Division of Finance and Business Operations

Procurement & Strategic Sourcing
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September 4, 2015

Minutes of the Pre-bid Conference

RFP Duplication Services for UME Student Printing dated August 27, 2015

The pre-bid conference for the **Duplication Services for UME Student Printing** was held on **September 4, 2015 at 11:00 am Valerie Kreher** reviewed the administrative requirements of the pre-bid package, especially concerning details such as bid due dates and who vendors may contact during the live bid process. **Ron Spalding** of the **Conjoint Teaching Service/UME Departments**, discussed the expectations and scope of work.

The pre-bid conference attendees sign in sheet and meeting minutes are available for downloading from the University Purchasing Web Site at **http://www.forms.procurement.wayne.edu/Adv_bid/Adv_bid.html**.

Numerous simple questions and answers were addressed at the pre-bid meeting. Some of the issues were as follows:

1. The new Deadline for project related questions is **September 11, 2015, 12:00 noon**.
2. **The New Bid due date is due September 22, 2015 at 4:00 pm**, to be time date stamped in Procurement & Strategic Sourcing located in the Academic/ Administration Bldg., 5700 Cass Avenue, 4th Floor – Suite 4200, Detroit, MI 48202.
3. Please include six samples of a copy and paper with your bid
4. There is a new cost schedule to be posted with this bid. The quantity has been changed from 176,000,000 to 5,000,000 impressions. Most copies are double sided
5. Files are usually in PDF format, sometimes, there is a first generation hard copy as an original
6. There are very few color copies in this contract
7. Typical number of sets is around 300, possibly 350 sets of each job.
8. There are very few, or zero archiving jobs every year.
9. All jobs will require a proof.
10. There are no binding requirements, all jobs are 3 hole punched
11. Paper should be bright white paper and should be heavy enough not to see through
12. There will be several jobs for duplication almost every day of the contract. Pick up is generally once per day, however, there are occasions where they may be more than once per day.
13. Turnaround time is 24-48 hours, occasionally the turnaround will be requested in less time
14. We will require an original plus one copy **(2 total)** of your proposal. In addition, an electronic version is required, which should be submitted to our secure mailbox at **rfp@wayne.edu**
15. Any responses, materials, correspondence, or documents provided to the University are subject to the State of Michigan Freedom of Information Act and may be released to third parties in compliance with that Act, regardless of notations in the VENDOR's Proposal to the contrary.
16. Parking on WSU campus lots and structures are $7.00/access. Vendor must build parking into their lump sum bid. There is no parking allowed on the malls.

Questions since the Prebid:

Question:
1-sided copying 2000; Is all of this black ink or do you want a price on color as well?

Answer:
Please submit a price per page for color copies.

Question:
2-sided copying 5 Million, is all of this black ink or do you want a price on some color and if you want color ink on some, how many?

Answer:
Please submit a price per page for color copies.

Question:
When quoting collating will the file come in the correct order? 21742 sheets or 21742 jobs and how many sheets to a job?

Answer:
Yes. We are not sure what the second part of the question means. If it is not answered within the minutes or the questions, please resubmit the question.

Question:
What is the average number of sheets in a rubber band?

Answer:
Anything greater than 50 pages

Question:
What is the average number of sheets to be stapled?

Answer:
Anything less than or equal to 50 pages

Question:
Color paper 300, is that the standard colors or does it need to be the fluorescent colors?

Answer:
Fluorescent Colors

Question:
Can the slip sheets be 20# bond? Do they need to be 3 hole punched?

Answer:
yes and yes

Question:
NCR paper and printing, is it 8.5 x 11, or 8.5 x 5.5. What is the average quantity to be ordered at a time of each size?

Answer:
8.5x 11 – 350 copies. No 8.5 x 5.5.

Question:
2400 Card Stock, Do you want a price including the printing? Is 67# Vellum Bristol an acceptable choice?

Answer:
Just the cost of the card stock and please submit a sample of the card stock.

Question:
220 Pickup/delivery, are you saying you will have 110 deliveries and 110 pick -ups, so should I quote each separately?

Answer:
Quote the cost of delivery/pick up as a single expense per time. So the answer is yes.

Question:
Proof cost, if delivered as a PDF do we have to print a hard copy from your file as a proof? Does the file arrive as a word file and we have to change it into a PDF and if so will the fonts be imbedded or tell us which fonts you will be using to assure we have them? Post office charges .185 per piece for postage.

Answer:
The proof has to be a hard copy. We provide a pdf of the document. The original is emailed; the University is not asking vendors to mail anything.

All questions concerning this project must be emailed to: **Valerie Kreher**, Procurement & Strategic Sourcing at **313-577-3720** Email: **ab4889@wayne.edu** (copy to **Leiann Day**, Email: **leiann.day@wayne.edu@wayne.edu)** by 12:00 p.m., **September 11, 2015.**

Do not contact the Conjoint Teaching Service/UME Departments, or other University Units, directly as this may result in disqualification of your proposal.

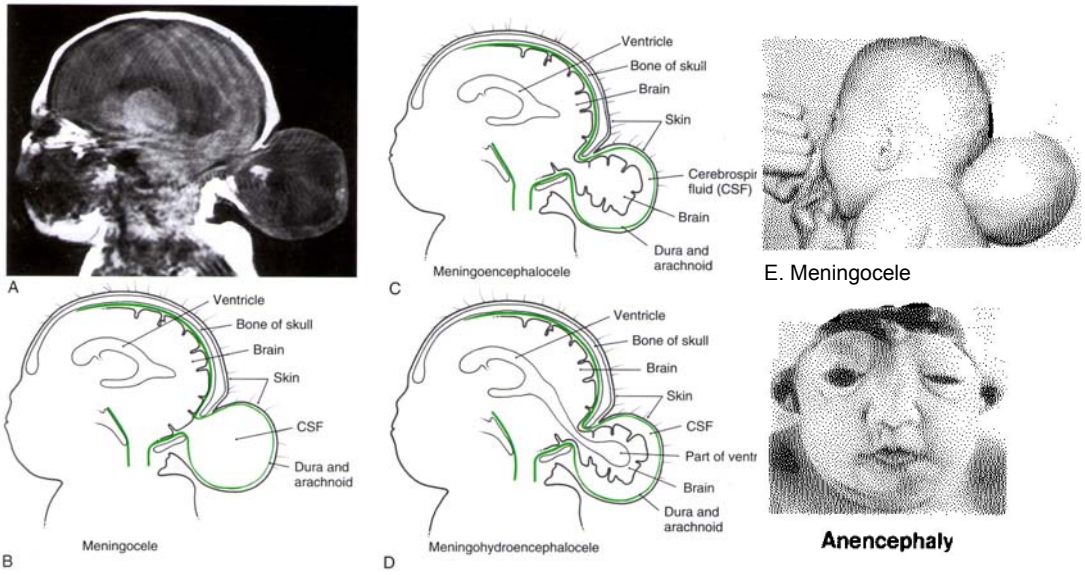
Thank you

Valerie Kreher,
Senior Buyer, Purchasing
313-577-3720

CC: **Ron Spalding and Tamara Taylor**, **Leiann Day**, Attendees list.

2. Cranial (Anterior) Neuropore Closure failure: defect commonly in the occipital region of the skull but may occur further rostrally (listed in order of ↑severity); associated with mental retardation and motor problems
   a. Meningocele: meninges herniated outside skull
   b. Meningoencephalocele: cyst, protrusion of meninges and brain tissue
   c. Meningohydroencephalocele: cyst, protrusion of meninges, brain tissue and part of ventricle
   d. Anencephaly: fatal, forebrain does not form

Fig. 4: Different types of closure failures of cranial neuropore (below). From Haines and also from Sadler



D. Early cell division and migration of cells – formation of ventricular, intermediate (mantle) and marginal zones, basic early plan in formation of brain and spinal cord, some areas such as cerebral cortex or cerebellum later have more complex development, for example additional layers form (Fig. 5)

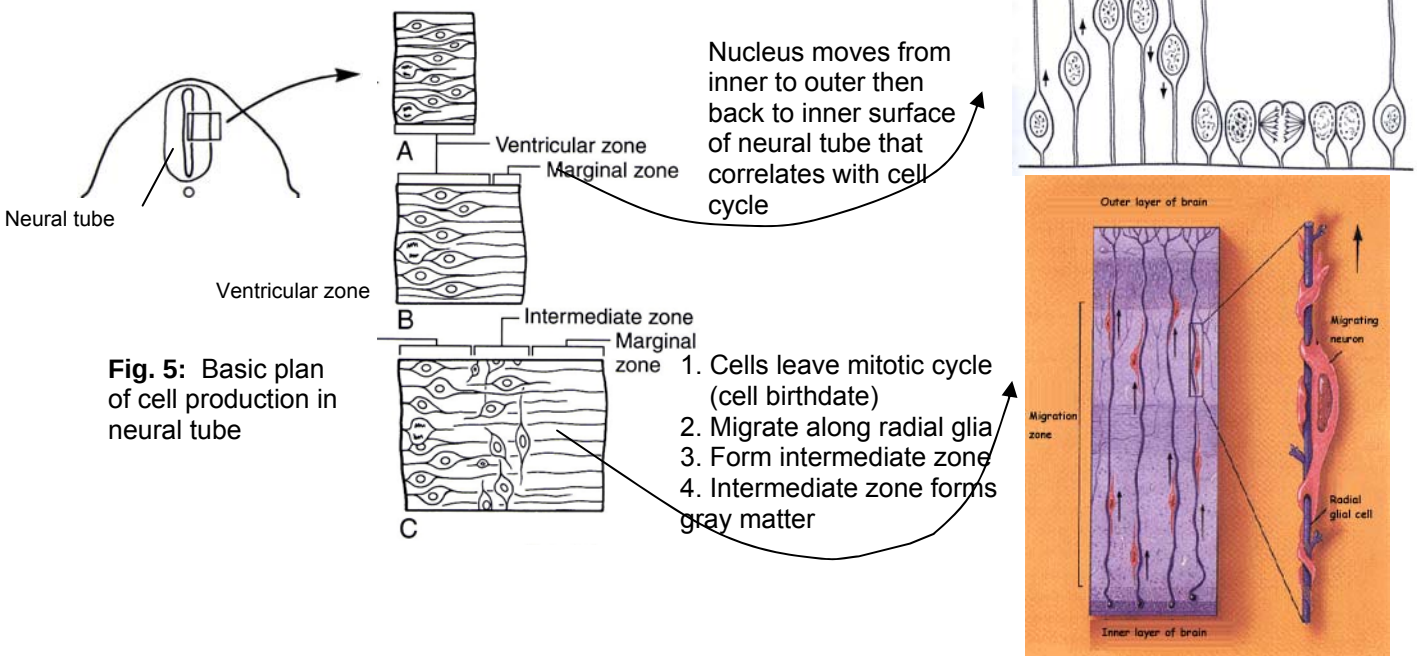


Fig. 5: Basic plan of cell production in neural tube

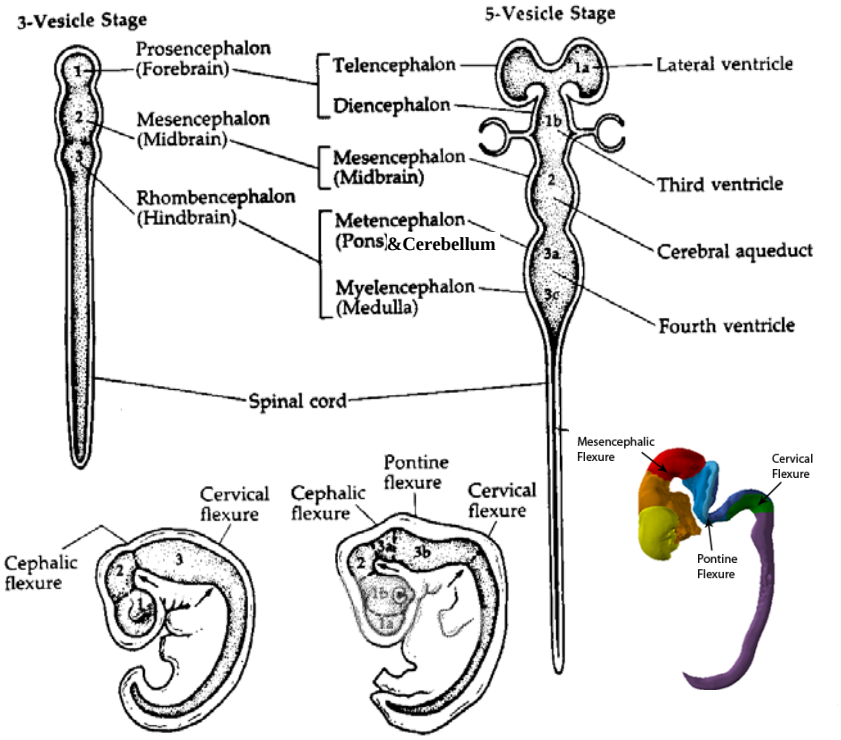


Fig. 7: Upper left:

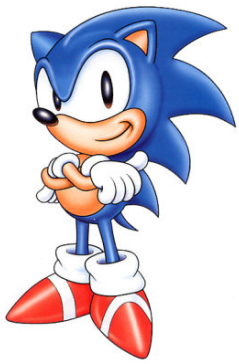
Longitudinal section thru embryo at 3-vesicle stage of brain development. To obtain such a section and also section in upper right, it would be necessary to stretch out the developing nervous system because a number of flexures are present at these stages.

Upper rt.:longitudinal section thru embryo at 5-vesicle stage. The forebrain & hindbrain are each divided into 2 regions.
Below: Lateral view of the embryos at 3-vesicle (left) and 5-vesicle stage (right).

II. Late Development of Nervous System (overlapping processes)

A. Molecular Regulation of Differentiation of Cells

1. Multipotent stem cells give rise to neurons, astrocytes and oligodendrocytes
2. Small number of multipotent stem cells in adult brain (eg.hippocampus)
3. Gradients of numerous molecules and receptors involved
   - Dorsal: BMP & other TGFß family members
   - Ventral: Sonic hedgehog
   - Rostral-caudal (spinal cord & hindbrain): HOX genes
   - Rostral-caudal (midbrain and forebrain): LIM1 and OTX2
4. Certain molecules important in forming patterns



B. Axonal path finding

1. Growth cones have multiple filopodia that sample environment
2. Direction determined by interaction with molecules in extracellular matrix or on surface of other cells (such as NOGO, NCAM)

C. Synapse formation, elimination & plasticity (Activity dependent)

1. Overproduction of synapses, many later lost
2. Compete for synaptic space
3. Synapses modified throughout life

D. Myelination (Activity dependent)

1. May also be dependent on size of axons
2. Motor & sensory tracts myelinate before association areas
3. Myelination continues thru 3rd decade of life, possibly longer

5. Basal plate (motor): anterior/ventral
   a. visceral motor (GVE)→smooth m., autonomics, cells closer to sulcus limitans
   b. somatic motor (GSE)→ skeletal m.
6. Arrangement of cell groups from ventral (anterior) to dorsal (posterior):
   GSE, GVE, GVA, GSA

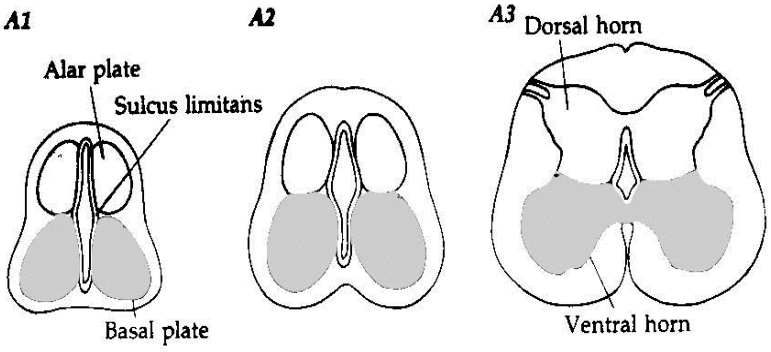


Fig. 8. Development of spinal cord showing alar & basal plates (from Martin)

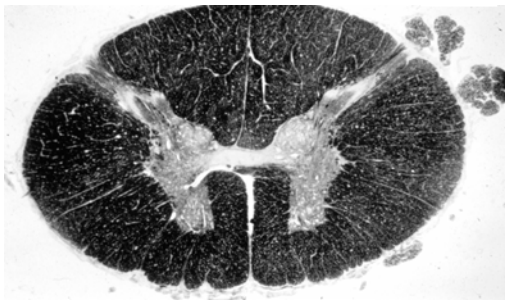


Fig. 9. Spinal cord from an adult, axons black (#5 from Rafols).

C. Axon outgrowth

1. Basal plate develops slightly earlier than alar plate, motor axons grow to myotubes (forms muscles).
2. Sensory axons (most from neural crest) follow motor axons. Note: The large peripheral branches are myelinated and have axonal morphology.

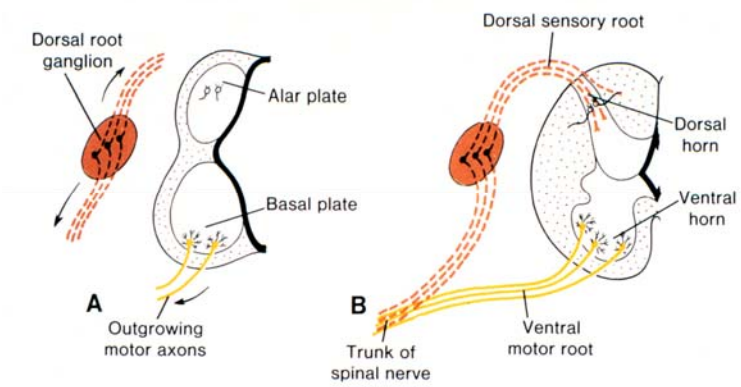
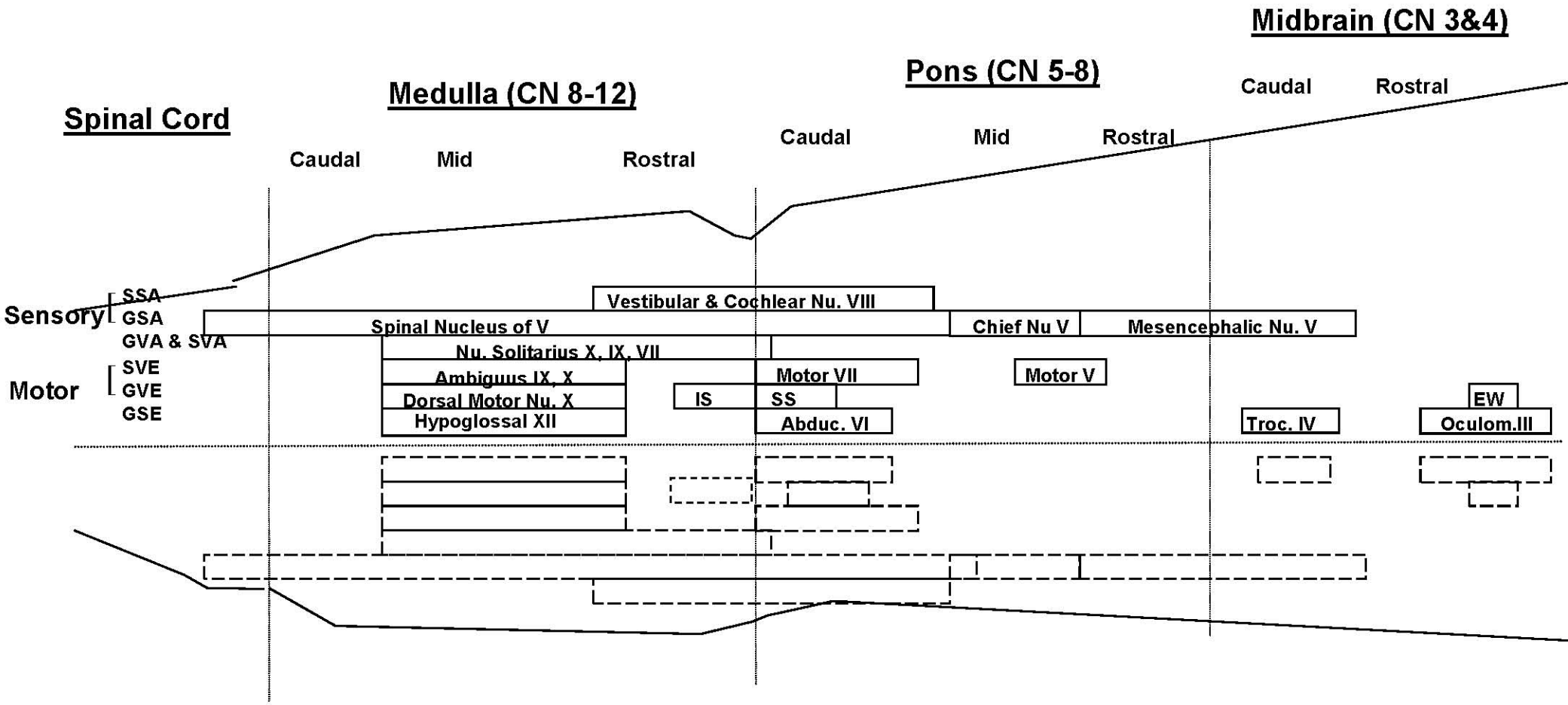
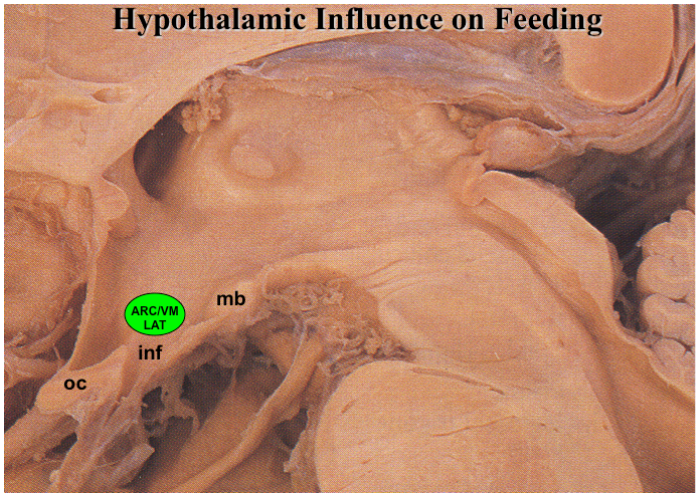


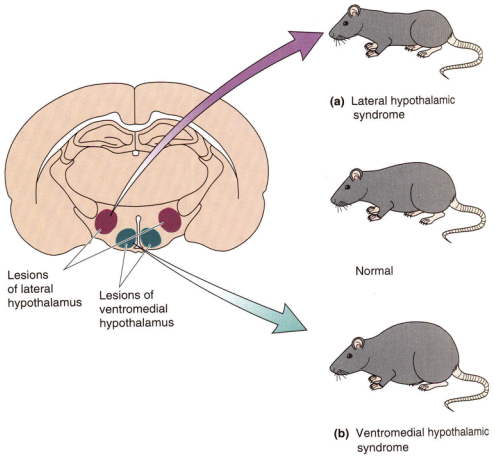
Fig. 10. Sensory axons follow path of motor axons.

Position of Cranial Nerve Nuclei in Brainstem



Control of Feeding



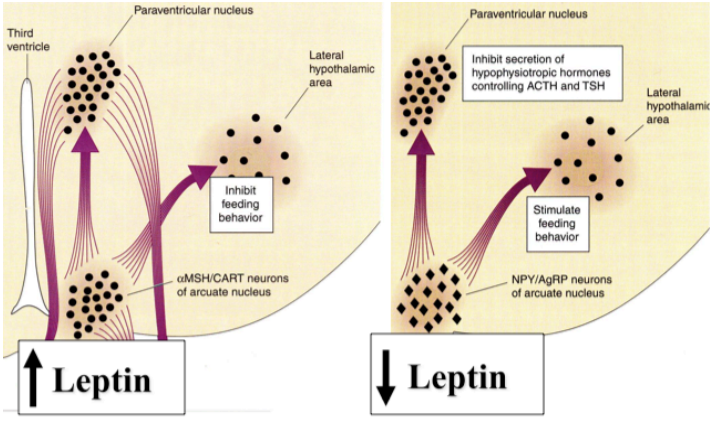


Feeding is controlled by nuclei at mid- hypothalamic levels.

Destruction of the ventromedial hypothalamic region causes hyperphagia (increased feeding). Therefore, this area of the hypothalamus is called the satiety center.

Destruction of the lateral hypothalamic area produces aphagia (decreased feeding to the point of malnutrition, dehydration and death). Therefore, this area of the hypothalamus is called the feeding center.

To Start Feeding: Cells of the arcuate nucleus possess leptin receptors that detect a drop in circulating leptin levels released from fat cells. The arcuate nucleus uses a set of neuropeptides to stimulate the lateral hypothalamus. This circuitry increases the desire to feed. The ventromedial hypothalamic nucleus is also thought to participate in this mechanism.



To Stop Feeding (Satiety): Gastrointestinal signals for gut distension and digestion of certain foods are transmitted by the vagus nerve to the solitary nucleus and then relayed to mid-hypothalamic levels. In addition, the arcuate nucleus senses a rise in blood leptin levels. As a result, the arcuate nucleus releases a different set of neuropeptides to inhibit the lateral hypothalamus to stop feeding.

Intake of Certain Food Types: Individual cell groups of the hypothalamus also regulate the intake of certain types of foods (fat, carbs, proteins). Certain neuropeptide transmitters used in the hypothalamus have been linked to cravings of certain food types.

II. Cognitive Declines in the Aging Population. *While some cognitive functions are severely affected with aging, other cognitive functions are age-stable and yet others improve with aging.*

- **Decreased with aging.**
- **A. Memory loss (all related to synapse loss in entorhinal cortex/hippocampal gyrus):**
- **B. Delayed short-term recall of verbal information**
- **C. Declines in speed of processing information and learning.**
- **D. Reduced spatial memory**
- **E. Altered executive function (related to cell loss in prefrontal cortex). Reduced and slow decision making.**

- **Age-stable cognitive functions include: long-term memory, attention span, vocabulary and verbal knowledge.**

- **Enhanced cognitive function with aging include: emotional components of memories and emotional stability.**

III. Structural Changes in Normal Brains (cont.). *Stereological methods of neuronal quantification and diffusion tensor imaging (assesses white matter) show very limited cell death in the normally (i.e., without neurological disease) aging brain . Nerve cell loss is very area-dependent and correlates with synapse loss and cognitive impairment.*

- **Brain Weight (av., approx. 1400 gms)**

  By age 80 there is 15% or less loss

  Most weight loss is due to cell loss.

- **Cell Loss**

  Prominent in prefrontal cortex (executive center of brain), medial temporal gyri (entorhinal cortex) and dentate gyrus of the hippocampus, last two centers processing working memory, short term recall and spatial memory.

  Brainstem catecholaminergic nerve cell loss in substantia nigra pars compacta and in locus ceruleus is also prominent.

  No nerve cell loss has been reported for nuclei in spinal cord, medulla, pons and hypothalamus.

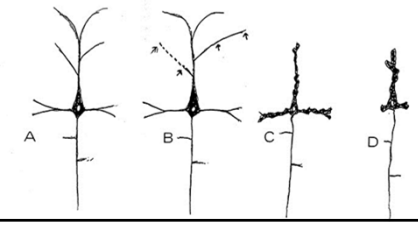
- **White Matter**

  Reduction in white matter density (diffusion tensor imaging) in prefrontal cortex and anterior corpus callosum. This reduction correlates with changes in executive function, short tem recall and processing speed

III. Structural Changes in Normal Brains (cont.). *There is growing experimental evidence of dendrite plasticity throughout the lifespan and into advance aging. This compensatory mechanism provides for stability of receptor surface to process cognitive and motoric information.*

Dendrites: Two populations of dendrites exist in the normally aging brain, a regressive and a growing population. Dendrite regression occurs due to synapse loss, whereas dendritic growth is a compensatory effort to maintain stability of receptor surfaces for "newly" sprouted synapses.

- Dendritic plasticity is retained into advanced aging (approx. up to 80 yrs of age).
- Beyond 80 there is net loses of dendritic surface accounting for cognitive loses.



In normal brains A and B occur up to 80 yrs of age. C and D occur after after 80 yrs.

III. Structural Changes (Cont). *Synaptic plasticity and its retention during the lifespan is the single, most important mechanism for maintaining critical brain function. Synaptic plasticity is defined as the capability of synapses to adapt or to be replaced in response to changes in their environment.*

D. Synapses

- As many as 27% of cortical synapses are lost. Synapse loss underlies most cognitive deficits experienced in aging.
- Synapses are eliminated during 'remodeling' of circuits. Synaptogenesis and synape loss are part of a synaptic "turnover" process which lasts for most of the life span.
- Astroglia and microglia play crucial roles in the removal of synapses, as well as guiding the newly formed synapses to reconnect with their postsynaptic targets.

A-D represent a single dendrite of a cortical pyramidal neuron. Clear synaptic terminal undergoes degeneration and is replaced by a newly sprouted terminal (dark). The degenerated terminal (dotted) is engulfed and stripped by an astroglia (arrow) process.