PLEASE PASS THE CAULIFLOWER: A RECIPE FOR INTRODUCING UNDERGRADUATE STUDENTS TO BRAIN STRUCTURE AND FUNCTION

Joan Masters and Margaret Christensen

*School of Nursing, Bouvé College of Health Sciences, Northeastern University, Boston, Massachusetts 02115*

Neurophysiology/pathophysiology content is a frequent source of anxiety for undergraduate students and their instructors. This learning module supplements traditional lecture and overhead presentations to offer a novel, nonthreatening, and entertaining introduction to neuropathology. The module is based on a ridiculous analogy between the human brain and the cauliflower. This module has been used with both underclassmen and more advanced health science undergraduate students and has produced enthusiastic student responses while deescalating both student and instructor anxiety.


Key words: neuroanatomy; neurophysiology; teaching neurophysiology

Human physiology and pathophysiology coursework is emphasized in diverse undergraduate programs. Students of clinical laboratory science and the allied health professions as well as physical education, pre-medicine, and physiology students enroll in undergraduate physiology classes in increasingly numbers every year (3). These students come with disparate levels of interest in, and preparation for, physiology coursework. One commonality among all undergraduate physiology/pathophysiology students is the level of anxiety evoked by the seemingly innocuous statement, "Next week, we'll start neuro." Knowledge acquired during states of anxiety is likely to be concrete, transient, and result in poor educational outcomes (4).

During the 1998–1999 academic year, we developed a nonthreatening learning module that has successfully deescalated the student anxiety historically triggered by the neurophysiology content of our undergraduate pathophysiology course. This module reviews neuroanatomy and physiology while simultaneously introducing pertinent neurophysiology. Thus novice students are motivated by an immediate understanding of the clinical relevance of the material rather than being bewildered by a series of seemingly disembodied strange-sounding names. This module has enhanced student learning and allowed us to expand the neuropathology content of our course. Most importantly, it has generated an unprecedented enthusiasm for neurophysiology and neuropathology among our students. The development of the module presented in the following sections of this paper was inspired by the observation of neurologist and neuroanatomist Stephen Goldberg (1) that, "the best memory techniques involve the use of ridiculous associations."

**MODULE OBJECTIVES**

1. To review the anatomy and physiology of the brain

2. To provide an introduction to common pathological alterations in brain physiology
3. To correlate brain pathophysiology and clinical presentation

MATERIALS

- One large well-proportioned cauliflower
- One large knife with a sharp point
- One large black plastic trash bag
- Handouts/overheads created with Microsoft Word

PREPARATION

With the use of a sharp knife, trim the green leaves from the cauliflower. Etch the sagittal sulcus, central sulcus (of Rolando) and lateral fissure (of Sylvius) on the surface of the cauliflower (see Fig. 1). Place the cauliflower, core-side down, in the trash bag. Before class, place the trash bag on the podium in a manner that reveals the general shape of its contents.

METHODS

Students are provided with printouts of all overheads that are presented during the lecture. We have found that these handouts free students from the distracting aspects of obsessive note taking. The handouts have ample white space to allow the students to jot down insights that develop during the presentation. The lecture begins with a traditional didactic format. An overhead of the gross anatomy of the brain is displayed, and the structures that comprise the central nervous system are identified. This slide is based on similar figures in classic physiology texts (2) but has historically produced a glazed expression in the eyes of many students. As the second or third student nods off, the instructor gestures toward the trash bag and speculates that use of an actual brain may clarify this discussion of brain pathophysiology.

The lecturer briefly explains that several years ago a very selfless patient, a victim of Parkinson’s disease, donated this brain for teaching purposes. This statement can be counted on to refocus student attention. The cauliflower is resurrected from the trash bag, and, despite the relieved laughter that it produces, the lecturer never concedes that this “brain” is, in fact, only a vegetable.

CORTICAL STRUCTURES

The lecturer orients the students to the anterior and posterior aspects of the brain and comments that the class is “obviously” viewing the lateral aspect of the left and dominant hemisphere of this brain. First, gyri (elevations), sulci (depressions), and fissures (smaller depressions) on the surface of the brain are noted. Next, the sagittal and central sulci and the lateral fissure are identified (see Fig. 2).

The relationship of these landmarks to the frontal, parietal, temporal, and occipital lobes is demonstrated. However, discussion of the pathophysiology in these areas is deferred. The motor cortex of the frontal lobe and the sensory cortex of the parietal
lobe (just anterior and posterior to the central sulcus) are identified. Unless an extremely well-proportioned cauliflower has been selected, the lecturer is reduced to searching out a mostly imaginary cerebellum.

The lecturer locates and, using the knife, resects “Broca’s area” (a small flowerette on the posterior aspect of the inferior temporal gyrus of the motor cortex of the frontal lobe). This introduces the common deficits in the production and/or comprehension of speech (aphasias) that result from specific cortical lesions. While exhibiting the resected Broca’s area to the class, the instructor explains that Broca’s aphasia is a motor aphasia that interferes with the production but not the comprehension of speech. Broca’s aphasia is characterized by halting telegraphic speech (e.g., “Student........came........class.”). Broca’s aphasia is sometimes termed nonfluent aphasia. Similarly, “Wernicke’s area” (a small flowerette on the posterior aspect of the superior temporal gyrus near the auditory area in the temporal lobe) is located and resected. Wernicke’s aphasia is characterized by an inability to comprehend language. Speech production is preserved (e.g., “The student trolley yes sir and baseball too!”) but meaning is absent. Wernicke’s aphasia is sometimes termed fluent aphasia. It is useful at this time to note the proximity of Broca’s area and Wernicke’s area on the cortex. Because both areas are perfused by the middle cerebral artery, isolated damage to only one area is rare. Most aphasia is global and has elements of impaired speech production as well as language comprehension.

Throughout the demonstration, the lecturer ostentatiously places all resected structures on the podium in clear view of the class. These structures will be used again later in the demonstration.

MIDLINE STRUCTURES

A complete midline transection is made along the anterior-posterior axis of the cauliflower. The lecturer demonstrates that this cut, along the sagittal plane, bisects the brain into a right and left hemisphere. Students can be oriented to the frontal and coronal planes at this time. The instructor sets the “right” hemisphere of the brain aside. An overhead illustrating a prototype neuronal pathway is displayed. Similar illustrations are found in all classic physiology texts (2). The instructor notes that the neuron, the basic functional of the brain, has evolved to transmit information and produce behavior. The components of a neuronal pathway are briefly reviewed (i.e., dendrites, cell body, axon, synaptic buttons, synapse, neurotransmitters, and receptors). The production of the neurolemma by oligodendroglia cells and the purpose of myelination and saltatory conduction are explained. A pathway is defined as a series of neurons. A tract is defined as a bundle of pathways. The student is reminded that a tract is the central nervous system equivalent of a nerve in the peripheral nervous system.

The organization of neuronal cell bodies in the cortex and the myelinated neuronal axons in the deeper white matter tract of the cerebrum is reviewed. The instructor exhibits the medial aspect of the “left” hemisphere to the class. Elaborating on the analogy between the brain and the cauliflower, the instructor draws the students’ attention to the clustering of neuronal cell bodies in the gray matter of the cortex (i.e., the small buds on the surface of the cauliflower.
flowerettes) and the convergence of the myelinated axon pathways to form deeper white matter tracts (the stalks of the flowerettes; see Fig. 3). The instructor comments that the white matter tracts from sensory and motor areas of the cortex come together as they pass through the internal capsulae of each hemisphere and proceed caudally to the brain stem and ultimately the spinal cord. The instructor notes that, because many tracts converge in the internal capsule, lesions of the internal capsule cause massive neurological deficits and have profound clinical consequences. At this point, the instructor also points out the corpus callosum and notes that it is the site of most interhemispheric information transfer.

The “basal ganglia” is resected next, and the lecturer allows the class to examine the structure. The instructor states that lesions of the basal ganglia result in involuntary and unexpected motor movements along with akinesia (decreased voluntary movement). The instructor reminds the class that this brain was donated by a victim of Parkinson’s disease. The class is instructed to pay special attention to the color of the substantia nigra, a nucleus of the basal ganglia. The substantia is normally dark due to the pigmentation of dopamine-producing cells. However, in Parkinson’s disease the dopaminergic cells are lost, and the substantia takes on the pale color that they observe. Loss of dopamine leaves Parkinson’s patients with a relative excess of acetylcholine. Parkinson’s disease is characterized by resting tremor, rigidity, and postural instability. It is useful at this point to contrast movement disorders associated with pathology of the basal ganglia with movement disorders associated with lesions of the cerebellum. Cerebellar lesions produce intentional tremors, difficulty coordinating fine motor movements, and gait ataxia (lack of coordination during voluntary movement).

Next, the ventricular system is introduced. Explaining that the brain is really a hollow tube with enlarged, cerebral spinal fluid (CSF)-filled ventricles, the instructor uses the knife to identify and carve out the left hemisphere’s lateral ventricle, the interventricular foramen (of Monro), the third ventricle, the cerebral aqueduct (of Sylvius), the fourth ventricle, the foramen of Luschka, and the foramen of Magendie. (see Fig. 3). The three meningeal membranes are identified: innermost the pia mater, the middle arachnoid membrane, and the outermost dura mater that clings to the periosteum of the skull. The instructor defines each area as it is carved out and explains that CSF circulates through the ventricular system to the subarachnoid space and is reabsorbed by the arachnoid villi in the superior sagittal sinus. The instructor explains that an obstruction anywhere in the system, an increase in the production of CSF, or a decrease in reabsorption of CSF will increase pressures in the ventricular system. The Monroe-Kellie hypothesis, the pathophysiology of increased intracranial pressure, and herniation syndromes can be introduced at this point if this content is consistent with the course objectives.

CEREBRAL CIRCULATION

At this point, the left hemisphere, mangled by dissection and resection, is set aside, and the instructor
displays the right hemisphere to the class. This hemisphere is used to describe cerebral circulation, the functional and behavioral capabilities of the cerebral lobes, and the clinical manifestations of specific cortical lesions. The expectation is that, at the completion of this section, students will be able to predict the pathophysiology associated with the occlusion of specific cerebral arteries.

The brain’s blood supply is provided by two arterial systems: the internal carotid system and the vertebral system. The instructor traces the paths of these two systems on the surfaces of the brain. The bifurcation of the internal carotid artery into the anterior and middle cerebral artery is illustrated. The anterior cerebral artery supplies blood to ~80% of the midline surface of the cerebrum. The middle cerebral artery supplies blood to ~80% of the lateral surface of the cerebrum. The vertebral system supplies blood to the cerebellum and the remaining 20% of the lateral and midline surfaces of the posterior cerebrum, including the occipital lobe (see Fig. 4).

**MOTOR SENSORY DEFICITS**

The instructor uses the right hemisphere to visually remind students that the motor and sensory cortices are located in the frontal and parietal lobe, respec-
tively, anterior and posterior to the central fissure and continue from the Sylvian fissure up the lateral aspect of the cortex to the sagittal sulcus and continue downward on the midline structures. The instructor maps the motor and sensory tracts on the surface of the right hemisphere and emphasizes that midline lesions of either the motor or sensory tract impair movement and sensation in the contralateral lower extremities (in this case the left leg), whereas lesions of the motor or sensory tract on the lateral aspect of either the cortex or sensory tract impair movement of the contralateral right upper extremities (in this case, the left arm). In contrast, cerebellar lesions affect movement on the side of the body ipsilateral to the lesion.

**BEHAVIORAL DEFICITS**

The previous discussion leaves students with the impression that discrete areas of the brain control specific functions. It is important to advise students that the brain, in fact, functions as an integrated whole. However, certain areas of the brain are more involved in some functions than others (see Fig. 5).

**The frontal lobe.** The instructor breaks off an area of the brain roughly corresponding to the right frontal lobe and explains that, in addition to motor function, the frontal lobe is involved in planning, problem solving, personality, inhibition of impulses, and the initiation of behavior. Thus a patient with a frontal lobe lesion may have problems dressing, reacting appropriately in social situations, and in starting or completing a task. The instructor can reinforce Broca's aphasia at this point by reminding the class that lesions of the right hemisphere are not associated with motor aphasia.

**The parietal lobe.** Next, the parietal lobe is broken off. In addition to perception of sensory input, the parietal lobe is involved in interpretation of visual information, right/left discrimination and the awareness of body parts, and the orientation of the body in space. Patients with parietal lobe lesions tend to ignore the side of the body contralateral to the lesion, have problems realizing the relationship of their contralateral side to potential hazards, and do not receive accurate sensory input from the side of the body contralateral to the lesion. These deficits put them at serious risk for injury.
The temporal lobe. The temporal lobe is broken off. The temporal lobe is involved in hearing, emotion, consolidation of memory, and some aspects of visual perception. Patients with temporal lobe lesions may have difficulty with face recognition (prosopagnosia) and memory consolidation and may experience unexpected mood swings as well as auditory and/or visual attention deficits.

The occipital lobe. The occipital lobe is removed. Occipital lobe lesions produce various visual defects ranging from cortical blindness (complete loss of vision without injury to the eye or optic nerve) to field cuts (visual loss in selected portions of the visual field).

The brain stem. After all of the lobes have been resected and carefully placed on the desk, the instructor is left holding the brain stem. The instructor characterizes the brain stem as the most primitive part of the brain controlling respiration, heart beat, alertness, and sense of balance. The vomiting center (the floor of the 4th ventricle) and the midbrain reticular activating system (consciousness) are also identified. Lesions of the brain stem are often associated with vertigo. Lesions of the brain stem threaten survival by interfering with breathing and heart beat.

WHAT TO DO WITH THE LEFTOVERS

The similarity between the mammalian brain and the cauliflower is a useful device for achieving the objectives of this module. Whereas the association is truly ridiculous, it facilitates review of the anatomy and physiology of the brain (objective 1) while simultaneously introducing students to common pathological alterations in brain physiology (objective 2) in a manner that eliminates anxiety as a barrier to learning. We have presented the module as a monologue. However, during the presentation, we take every opportunity to engage the student: questions are asked and elicited, clinical states are dramatized, and dialogue is encouraged. The final objective of the module is the correlation of brain pathophysiology with clinical presentation (objective 3). The ground work for this objective was laid during the presentation, and the objective is met by reviewing the presentation content. The instructor uses the resected areas of the brain, carefully and conspicuously arranged on the podium, to cue student responses to a series of questions. For example, students may be asked to:

Characterize deficits produced by pathophysiology of the internal capsule.

Students must correlate knowledge of neuronal organization, the physiology of the internal capsule, and the functional properties of the lobes. Hopefully, students will ask the instructor to specify the right or left internal capsule. A response that includes global motor and sensory deficits is acceptable. If necessary, the instructor will cue the students by retrieving the “internal capsule” from the leftovers on the podium and carefully replacing it in the remains of the left hemisphere.

Describe the deficits associated with occlusion of the left middle cerebral artery.

Students must correlate knowledge of the cerebral circulatory system and the functional properties of the cerebral lobes. A response that includes paralysis/paresis and sensory loss in the right upper extremity and aphasia is acceptable. Also the behavioral deficits associated with lesions of the frontal, parietal, and temporal should be included in the student responses. If necessary, the instructor will cue the students by retrieving Broca’s area and Wernicke’s area, asking students to replace them in the frontal and temporal lobes, and by reviewing anterior cerebral circulation. The appropriate lobes of the brain can be displayed to remind students of the behavioral deficits associated with occlusion of the left middle cerebral artery.

Describe the signs and symptoms of occlusion of the vertebral arterial system.

Students must correlate knowledge of the cerebral circulatory systems and the functional properties of the brain stem, cerebellum, and the occipital lobe. A response that includes ataxia, ipsilateral motor deficits, vertigo, visual-field deficits, nausea, change in level of consciousness, coma, or death due to cardiac or respiratory irregularities is acceptable. Usually, a conspicuous search for the “brain stem” and “cerebellum” will cue appropriate student responses.
Contrast movement disorders associated with pathophysiology of the basal ganglia and the cerebellum.

Students must specify differences in gait, posture, and fine motor movement (i.e., resting vs. intentional tremor) associated with basal ganglia and cerebellar lesions. The resected basal ganglia may be displayed to prompt recall of a movement disorder associated with the basal ganglia (i.e., Parkinson’s disease).

DISCUSSION

The learning module presented in this paper represents a typical presentation for our undergraduate pathophysiology course and amply fills our 65-min class period. However, the content of the module can be modified to meet the needs of specific groups of students and the time available. We have tailored this presentation to meet the requirements of more advanced undergraduate clinical courses and graduate pathophysiology courses. It is our observation that the ridiculous nature of the analogy between the brain and the cauliflower effectively engages students and demystifies neuroanatomy and pathophysiology content. Over the past year, student evaluations of this module have confirmed this observation. The following three responses typify student comments:

“I really enjoyed your (neuropathology) lecture. Great technique with the head of cauliflower. It helped in making your points clear, and it helped me visualize the areas you were discussing. You definitely held my attention throughout the class.”

“Your lecture was wonderful. I found it extremely interesting... the brain exhibit was great. Your lecture burned the topic into my brain!! Excellent learning tool.”

“The cauliflower brain provided a great visual aid in understanding the brain.”

Several themes have characterized the written comments. Students value the novel approach and enthusiasm demonstrated during the lecture, the interest added by the use of the cauliflower, and the humor inherent in the analogy. Thus, from the student point of view, the juxtaposition of the technical jargon of neuroanatomy (which we admittedly exaggerate for effect) and the anatomy of a large white protuberance of the mustard family is an effective device for introducing undergraduate health-care students to neuro-pathology. From the instructor’s point of view, this lecture is simply fun to do. It is hard to tell whether we or the students have been more astonished and delighted by their reactions to this learning module. However, we would like to add a word of caution to others preparing this recipe—a little ham greatly enhances the flavor.

Address for reprint requests and other correspondence: J. Masters, 408 Robinson Hall, College of Nursing, Northeastern Univ., Boston, MA 02115.

Received 8 February 2000; accepted in final form 8 August 2000

REFERENCES