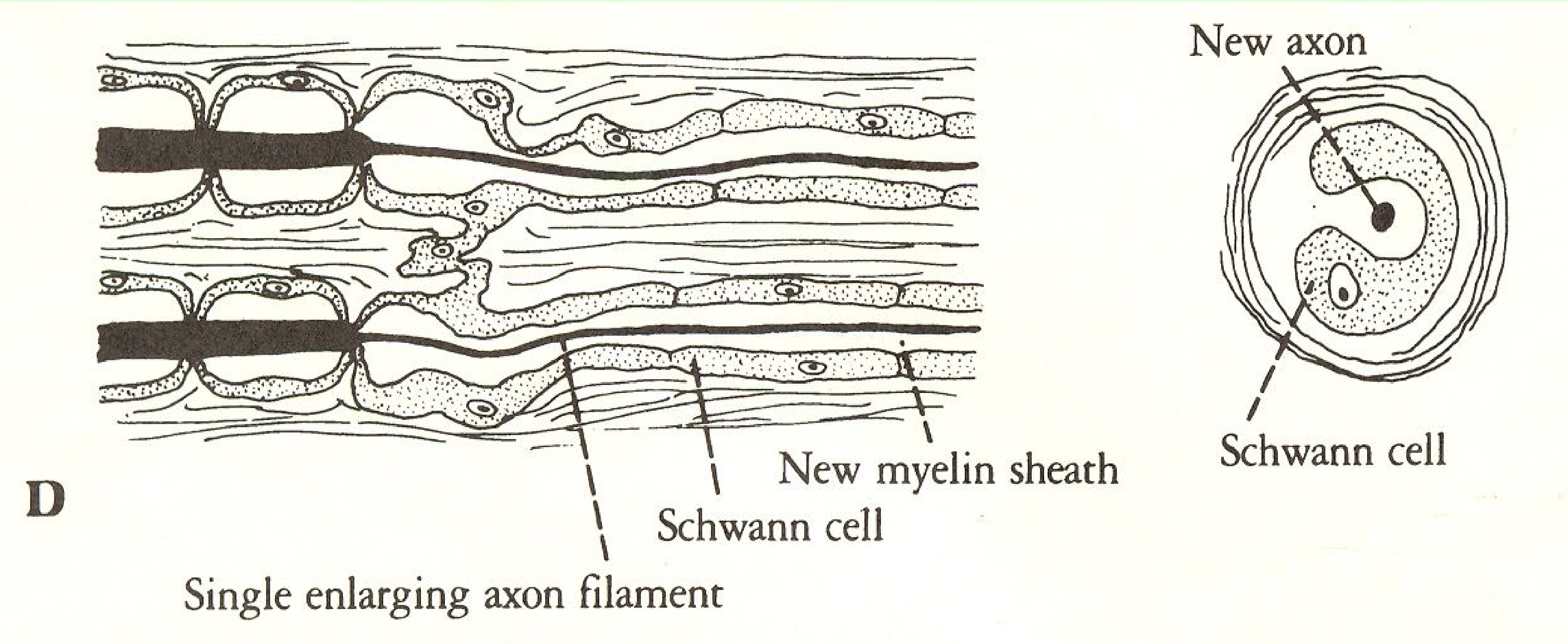
- When there is gap in the injury site Schwann cell will form the codes and bridge the gap if only endoneurial tube is intact.

(In CNS microglial cells phagocytosed the debris and astrocytes form a scar and no band fiber formation

- So regeneration of PNS depends on endoneurial tubes and Schwann cells
- Multiple sprouts arise from proximal axon and cross the gap through the codes of Schwann cell and enter in to distal segment.



But only one filament will persist and grows and reach the end organ.



- When axon reaches end organ Schwann cells begin to lay down the myelin sheath.
- It starts from injury site and spread distally.
- The time may be months to complete the process depending on the severity of injury

Recovery- reappearance of Nissl gran: due to Protein synthesis

Reduction of edema

Repositioning of nucleus

Recovered peripheral nerve may not be that efficient compare with the original nerve.

Why?

1. Reduced conduction velocity

   (Axon that reaches end organ will have 80% original diameter)

2. Muscle control will be less precise

   (Innervation of more muscle fibers)

No nerve regeneration in Central nervous system as in PNS.

Why?

1. Absence of endoneurial tubes
2. Failure of oligodendrocytes to serve as in the same manner as schwann cells in PNS
3. Laying down of scar tissue by active astrocytes cells
4. Absence of nerve growth factors, or
5. Production of nerve inhibitory factors in CNS

Changes of end organs supplies by the nerve

A. Loss of function
B. Denervation hypersensitivity (supersensitivity)

Loss of function

1. Skeletal muscles – atrophy
2. Sensory loss -cutaneous
3. Vasomotor-loss of sympathetic control impaired blood supply.
4. Sudomotor –loss of sweating & skin become dry

5. Trophic changes

Local tissue changes due to:

Nutrition/blood supply

Disuse & Loss of sensation

(E.g. in nail, bone)

Denervation hypersensitivity
(Supersensitivity)

Hypersensitivity of end organs supplied by denervated nerve due to increase response to neurotransmitter

Denervation hypersensitivity can occur in;

- Skeletal muscles

  Fine irregular contraction of individual fibers called *fibrillation*.
- smooth muscles
- Exocrine glands except sweat glands

This is due to :

1. Receptor up regulation in end Organs
2. Increase neurotransmitter levels at the site due to reduce uptake by the nerve

The factors that effect regeneration

- Type of injury
  - Gap between nerve ends
  - Distance to cell body
  - (Worse with proximal injuries)
  - Damage to adjacent tissue
  - Presence of foreign bodies
- Ischaemia &Infections
- Delay between injury and repair
- After care

Table 1. Adaptation of Seddon's Classification of Nerve Injury4

	Neuropraxia	Axonotmesis	Neurotmesis
Motor loss	Complete	Complete	Complete
Sensory loss	Partial sparing	Complete	Complete
Autonomic function	Spared	Absent	Absent
Nerve conduction distal to injury	Present	Absent	Absent
Fibrillation on EMG*	Absent	Present	Present
Recovery	Rapid, Complete	1mm per day, good	1mm per day, always incomplete

* electromyography