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CLINICAL NEUROSCIENCES COURSE

SAINT LOUIS UNIVERSITY

INTRODUCTION

The nervous system is a fascinating and complex organ that consumes an abundance of energy in carrying out a variety of functions. Electrical impulses are generated to sense the environment, control movement, modulate emotive tone and generate cognitive activities. This unique organ system is not only immunologically privileged, but also secretes powerful neuropeptides. A comprehensive grasp of these activities and their mechanisms are beginning to unfold and, like man’s understanding of the circulation, will constitute one of the great milestones in medicine. Given the increasing pace of advances in neuroanatomy, neurochemistry, neuropharmacology and neurophysiology, the unraveling of the nervous system’s mysteries may well be reached in the next half century, the natural span of one’s professional life. This process should bring the knowledge that is needed for improved management and eradication of many neurological disorders. Therefore, the overall goal of this course is to provide a useful and stimulating introduction into the clinical neurosciences.

Up to 10% of patients seen by family practitioners present with neurologic symptoms and pose neurologic questions to their physicians. Only 16% of the 45 million Americans who visit a physician for a chief complaint referable to the nervous system are ever evaluated by neurologists. Clearly, primary care physicians are routinely called upon to evaluate and manage patients with neurologic disease. Practicing physicians require a firm understanding of the general principles of clinical neurology. The most suitable setting in which to lay the foundation for that understanding is in a neurology clerkship in the clinical phase of medical school.
Goals and Objectives

Phase 3 Objectives

After reading, independent study and participating in the Phase 3 clerkships and electives, the student should be able to:

1. Given a real or simulated patient, obtain a comprehensive or problem focused history, as warranted by the clinical setting.

2. Given a real or simulated patient, perform an accurate, complete and thorough physical examination or problem-focused exam as warranted by the clinical setting.

3. Deliver a clear, organized, concise oral presentation of a patient’s history and physical.


5. Given a real or simulated patient, develop a complete, prioritized differential diagnosis for the presenting complaint.

6. Recommend those diagnostic studies that best assist in determining a patient’s diagnosis and treatment plan.

7. Determine a final diagnosis by using a patient’s history and physical, and results from diagnostic studies.

8. Develop a management and treatment plan that involves the patient (and the patient’s family, if appropriate) and includes patient education, health maintenance, and disease prevention issues.

9. Search and interpret the medical literature in order to maintain a critical, current, and operational knowledge of new medical findings and its application to patient care and clinical decision making.

10. Demonstrate effective communication skills in dealing with patients, their families, and all others who participate in providing patient care.

11. Demonstrate effective interpersonal skills in dealing with colleagues, staff, and all others who participate in the educational process.
12. Maintain a professional attitude and behaviors as described in the SOM’s Code of Professional Conduct.

13. Demonstrate a willingness to accept professional limitations in one’s self and in other healthcare professionals.

14. Demonstrate an appreciation of how one’s culture and values impact on one’s perceptions and reactions to patients and their families.

15. Demonstrate a minimum knowledge base and skill level by having met the defined requirements for each clinical clerkship and elective.
Neurology clerkship-specific goal and objectives

During the neurology clerkship, the student’s **goal** is to acquire and/or reinforce the principles and skills underlying the recognition and management of the neurologic diseases a general medical practitioner is most likely to encounter in practice. This goal is achieved by attaining the following **objectives**, which are divided into **procedural skills** and **analytical skills**.

<table>
<thead>
<tr>
<th>Objectives: Procedural skills</th>
<th>Type of performance</th>
<th>Level of mastery</th>
<th>Means of assessment</th>
<th>MD degree program objective</th>
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<tbody>
<tr>
<td>Obtain a complete and reliable history</td>
<td>Can do</td>
<td>Student mastery</td>
<td>Performance assessments, small group discussions</td>
<td>Skills</td>
</tr>
<tr>
<td>Perform a focused and reliable neurologic exam, including on patients with altered mental status</td>
<td>Can do</td>
<td>Student mastery</td>
<td>Performance assessments</td>
<td>Skills</td>
</tr>
<tr>
<td>Deliver a clear, concise, and thorough oral presentation of a patient’s H &amp; P</td>
<td>Can do</td>
<td>Student mastery</td>
<td>Performance assessments</td>
<td>Skills</td>
</tr>
<tr>
<td>Prepare a clear, concise, and thorough written presentation of a patient’s H &amp; P</td>
<td>Can do</td>
<td>Student mastery</td>
<td>Performance assessments</td>
<td>Skills</td>
</tr>
<tr>
<td>Objectives</td>
<td>Type of performance</td>
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<tr>
<td>Analytical skills</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Recognize symptoms which may signify neurologic disease</td>
<td>Know</td>
<td>Student mastery</td>
<td>Performance assessments, small group discussions, multiple choice exams</td>
<td>Knowledge</td>
</tr>
<tr>
<td>Distinguish normal from abnormal findings on a neurologic exam</td>
<td>Know how to Can do</td>
<td>Student mastery</td>
<td>Performance assessments, small group discussions</td>
<td>Skill</td>
</tr>
<tr>
<td>Localize the likely site(s) of nervous system disturbance to account for symptoms and signs</td>
<td>Know how to Can do</td>
<td>Intermediate</td>
<td>Performance assessments, small group sessions, multiple choice exam</td>
<td>Skill</td>
</tr>
<tr>
<td>Formulate a differential diagnosis based on lesion localization, time course, and relevant historical and demographic features</td>
<td>Know how to Can do</td>
<td>Intermediate</td>
<td>Performance assessments, small group sessions</td>
<td>Skill</td>
</tr>
</tbody>
</table>
### Analytical skills objectives (cont.)

<table>
<thead>
<tr>
<th>Use and interpret common tests used in diagnosing neurologic diseases</th>
<th>Know how to</th>
<th>Introductory</th>
<th>Performance assessments, small group sessions</th>
<th>Skill</th>
</tr>
</thead>
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<tr>
<td>Demonstrate a systematic approach to the management of common neurologic diseases (including emergency situations)</td>
<td>Know how to</td>
<td>Introductory</td>
<td>Direct observation during inpatient rounds, outpatient clinics, small group sessions, multiple choice exams</td>
<td>Skill</td>
</tr>
<tr>
<td>Recognize situations in which it is appropriate to request neurologic consultation</td>
<td>Knows</td>
<td>Intermediate</td>
<td>Direct observation during inpatient rounds, outpatient clinics, small group discussions</td>
<td>Knowledge</td>
</tr>
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Content of Subjects to be Learned

A. The neurological examination (as an integral component of the general medical examination)
   1. how to perform a focused but thorough neurologic examination
   2. how to perform a screening neurologic examination
   3. how to perform a neurologic examination on patients with an altered level of consciousness
   4. how to recognize and interpret abnormal findings on the neurologic examination

B. Localization - general principles differentiating lesions at the following levels:
   1. cerebral hemisphere
   2. posterior fossa
   3. spinal cord
   4. nerve root/plexus
   5. peripheral nerve (mononeuropathy, polyneuropathy and mononeuropathy multiplex)
   6. neuromuscular junction
   7. muscle

C. Symptom Complexes – a systematic approach to the evaluation and differential diagnosis of patients who present with:
   1. focal weakness
   2. diffuse weakness
   3. clumsiness
   4. involuntary movements
   5. gait disturbance
   6. urinary or fecal incontinence
7. dizziness
8. vision loss
9. diplopia
10. dysarthria
11. dysphagia
12. acute mental status changes
13. dementia
14. aphasia
15. headache
16. focal pain
   a. facial pain
   b. neck pain
   c. low back pain
   d. neuropathic pain
17. numbness or paresthesias
18. transient or episodic focal symptoms
19. transient or episodic alteration of consciousness
20. sleep disorders
21. developmental disorders

D. **Approach to Specific Diseases - general principles for recognizing, evaluating and managing the following neurologic conditions (either because they are important prototypes, or because they are potentially life-threatening):**

1. potential emergencies
   a. increased intracranial pressure
   b. toxic-metabolic encephalopathy
   c. subarachnoid hemorrhage
   d. meningitis-encephalitis
   e. status epilepticus
   f. acute stroke (ischemic or hemorrhagic)
g. spinal cord or cauda equina compression
h. head trauma
i. acute respiratory distress due to neuromuscular disease
j. temporal arteritis

2. strokes
3. seizures
4. Alzheimer’s disease
5. Parkinson’s disease
6. essential tremor
7. multiple sclerosis
8. migraine
9. Bell’s palsy
10. carpal tunnel syndrome
11. diabetic polyneuropathy
12. brain death

Guidelines for a Comprehensive Neurologic Examination

A. Mental Status
   1. level of alertness
   2. language function (fluency, comprehension, repetition, naming)
   3. memory (short-term and long-term)
   4. calculation
   5. visuospatial processing
   6. abstract reasoning

B. Cranial Nerves
   1. vision (visual fields, visual acuity and funduscopic examination)
   2. pupillary light reflex
   3. eye movements
   4. facial sensation
5. facial strength (muscles of facial expression)
6. hearing
7. palatal movement
8. speech
9. neck movements (head rotation, shoulder elevation)
10. tongue movement

C. Motor Function
1. gait (casual, on toes, on heels, tandem gait)
2. coordination (fine finger movements, rapid alternating movements, finger-to-nose, heel-to-shin)
3. involuntary movements
4. pronator drift
5. tone (resistance to passive manipulation)
6. bulk
7. strength (shoulder abduction, elbow flexion/extension, wrist flexion/extension, finger flexion/extension/abduction, hip flexion/extension, knee flexion/extension, ankle dorsiflexion/plantar flexion)

D. Reflexes
1. deep tendon reflexes (biceps, triceps, brachioradialis, patellar, Achilles)
2. plantar responses

E. Sensation
1. light touch
2. pain or temperature
3. proprioception
4. vibration

Guidelines for a Screening Neurologic Examination

A. Mental Status (level of alertness, appropriateness of responses, orientation to date & place)
B. Cranial Nerves
1. visual acuity
2. pupillary light reflex
3. eye movements
4. hearing
5. facial strength (smile, eye closure)

C. Motor Function
1. gait (casual, tandem)
2. coordination (fine finger movements, finger-to-nose)
3. strength (shoulder abduction, elbow extension, wrist extension, finger abduction, hip flexion, knee flexion, ankle dorsiflexion)

D. Reflexes
1. deep tendon reflexes (biceps, patellar, Achilles)
2. plantar responses

E. Sensation (one modality at toes – can be light touch, pain/temperature or proprioception

*Guidelines for the Neurologic Examination in Patients with Altered Level of Consciousness*

A. Mental Status
1. level of arousal
2. response to auditory stimuli (including voice)
3. response to visual stimuli
4. response to noxious stimuli (applied centrally and to each limb individually)

B. Cranial Nerves
1. response to visual threat
2. pupillary light reflex
3. oculocephalic (doll’s eyes) reflex
4. vestibulo-ocular (cold caloric) reflex
5. corneal reflex
6. gag reflex

C. Motor Function
1. voluntary movements
2. reflex withdrawal
3. spontaneous, involuntary movements
4. tone (resistance to passive manipulation)

D. Reflexes
1. deep tendon reflexes
2. plantar responses

E. Sensation (to noxious stimuli)

The following pages give the necessary instructions and schedules for the course. This material is followed by reading assignments and the case histories for discussion in the didactic sessions. There is also useful additional information to serve as a guideline to major clinical material, neuroanatomy, neuroepidemiology and, especially, the neurologic examination.

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Code of Professional Conduct for Students of
Saint Louis University School of Medicine’s MD Degree Program

I. Preamble

Saint Louis University School of Medicine, as part of its mission of education, will assist students to become competent physicians who manifest in their lives exemplary ethical and professional attitudes. Among these are:

- respect for the sanctity of human life
- commitment to competence
- respect for the dignity of patients in the provision of medical care
- devotion to social justice, and to addressing inequalities in the availability of health care
- personal humility and an awareness of medicine’s inherent limitations
- appreciation of the role of non-medical factors in a patient’s state of well-being or illness
- maturity and balanced personal and professional behavior.

Students entering the MD program should realize the importance of this professional ethic and the necessity of assuming responsibility to develop, review, and maintain these ideals for themselves, their colleagues, and for the medical profession. The fundamental principle involved is personal responsibility for the development and maintenance of professional conduct based on the ethic espoused by the medical profession of self-effacing service to society. Development of a professional ethic must begin with admission to the MD program, and must continue for as long as students of medicine live and represent the medical profession.

The Code of Professional Conduct is founded on a long tradition extending at least from the Hippocratic School. This tradition acknowledges the unique privileges and responsibilities of the medical profession. The individual deprived of perfect health becomes vulnerable and dependent on the knowledge entrusted to the healing professions who have pledged that the welfare and autonomy of the patient takes precedence over the
self-interest of the healer. The Code speaks to the relationship of the physician and patient, as well as to the relationship of trust and respect among students, faculty, staff, and society in general.

The Code of Professional Conduct extends recognition of the sponsoring influence of the Catholic faith and The Society of Jesus which conceives of the person as a free and responsible agent capable of making a difference for good or ill in the world. Students of medicine must develop as critically reflective and socially responsible persons capable of exercising leadership in advancing the cause of human good.

The Code of Professional Conduct for students is not intended to dictate behavior. It does, however, establish minimum expectations which provide a disciplinary framework for those who choose not to abide by these professional standards, and it serves to remind everyone that a procedural framework for enforcing the code is in place.

II. The Code of Professional Conduct for Students

I will conduct myself so that as a student of medicine and member of the profession of medicine, I will be able to promise those for whose care I am entrusted, competence, integrity, candor, personal commitment to their best interests, compassion, and absolute discretion and confidentiality.

I shall do by my patients as I would be done by, shall obtain consultation when they desire or I believe there is a need, shall include them to the extent possible in all important decisions, and shall minimize suffering when a cure is not attainable, recognizing that a dignified death is an important goal in everyone’s life.

I shall accept all patients in a non-judgmental manner, respecting the full human dignity of each individual regardless of their value system. I shall at all times and in all places conduct myself with honor and integrity, and I shall respect the rights and dignity of all individuals. I promise to attend to my own physical, intellectual, mental, and spiritual development in the interest of best serving others.

III. Expectations

The following statements are examples of the School of Medicine’s minimum expectations for students.
A. Respect for all individuals in the School of Medicine’s community.

Students, faculty, and staff recognize the right of all individuals to be treated with respect without regard to position, race, age, gender, handicap, national origin, religion, or sexual orientation.

Sexual harassment is prohibited by Title IV Civil of the Rights Act of 1964 and by Title IX of the Education Amendments of 1972. The School of Medicine shall take steps reasonably necessary to prevent sexual harassment from occurring, which will include, but is not limited to: providing a process for filing and handling complaints; educating the medical school community; affirmatively raising the subject and expressing strong disapproval thereof; developing appropriate sanctions; informing faculty, staff, and students of their rights and developing appropriate sanctions; informing faculty, staff, and students of their rights and developing methods to sensitize all concerned. (For complete policy and procedure, see Saint Louis University’s general student rules and regulations published in the Saint Louis University Handbook.)

B. Appropriate handling of information, records, or examination materials

Any form of cheating or providing false information is a violation of the trust placed in physicians and is a serious infraction of the Code of Professional Conduct.

C. Respect for patients’ confidentiality and safety.

Patients’ privacy, modesty, and confidentiality must always be honored. Patients must be treated with kindness, gentleness, dignity, empathy, and compassion. Patients should not be publicly identified without adequate justification or permission. Patients’ records must be accurate and legible.

D. Proper deportment as a medical professional.

Appearance, speech, and behavior should be above reproach during patient care activities. Identification badges issued by the School and by affiliate hospitals should be clearly displayed. Dress should be appropriate for professional activities, and personal hygiene should be exemplary.
Possession or use of stimulants, depressants, narcotics, or hallucinogenic drugs and other agents having potential for abuse, except on a physician’s or dentist’s prescription, are forbidden by medical school policy and local, state, and federal law. Such conduct is prohibited on Saint Louis University’s premises or as part of any university activities. The selling, bartering, exchanging, or giving away of such drugs to any person is illegal and prohibited. Students engaged in the use of illegal substances will be subject to arrest by law enforcement agents and will seriously jeopardize their status as students at this School. (For complete policy and procedure, see Saint Louis University’s general student rules and regulations published in the Saint Louis University Student Handbook.)

E. Respect for laws, policies, and regulations.

Students are expected to recognize that laws are established for the benefit of society as a whole. Specific rules, policies, and regulations have been established for the benefit of the School of Medicine’s community. Laws, policies, and regulations are not to be disregarded or violated. Students who feel that the policies, procedures, or regulations of the School require modification are encouraged to suggest improvements to appropriate administrative, faculty, or student offices.

F. Respect for property and instructional material.

All property and instructional material must be respected.

IV. Code Violations

A violation of the Code of Professional Conduct occurs when any student acts contrary to the values and responsibilities expected of those engaged in the profession of medicine. Violations also occur when any student jeopardizes the welfare of a patient, or disregards the rights or dignity of another individual, or allows or assists another in doing so. For example, students are subject to discipline under the Code if they have knowledge of an infraction of the Code, but fail to report it to the appropriate authority.

Appendix A lists examples of unacceptable behavior under each category of expectations. The School of Medicine reserves the right to initiate action and impose sanctions for any conduct that is not specifically listed in Appendix A, but is determined to be a violation.
of the Code, regardless of whether the violation occurs on or off the premises of the School.

V. Procedures for Investigating Code Violations

The School of Medicine has a responsibility to ensure a professional climate and to provide equity for all parties involved in a breach of professional conduct. Therefore, the School has adopted procedures to respond to infractions of the Professional Conduct Code. Individuals reporting an infraction of the Code can first speak with offenders about the infraction and remind them of their professional conduct. However, if the circumstances do not allow the claimant to approach the offender, and the claimant believes the alleged infraction requires further action, the allegation may be referred to the Student Professional Conduct Council, after consultation with one of the Assistant Deans for Students.

A. Student Professional Conduct Council

The Student Professional Conduct Council (SPCC) is empowered to investigate violations of the Code of Professional Conduct and to impose sanctions or remediation. The SPCC will consist of a chairperson (non student member), two students selected by the Dean from the second and third year classes, one faculty member, and one member from the Dean’s staff. All members are appointed by the Dean of the School of Medicine. Student terms begin in Year 2 and terminate at the end of their third academic year. Faculty, administrative staff, and the chairperson will serve for four years. In the event that a student leaves the Council, a temporary vacancy should be filled by the Dean. In an emergency event where the designated student members cannot be gathered at a specific time, the position(s) may be filled by alternates named by the Dean. In the absence of the chairperson, meetings shall be chaired by a vice chairperson appointed by the Dean among non-student members. A vacant faculty or administrative position may be filled at the discretion of the Dean of the School of Medicine. In the event a vacancy is only temporary, it is understood that the member appointed to the SPCC shall be bound by the same charges of confidentiality as the other members of SPCC. If a temporary member has been appointed, this member will remain on the SPCC until the review has
been completed. Minutes will be kept of all deliberations, and are the responsibility of the chairperson or a member designated by the chairperson.

**B. Procedure to Investigate an Infraction by the Student Professional Conduct Council**

1. Students, faculty, or staff making an allegation should do so to one of the Assistant Deans for Students, who will make every effort to resolve the issue(s) at an informal level within ten working day. If it is determined that the issue cannot be resolved at an informal level, the assistant Dean will contact the chairperson of the Council.

Should a complaint suggest a threat to the safety and welfare of patients or other members of the School of Medicine’s community, at the discretion of the Assistant Dean for Students, the accused student may be placed on an interim suspension from academic activities until a full investigation is complete. Students accused of violating local, state, or federal laws may be arrested and referred to law enforcement authorities for appropriate action. The School reserves the right to pursue disciplinary action pursuant to the Code, regardless of where the conduct occurred, and independent of any civil or criminal proceedings.

2. If a mutually agreeable solution is not reached between the complainant and the Assistant Dean for Student Affairs, the Assistant Dean will ask the chairperson of the SPCC to proceed with convening a meeting within ten working days.

3. The Assistant Dean for Student Affairs will present a written report to the SPCC including the following:

   - a description of the complaint with pertinent documentation, if any;
   - channels already pursued to resolve the issue(s)
   - results of previous discussion/action

4. The SPCC will review the information received and interview pertinent individuals, including the accused student. Should the student accused of a violation fail to appear, the SPCC will make its decision based on the information presented.
Students may have an advisor present to assist them in proceedings before the SPCC. The advisor’s participation is limited to consulting with the student. Since the proceedings before the SPCC are not legal proceedings, a student will not be allowed to have legal counsel serve as an advisor unless criminal charges arising out of the same conduct are currently pending.

5. After all testimony has been received, the SPCC will proceed in private for the purpose of deliberation.

6. The SPCC will conclude that a violation of the Code has taken place by a majority vote of the members present. The SPCC will consider at least the following options:

   • written censure by the SPCC maintained in a confidential file in the Registrar’s office separate from the student’s permanent file;
   • recommendation of required restitution action;
   • placement of a letter concerning the incident in the student’s permanent file;
   • suspension or dismissal from the School of Medicine.

7. If the recommendation for dismissal is not unanimous, a minority report will also be submitted.

8. The recommended course of action will be communicated in writing to the student and the Dean. If dismissal is recommended for the student, the School of Medicine Policy and Procedure for student dismissal will apply. A student who is dismissed from the School of Medicine may appeal that decision to the School’s Appeals Council. Any other appeal will be handled through existing policies.

9. A student’s failure to comply with the sanctions imposed under the Code may result in further disciplinary action including dismissal.
APPENDIX A

Examples of Unacceptable Behaviors

A. Respect for all individuals in the School of Medicine’s community.

Unacceptable behavior includes (but is not limited to):

- expressing racial, sexual, sexist or religious slurs
- committing racial or sexual harassment
- using inappropriate, offensive or threatening language
- committing physical acts of violence or threats of violence
- not shouldering a fair share of the educational team responsibility
- manipulating clinical schedules for one’s own benefit
- causing or encouraging disruptions during educational sessions
- showing lack of respect to technicians, other professionals, and support staff
- failing to comply with a reasonable request or instruction from faculty, staff or administrators
- using computer e-mail or internet in a harassing or libelous manner

B. Appropriate handling of information, records, or examination materials

Unacceptable behavior includes (but is not limited to):

- giving or receiving any information except as allowed by the course director or teaching faculty during the course of an exam
- plagiarizing, forging, or falsifying academic records, financial aid information, patient records, research, or scientific data
- tampering with examination material, or dishonesty in connection with an examination
- abusing computerized information or technology

C. Respect for patients’ confidentiality and safety
Unacceptable behavior includes (but is not limited to):

- failing to ask for assistance from appropriate faculty or staff when needed
- writing offensive or judgmental comments in the patient’s chart
- sharing medical or personal details of a patient with anyone other than health professions who need to know for the patient’s care
- engaging in discussion about a patient in public areas

D. Proper deportment as a medical professional.

Unacceptable behavior includes (but is not limited to):

- inducing or allowing the patient or others to believe ones’ status as a student is other than it is
- exhibiting personal appearance that gives the impression of uncleanness or carelessness
- failing to maintain professional composure during stressful circumstances
- engaging in an inappropriate relationship with patients or their family members
- using alcohol, drugs, or other substances inappropriately or excessively, or in a way that could affect the quality of patient care or academic performance

E. Respect for laws, policies, and regulations

Unacceptable behavior includes (but is not limited to):

- disobeying federal, state, or local laws and ordinances
- disregarding or acting contrary to institutional regulations and policies
- refusing to provide information or testify in Professional Conduct Council proceedings (see Section V.A.).

F. Respect for property and instructional material

Unacceptable behavior includes (but is not limited to):

- treating cadavers and body parts in a disrespectful manner
• treating animals used for instruction in an insensitive or disrespectful manner
• defacing or destroying other instructional materials, including software
• defacing or destroying University or personal property, or any written material other than one’s own
• removing information, mail, or property from mailboxes or lockers not one’s own
• falsifying or defacing transcripts, evaluation forms, or other official documents.
Instructions to clinical neurosciences students

Day 1

This 8 week block will entail 4 weeks each of Neurology and Psychiatry. The first day of the rotation is orientation. The morning is orientation for the psychiatry segment and the afternoon is for the neurology segment. For most of the rotations, the neurology orientation will begin at 1:00 pm with a review of the goals and expectations. A neuroanatomy review session given by Dr. Paul Young will occur from 1:30-3:15. A review of the neurological examination by an upper-level neurology resident will occur from 3:15-4:00. The “localization case review” will occur from 4:00-5:00pm. Meeting sites for morning rounds beginning at 8:00 am on the second day of the rotation are University Hospital 5th floor, Cardinal Glennon Children’s Hospital 2 south conference room, St. Mary’s Hospital 6th floor, and John Cochran VA Hospital room A634.

Responsibilities

Clinical activities during the neurology rotation will include ward rounds, outpatient clinics, case-based didactic sessions, instructional slide quiz, observation of exams skills, neurology department grand rounds, evening and weekend call, and provision of time available for independent reading and for completion of the multiple-choice quiz on Blackboard.

Patients assigned to students are part of their continuing clinical responsibility. Students are to attend daily rounds (6 days per week with either Saturday or Sunday free), present initial findings and daily developments to the attending physician, and participate in discussions pertaining to diagnostic and therapeutic maneuvers, discharge plans and follow-up programs. Students are expected to conduct a thorough neurologic examination, including completion of the neurologic check sheet, sensory, optic fundus and visual field examinations for all patients that they present to the attending neurologist. An observed neurologic examination must be documented in the FACTS form. The student is expected to evaluate a minimum of 2 patients per week. For patients admitted in off-hours, the students need to determine in advance who is
responsible to present these patients on morning rounds and to come in earlier to see these admitted patients if more time is needed to evaluate them. Students must select at least 2 supplemental experiences (clinics and EMG/NCV sessions) to attend during the 8 weeks. The sign-up sheet is posted in Ms. Grubb’s office (room 118 in the Department of Neurology and Psychiatry at 1438 S. Grand). Neurology case discussion sessions are also mandatory and may partially reduce time spent in the clinic. It is not feasible for each student to encounter all of the more common neurological disorders in a 4 week period of time. In order to expose students to the common disorders, students attend a series of case-based discussions of symptoms and signs found in these disorders. Students are expected to conduct themselves in a courteous and professional manner at all times. The student conduct code must be strictly adhered to.

Evening and Weekend Call

In order to enhance exposure to assessment and management of acute neurologic problems, students are given an evening call until 9:00 pm and a weekend call until 6:00 p.m. at the University Hospital/Cardinal Glennon Children’s Hospital (the neurology resident covers both hospitals). While on call, students on an adult service should try to see pediatric consultations/admissions and vice-versa. It is the student’s responsibility to notify the neurology resident on call that he/she is on call for that evening. In order to derive the greatest benefit from time spent on call, students should seek out and accompany the resident during the various work tasks, which will include admissions, consults, and emergency room calls. Call starts when the students’ responsibilities at their assigned sites are complete.

Requirements to pass

1. Full attendance at daily clinical rounds. For extenuating circumstances, attendance on rounds may be excused by the assigned faculty supervisor on a case-by-case basis, but must be “made-up” if more than 3 work days are missed. Official University holidays are Labor Day, Memorial Day, and Thanksgiving (to be dismissed by 6:00 pm Wednesday
and return on Monday morning for rounds). The clerkship ends following the final written examination on the 8th Friday of the rotation. Ward responsibilities end on the 8th Wednesday after morning rounds. You must receive a pass, near honors, or honors evaluation for clerkship performance by the assigned faculty supervisor(s). The clerkship experience is intended to acquaint the student with skills in obtaining a neurologic history and techniques of the neurologic examination along with the general principles of neurologic diagnosis and management. The areas of evaluation include: knowledge of neurology, history taking, neurologic examination, organizing and recording data, communication skills, differential diagnosis, diagnostic and therapeutic program planning, professionalism, motivation and attitude, patient rapport, and healthcare team rapport. The FACTS form must be completed, including documentation of an observed exam. Each student should undergo a performance review with his/her attending half-way through the rotation in order to receive guidance for improvement, and again at the end of the rotation. These feedback encounters must be documented in the FACTS form.

2. Each student must attend at least 2 supplementary neurology experiences (mostly clinics) and have an evaluation card completed by the attending physician for each of these sessions.

3. Full attendance is expected at all didactic sessions. These are case-based discussions covering many of the major neurological conditions you should learn (stroke, epilepsy, headache, CNS infections, neuromuscular diseases, toxic/metabolic conditions, coma). You should read the specified cases from the Blueprints in Neurology clinical cases textbook prior to each session.

4. One evening and one weekend call are assigned to provide opportunity for involvement in acute neurological problems in the ER and other inpatient situations. The resident on call should initial the FACTS card to document your on-call attendance.

5. A multiple choice quiz on Blackboard must be completed by each student. This is provided as an additional learning experience and as practice for the NBME neurology subject examination. To access the quiz, go to http://gateway.slu.edu/cp/home/login and enter your SLU Net ID and password, then click login. Click on “tools”. Click on Blackboard Learning System. Click on NE-G301. Click on self test. Start the quiz.
6. Each student must achieve a passing or honors grade on the final written examination (NBME neurology subject exam) which is administered at 9:45 am - 12:00 pm on the final Thursday of the course. It serves to measure the student’s degree of attainment of the content objectives and will account for 40% of the final grade. A score of less than 5th percentile will result in a deferred grade pending the successful completion of a repeat subject examination at the end of the academic year. The student will acquire knowledge to attain this objective through a combination of methods including exposure on rounds and in clinic, didactic sessions, and independent reading.

**Quantifying Criteria**

Students will be expected to personally evaluate in full a minimum of 2 new patients per week. Evaluation will include eliciting a complete history, performing comprehensive general and neurological examinations, and presenting the information in written and verbal forms. In addition, students must perform each component of the neurological examination while being directly observed by a neurology resident or attending physician in order to assure proper technique. Each student must see, read about, or discuss at least one patient from each of the 6 major categories of neurologic disorders and be prepared to discuss the differential diagnosis for each one. These major categories include cerebrovascular disorders, degenerative disorders, dementia, headache and other pain syndromes, neuromuscular disorders, and seizure disorders

**FACTS (Feedback and Clinical Tracking System)**

The FACTS program is designed to ensure that during your core clerkships, you

1. Demonstrate competency in key clinical skills
2. Encounter essential clinical problems and/or diagnoses
3. Receive regular and timely feedback regarding your performance (on knowledge, skills, and behavior) from faculty.
You will receive a copy of the FACTS form and further instructions at your clerkship orientation. Completion of the FACTS form is a required component of each clerkship, beginning in AY 2007-2008. We strongly advise you to make a copy of your FACTS form periodically, in the unfortunate event that you lose your form. Extra copies may be available, but it is your responsibility to make sure that the form is completed and turned in to the clerkship director at the end of your rotation. Clerkship directors will use your forms in considering your level of professionalism.
Recommended textbooks

In order to gain a comprehensive familiarity with general neurology, students are required to read the following textbook:

**Blueprints in Neurology, clinical cases**

By Sheth, et al., Lippincott Williams & Wilkins, 2007

This text is available in Matthews Medical Bookstore for about $30 and is concise enough to be completed in a 4-week time frame. This book is comprised of 60 clinical cases with thought questions, answers, and discussions as well as 81 mini-cases with discussions in the review Q&A section. For each of the didactic sessions, all of the relevant cases have been identified for you to review before the session, which will help you to participate in the case-based discussions. Some of these cases are listed more than once, as they apply to more than one topic. Also, read the cases to be discussed, most of which are in this handbook.

There are a number of clinical subspecialty textbooks in neurology, eg. cerebrovascular disorders, epilepsy, peripheral nerve and muscle disorders, neurocritical care, neurophysiology and neuro-ophthalmology. Some of these can be found in the faculty/resident library in the department of neurology and psychiatry administrative offices. You are welcome to consult them there.

Schedule of activities

Morning hours are spent preparing for and attending rounds and completing patient-related activities. Afternoons are spent in clinics, case-based discussions, completing patient care, and occasionally attending evening rounds. Unassigned hours should be used for independent study, necessary hospital work such as follow-up of cases or diagnostic procedures, informal discussions with residents or faculty and interchanges with other health care professionals such as nurses, social workers, and therapists.
**Conclusion**

At the end of the clerkship, the student is expected to be able to:

1. Recognize neurologic dysfunction in any part of the peripheral or central nervous system.

2. Formulate a hypothesis which localizes the disorder and accounts for the pathologic and pathophysiologic nature of the derangement.

3. Possess the basic information needed to pursue further diagnosis of a neurologic condition, prescribe treatment in an orderly and logical manner, and seek selected information from reference sources.

4. Be acquainted with the management of major neurological, neurosurgical, and neuro-ophthalmological emergencies, including spinal cord compression, status epilepticus, neuromuscular respiratory failure, impending or completed stroke, brain herniation, acute visual loss, and acute diplopia.

5. Be familiar with the prevalence and incidence of common neurologic diseases.

Please Note: Students are expected to dress appropriately at all times, including a white jacket.
DEPARTMENT OF NEUROLOGY FULL-TIME FACULTY

Aninda Acharya, M.D. Dr. Acharya gained both his bachelor’s degree in 1992 and his medical degree in 1996 from the University of Kansas. He completed his neurology residency at Washington University in 2000. Afterward, he continued there as a stroke fellow. He began as a full-time faculty member in July, 2002. His principal interests are in methods promoting recovery and rehabilitation of the brain.

Wegdan Andrews, M.D. Dr. Andrews received her medical degree from Asyut University in 1992, and then completed her internship year at Michigan State University. She trained in neurology and neurophysiology at the University of Alabama, Birmingham from 2001-05 and subsequently joined the St. Louis University neurology faculty as an Assistant Professor.

Deepa Arun, M.D. Dr. Arun graduated from Sri Ramachandra Medical College and Research Institute in 1997. She completed a pediatric residency at Cooper Hospital/University Medical Center in New Jersey, then pediatric neurology residency at Washington University in St. Louis. She has been practicing pediatrics and pediatric neurology at the Southern Illinois Healthcare Foundation since 2006 and joined the St. Louis University neurology faculty in 2009.

Steven R. Brenner, M.D. Dr. Brenner graduated from Kansas State University in 1968 with a Bachelor of Science degree. He received his medical degree from the University of Kansas in 1972. Later, he trained in neurology at Saint Louis University and remained as a member of the faculty. He is currently an Assistant Professor of Neurology. His primary clinical and teaching activities take place at the DVAJCH. He has an ongoing research interest in the effect of aluminum on neuronal cell cultures.

William J. Burke, M.D., Ph.D. Dr. Burke received his Bachelor of Science degree from Marquette University. He holds both Medical and Doctor of Philosophy (biochemistry) degrees from Saint Louis University (1972). He also was a neurology resident at Saint Louis University Hospital and became a member of the faculty upon completion of his training in 1976. He is currently a full Professor of Neurology and conducts basic research in neuronal cell death and the biochemistry of Alzheimer’s brains at the DVAJB.
**Randall Edgell, M.D.** Dr. Edgell graduated from the Baylor College of Medicine in 2000, subsequently training in neurology at New York University. After completing fellowship training in stroke and in endovascular neurology, he joined the faculty of St. Louis University in 2007 and is currently an assistant professor. His areas of special interest include endovascular stenting and developing tools for multi-institutional data collection.

**Isaac Faibisoff, M.D.** Dr. Faibisoff received his medical degree from Rush University in Chicago and subsequently underwent neurology residency training at the same institution. He practices general neurology and is also a flight surgeon for the Air force reserves. He is board certified in medical acupuncture. He joined the Saint Louis University neurology faculty in 2007 and sees patients at the John Cochran Veterans Administration Hospital.

**Eli Feen, M.D.** Dr. Feen obtained his medical degree from the University of Maryland, subsequently completing a neurology residency at Case Western University. He then completed a 2-year fellowship in stroke and neurocritical care. He joined the neurology faculty of St. Louis University in 2007, coming from the University Hospitals of Cleveland, where he was interim director of the neurocritical care unit. His area of special interest relates to ethical dilemmas involved in neurocritical care.

**Pratap Chand, MD** Dr. Chand has had medical/neurology training in India, Ireland, and most recently in movement disorders at the University of Louisville in Kentucky. He has extensive experience in clinical neurology practice worldwide, including Malaysia, Oman, United Kingdom. He joined the neurology department faculty of St. Louis University in 2008. He specializes in movement disorders.

**Salvador Cruz-Flores, M.D.** Dr. Cruz-Flores took his undergraduate studies at the Autonomous University of Nuevo Leon in his native Mexico where he subsequently received his degree of a Doctor of Medicine. He first trained in Internal Medicine and Neurology in the same institution until 1992. He then trained in Neurology at Saint Louis University Hospital, where he subsequently completed a fellowship in Cerebrovascular Diseases and Intensive Care. He joined the program in December, 1997.
Glen Fenton, M.D. In 1981, Dr. Fenton received his degree of Bachelor of Arts from the University of Colorado. He then attended Saint Louis University School of Medicine, graduating in 1985. His pediatric residency and child neurology residency were both completed at Cardinal Glennon Children’s Hospital. After a fellowship in clinical neurophysiology and board certification, he joined the neurology faculty in 1992.

Thomas Geller, M.D. Dr. Geller took his four years of undergraduate studies at Notre Dame University. He received his Doctor of Medicine degree from Saint Louis University in 1980. He trained in pediatrics at the Naval Hospital, Oakland, CA, following which he became a child neurology resident at the University of Illinois, Chicago, IL. Before becoming Assistant Professor in Neurology and Pediatrics at Saint Louis University, he was Staff Pediatric Neurologist at the USAF Medical Center, Wright-Patterson AFB, OH, with a concurrent appointment as Assistant Clinical Professor of Neurology, Wright State University.

Ghazala Hayat, M.D. Dr. Hayat received her Bachelor of Science degree from Punjab University in 1975 and her medical degree in 1977 from King Edward Medical College, both in her native country of Pakistan. She trained in neurology at the Medical College of Virginia, where she also completed a fellowship in neuromuscular diseases. In 1986, she joined the faculty at Saint Louis University. She is an Associate Professor of Neurology and is the Director of the Neuromuscular Service in the SLUH.

Henry Kaminski, M.D. After graduating from Case Western Reserve Medical School in 1985, Dr. Kaminski was trained in neurology at the University Hospitals of Cleveland. He underwent subspecialty training in neuromuscular disorders and is internationally recognized as a leading authority in disorders of neuromuscular transmission. He assumed the position of Chairman of the Department of Neurology and Psychiatry at St. Louis University Medical Center in 2007.

Laurence Kinsella, M.D. Dr. Kinsella received his medical degree from Saint Louis University in 1981, and completed his residency in neurology at Brown University in 1991, and a fellowship in neuromuscular disorders at Presbyterian Hospital in New York in 1992. He is currently an attending physician at Forest Park Hospital and a member of
the faculty and attending physician in the department of neurology at Saint Louis University Hospital.

**Raman Malhotra, MD**  
Dr. Malhotra obtained his medical degree from the University of Missouri-Kansas City in 2000. He then completed a neurology residency at the University of Michigan in 2004 and went on to obtain fellowship training in Clinical Neurophysiology and Sleep Medicine at the same institution. He joined the Neurology faculty at St. Louis University in 2008. He specializes in disorders of sleep.

**John McGarry, M.D.**  
Dr. McGarry obtained his Bachelor of Science degree from the University College in Ireland in 1970. Later in 1978 he obtained his medical degree from the National University of Ireland. Trained in neurology at Saint Louis University, Dr. McGarry has been on the faculty for a number of years, and became a full Clinical Professor in 1977. His primary clinical responsibilities in the Spinal Cord Unit at DVAJB.

**Francis Mithen, M.D., Ph.D.**  
In 1967, Dr. Mithen received his Bachelor of Science degree from Tufts College. Later, in 1971, he received his Doctor of Medicine degree from Columbia University in New York and, in 1981, his Doctor of Philosophy degree (neurobiology) from Washington University, where he subsequently trained in neurology. He joined the faculty of Saint Louis University in 1983 and is currently an Associate Professor of Neurology. His clinical activities are mostly conducted in the DVAJCH.

**Yi Pan, M.D.**  
Dr. Pan received her medical degree from Beijing Medical College in 1982. After serving as a Postdoctoral Fellow in cerebral ischemia at the Massachusetts General Hospital in Boston, she came to Saint Louis University to complete a residency in neurology in 1997 followed by a fellowship in clinical neurophysiology. She joins the department of neurology as a clinical neurologist and stroke researcher in August, 2000.

**Paisith Piriyawat, M.D.**  
Dr. Piriyawat received his medical degree in 1992. His neurology residency was completed at Saint Louis University in 2000. Subsequently, he pursued a stroke fellowship at the University of Texas at Houston. He is certified by the American Board of Psychiatry and Neurology for Neurology and Vascular Neurology (Stroke). His works focus on treatment of acute ischemic and hemorrhagic stroke.
Amy Rauchway, D.O.  After completion of neurology residency training at Saint Louis University, Dr. Rauchway joined the faculty. She earned her Bachelor of Science degree at Cornell University, and her medical degree at Western University of Health Sciences, College of Osteopathic Medicine in California. Her areas of special interest are multiple sclerosis and neurorehabilitation. Dr. Rauchway assumed the position of Residency Program Director for the Division of Neurology in July 2008.

John Selhorst, M.D. Dr. Selhorst attended Spring Hill College until 1963 when he received his Bachelor of Science degree. He graduated in 1967 from Saint Louis University School of Medicine. He trained in internal medicine at Saint Louis University, neurology at Case Western Reserve and neuro-ophthalmology at the University of California, San Francisco. After a ten-year career at the Medical College of Virginia, he served as the Chairman of Neurology at St. Louis University from 1985 until 2007.

Florian Thomas, M.D., Ph.D. Dr. Thomas obtained degrees in medicine, psychology and microbiology from the University of Bonn, Germany, in 1982/1983, which included training at Harvard University and Case Western, where he later completed neurology training under the tutelage of Dr. Maurice Victor. After 3 years of research in neuroimmunology at Columbia University, he trained in AIDS neurology at the Montreal Neurological Institute and obtained a PhD in experimental medicine from McGill University. In 1995, he joined the Saint Louis University (SLU) Department of Neurology with secondary appointments in molecular microbiology and immunology and the institute for molecular virology. In 2005 he was promoted to the rank of Professor of Neurology at SLU and Director of the Spinal Cord Injury/Dysfunction at the Saint Louis VA Medical Center (VAMC). He also directs the MS Society affiliated multiple sclerosis centers at SLU and the VAMC. His clinical and research interests focus on MS/Neuroimmunology, neurogenetics, neuromuscular diseases, spinal cord injury, AIDS, rehabilitation, sexual health, and pain management.

Alpa Vashist, M.D. Dr. Vashist is an assistant professor in neurology and pediatrics, having joined the faculty of St. Louis University in 2006. After completing her medical school training in India, she completed her pediatric neurology residency and clinical
neurophysiology fellowship at State University New York. Her area of special interest is pediatric epilepsy.

**Nirav Vora, M.D.** Dr. Vora received his MD from the University of Alabama. He then underwent neurology residency training at the Cleveland Clinic. Most recently he had fellowship training in vascular neurology and interventional neuroradiology/endovascular neurosurgery at the University of Pittsburgh. He joined the Neurology faculty at St. Louis University in 2008. He specializes in neurovascular disorders, including endovascular intervention.

**David Walsh, M.D.** After receiving his BS in chemistry from Georgetown University in 1968, Dr. Walsh attended the Medical University of South Carolina, from which he graduated in 1973, thereafter staying on for training in child neurology. He has completed additional fellowships in neurophysiology at Harvard and in medical education at the Medical College of Wisconsin. He joined St. Louis University’s department of neurology in 2004 as the director of pediatric neurology at Cardinal Glennon Children’s Hospital.

**L. James Willmore, M.D.** After receiving his undergraduate and medical degrees from Saint Louis University and serving in the U.S. Navy, Dr. Willmore completed his residency in neurology at University of Virginia. His subsequent clinical and research interest was as an epileptologist, first at the University of Florida, Gainsville, and then in 1983 at the University of Texas-Houston’s School of Medicine. In 1999, he joined the department of neurology of Saint Louis University as a faculty member specializing in epilepsy and also serves as an Associate Dean for admissions to the School of Medicine.
### Neurology residents

**Year 4 residents**
- Husain, Sameea
- Koch, James
- Kumar, Vikas (chief)
- Nakarawat, Weeraworn
- Thaquisuttikul, Iyavut (chief)

**Year 3 residents**
- Alam, Tariq
- Choi, Youngbin
- Choudhary, Saher
- Pan, Fei
- Sriram, Ashok

**Year 2 residents**
- Damodaram, Srikantha
- Hake, Austin
- Kandukurthi, Kalyan
- Lu, Xinrong
- Varma, Siddartha

**Year 1 residents**
- Jun, Bokkwan
- Kassar, Darine
- Maarouf, Jamal
- Rickard, Mary
- Weber, Daniel
- Wilbanks, Leslie

### Child Neurology residents

- Nayak, Anu
- Aldahondo, Otto
- Chrusciel, Deepti
Grading Policy

The final grade will be determined by a composite of the clinical evaluation (60%) and the NBME subject examination in neurology (40%). The clinical evaluation is made up of ward attending(s) (40%) and resident (15%) and clinic (5%) evaluations. Honors will be given to the top 25% of the class and Near Honors to 25%. In accord with the academic policies of the school of medicine, “final scores lower than 2.5 standard deviations below the mean will earn the grade of Fail. In rare circumstances the administrative symbols Deferred or Incomplete may be used.” In exceptional circumstances, a cumulative score higher than 2.00 standard deviations below the mean may result in a grade of Fail. Such circumstances would require a discussion between the clerkship director and phase director. Final scores between 2.00 and 2.5 standard deviations below the mean may earn the grades of Pass or Fail. A subject exam score at the 5th percentile or higher is required to pass the exam. Failure to achieve this score will result in a deferred grade.

Students may appeal grades that are earned for individual clerkships. Appeals are made to the Associate Dean for Curriculum, who will investigate the matter and issue a final decision.
Reading assignments for didactic sessions: you are expected to read the following cases prior to each didactic session in order to prepare for the discussions.

Toxic, Metabolic, Nutritional disorders and Movement disorders
Major cases: 7, 8, 9, 23
Minor cases: 18, 23, 27, 52, 54, 58, 59, 60, 61, 65, 66, 67, 68, 74, 78

Neuromuscular disorders
Major cases: 31, 32, 34, 41, 43, 44, 45, 46, 48, 55
Minor cases: 14, 22, 24, 25, 26, 28, 31, 32, 33, 40, 41

Cerebrovascular disorders
Major cases: 5, 11, 12, 22, 27, 28, 29, 30, 47, 51, 52
Minor cases: 6, 7, 8, 9, 10, 11, 12, 13, 16, 20, 21, 30, 35, 36, 37, 38, 42, 44, 48, 49, 69, 77

Epilepsy
Major cases: 1, 2, 3, 4, 5, 6, 10
Minor cases: 9, 15, 17, 18, 19, 34, 45, 58, 71

CNS infections and MS
Major cases: 4, 16, 20, 24, 54, 56
Minor cases: 1, 2, 3, 4, 5, 10, 22, 39, 40, 46, 47, 50, 62, 79

Headaches and cranial nerve disorders
Major cases: 11, 12, 13, 14, 15, 16, 17, 20, 53, 57
Minor cases: 2, 3, 4, 6, 8, 9, 10, 12, 16, 21, 38, 39, 51, 54, 55, 56, 63, 69, 70, 81

Dementia and Coma
Major cases: 18, 19, 20, 23, 26, 28
Minor cases: 5, 47, 52, 53, 64, 73, 74, 80
**Principles of Localization**

1. A 7-year-old boy has an episode of generalized tonic-clonic seizure early this morning while asleep. He also has episodes of twitching of his right face, numbness of the right cheek, and inability to speak.

2. A 27-year-old woman has seizures which begin with a rising feeling in her abdomen, followed by loss of awareness, deviation of the head and eyes to the left side followed by a generalized convulsive seizure lasting 2 minutes.

3. A 52-year-old man with pyelonephritis develops sudden paraplegia. He cannot feel a pin below the lower ribcage but can sense vibration in his toes.

4. A 62-year-old woman has an episode of visual loss affecting her right eye. She also has had headaches and jaw pain while chewing.

5. A 53-year-old man develops sudden low back pain while lifting his grandson. The pain radiates down the posterolateral aspect of his right leg into the foot.

6. A 73-year-old man has been speaking nonsense since this morning. His wife says that he doesn’t seem to understand her and he’s not making sense when he talks. Many of the words he says are not real words.

7. A 62-year-old hypertensive woman is brought to the hospital after collapsing suddenly. She cannot move her limbs. Pupils are pinpoint and react to light. She is very lethargic.

8. A 23-year-old man was involved in a motor vehicle accident this morning. He was unresponsive when pulled from the automobile. In the emergency room, his left pupil is noted to be 3 mm larger than the right and poorly reactive to light.

9. A 13-year-old girl notes severe left-sided facial weakness one morning when she awakens. She cannot close her left eyelid. She reports no change in her hearing.

10. A 26-year-old man has noticed right-sided facial weakness for the past couple of weeks. He also notes that sounds are muffled in his right ear.

11. A 42-year-old woman comes for further evaluation of chronic fatigue syndrome. She feels physical tired in the late afternoon and evening. She has to rest often. When especially tired, she develops diplopia. Examination is normal.
12. A 6-month-old boy has had weakness of the right arm since birth. He holds the limb internally rotated and adducted at the shoulder, pronated and extended at the elbow. The biceps reflex is absent.

13. A 16-year-old boy has epilepsy treated with phenytoin. For the past 2 days, he has felt unsteady when he walks. He is unable to perform a tandem walk.

14. A 16-year-old boy with hemophilia is brought to the hospital after crashing his bicycle. He is having trouble using his left hand, which is tremulous. He misses the target when reaching for an object with the left hand.

15. A 57-year-old man has developed slowness. He walks with short steps, has trouble with his balance when turning, and has tremor in his hands.

16. A 7-year-old girl has had difficulty using her right hand for the past 2 weeks. She seems fidgety and moody. She can’t control involuntary movements of her right arm and hand.

17. A 35-year-old woman with multiple sclerosis notes diplopia upon awakening. She reports the images are side-by-side and maximally separated when she looks to her right. She has deficient medial movement of the left eye while the right eye fully abducts with some nystagmus.

18. A 53-year-old man comes for evaluation of weakness which began 6 months ago in his right leg. Recently he noticed his left hand getting weak. On examination, he has depressed tendon reflexes and extensor plantar responses. His muscles ripple under the surface of the skin.

19. A 23-year-old woman has noticed progressive weakness in her legs for the past 3 days, today spreading to her hands. She has no deep tendon reflexes in her lower limbs, and they are depressed in the upper limbs. Sensation is intact. Plantar responses are downgoing. She is unable to dorsiflex either foot.

20. A 56-year-old man with history of hypertension and diabetes develops acute onset of vertigo, nausea and vomiting. Exam shows left pupil 2 mm smaller than right, left ptosis, left facial numbness, loss of sensation to pain and temperature on the right body and limbs, right leg ataxia, hiccups, and difficulty swallowing.
Headache

Case 1:
A 36 year old woman presents with a history of headache since she was a teenager. She says she would like “something to take when I have a bad headache, but I don’t want to take drugs all the time.” So, how will you begin...

Case 2:
A 43 year old male has come to see you because of recurrent headaches. For the past month he has had severe headache every day. They hurt a lot. So, how will you begin...

Case 3:
A 38 year old woman has pain in her face. It seems centered near a tooth in her maxilla recently filled with a composite filling material. Talking, eating and brushing her teeth sets the pain off. She has had to stop teaching because it hurts to talk and she has lost 10 lbs. Her dentist and an oral surgeon can find nothing wrong with the tooth but are reluctantly willing to take it out. So, how will you begin...

Case 4:
A 58 year old woman of Hungarian descent has a 20 year history of headache. They were described as a bitemporal, squeezing sensation and occurred almost daily – frequently present first thing in the morning. However, the same headache seems to have gotten worse the past 6 months or so. It hurts to chew a steak….by the way. So, how will you begin...

Case 5:
A 28 year old woman has had migraines since her teens. She was pumping iron when she suddenly developed a severe migraine, mainly right sided with vomiting and restlessness and her husband got concerned and brought her to see you. So, how will you begin...

Case 6:
A 39 year old man began to have occasional headaches at age 10. They gradually increased in frequency until by age 19 they were happening daily and that’s been pretty much it since then. He is a college graduate, employed as a grocery store manager, economically and socially well off and neither smokes nor drinks very often. He has been spending between $1,200 to $3,600 per year on various physical therapists, massage and chiropractic treatments as well as his medicine (6-8 tablets of acetaminophen + codeine a day) without any clear benefit. So, how will you begin...
**Coma**

**Case 1**
A 65-year-old grandmother is brought to the emergency room by her husband, who found her unresponsive when he awoke this morning. Her past medical history is significant only for glaucoma for which she takes eye drops and some “pills.”

**Case 2**
A 63-year-old married suburbanite seeks a medical evaluation because of “poor memory” and “difficulty sleeping.” He retired from his position as a bank vice-president six weeks ago. His position was eliminated after the unexpected acquisition of his bank by an international banking conglomerate. He disregards his personal hygiene and spends his time “watching soap operas and game shows.” The patient occasionally bursts into tears without provocation and has difficulty shopping for household items.

The patient’s general physical and neurological examinations are normal except for a blood pressure of 160/100 and slightly brisk DTRs in the left lower extremity. The plantar responses are equivocal. A screening mental status exam is normal except for an increased latency to respond to questions and commands and a depressed affect.
CNS Infections/Multiple sclerosis

Case 1

A 40-year-old woman presented with numbness and tingling of her right hand and leg. This was present on waking three weeks prior and was worse in the hot than in the cold weather.

Past history was positive for a loss of vision at age sixteen. She was born in New Orleans and moved to Boston at age 15. One sibling had difficulty walking. There was no other relevant history.

Examination revealed a slightly euphoric mood. Cranial nerve examination revealed nystagmus of the right eye on gaze to the right with incomplete abduction of the left eye.

Muscle power was normal. Tone was normal. Coordination was remarkable for ataxia on heel-to shin testing. Deep reflexes were symmetrical but brisk. Ankle clonus was noted bilaterally. Abdominal reflexes were asent, plantar responses flexor. Sensation was abnormal with decreased pin prick on the left arm and leg. There was decreased proprioception on the right let. There was no sensory level.

Case 2

A 20-year-old man presented with headache, confusion and stiff neck; his temperature was 100 F. While being examined he had a partial motor seizure lasting 1-2 minutes.

Neurological exam was unremarkable except for some weakness on the side of the seizure. Initial testing should include what?
Toxic/Metabolic/Nutritional disorders, Movement disorders

Case 1
A 54-year-old female was brought in by her family due to being “slow”. She had been dropping things and had difficulty walking. Her past medical history was significant for hypertension.

On examination she had flat affect. Her mental status examination was normal. She exhibited cogwheel rigidity and rest tremor. The sensory and cerebellar examinations were normal.

Case 2
A 36-year-old male had been consuming alcohol 3-4/week for about 5 years. He was brought to the emergency room by the police officer who found him “jerking all over” on the sidewalk.

On examination he was lethargic and oriented to name and “hospital” only. His motor and cerebellar examinations were normal. His sensory examination was unreliable due to inconsistent responses. His reflexes were symmetric except for absent ankle jerks.
Case 3

A 34-year-old female presents to you for “tingling” in the feet. About six months ago she underwent gastroplasty for weight reduction. The post-operative period was uneventful. Her husband also complained that she had made errors while balancing the check book.

Her examination was significant for fair to poor recent memory. Her concentration was poor. She had increased threshold to vibratory sensation in the lower extremities. Ankle and knee jerks were absent.

Case 4

A 26-year-old female was admitted for altered mental status. She has been an I/V drug use for the last 6 months. Her past medical history was negative.

On examination she had jaundice and intermittent extension and flexion of the wrists. She was oriented to name only. The rest of the neurological examination was normal.
Neuroanatomy outline

Often the clinical neuroscience student is dazzled by recall of the details of past neuroanatomic study and overwhelmed with the prospect of recalling this material at the beginning of this course. A time-consuming review of neuroanatomy is not necessary. Clearly, a grasp of gross anatomic structures and their relationships to one another as well as the major sensory and motor pathways are most relevant to clinical neurology. As the course begins, the following outline of major structures is presented to facilitate a cursory review. These structures are illustrated in many texts. Attention to the anatomy of imaging procedures is also helpful. In addition, the student is advised to review those anatomic structures that need reinforcement as they are encountered during the rotation.

**GROSS ANATOMIC STRUCTURES AND RELATIONSHIPS**

**LEPTOMENINGES:** epidural space and veins, dura matter, subdural space, arachnoid, subarachnoid space and vessels, pia mater.

**CEREBROSPINAL FLUID PATHWAYS:** choroid plexus, lateral ventricles, foramen of Moro, third ventricle, aqueduct, fourth ventricle, subarachnoid space, basal cisterns and subarachnoid villi

**SPINAL CORD:** segments C1-8, T1-12, L1-5, S1-5; dorsal and ventral gray matter, dorsal, lateral and anterior columns

**BRAIN STEM:** medulla, pons, midbrain, diencephalon

  *medulla*: CN XII, XI, X, IX, pyramidal decussation, inferior olivary nucleus, inferior cerebellar peduncle, descending tract of V

  *pons*: CN VIII, VII, VI, V, middle cerebellar peduncle, IV ventricle
midbrain: CN IV, III, superior cerebellar peduncle & decussation, aqueduct, periaqueductal grey, red nucleus, substantia nigra, cerebral peduncle, inferior & superior colliculi

diencephalon: optic chiasm and tracts, hypophyseal stalk, mamillary bodies, third ventricle, thalamus, pineal body, medial and lateral geniculate bodies

CEREBELLUM: inferior, middle and superior peduncles, granule & Purkinje cells, dentate nucleus and paramedian nuclei

BASAL GANGLIA: caudate nucleus, putamen, globius pallidus

CEREBRAL HEMISPHERES: frontal, temporal, parietal and occipital lobes; corona radiata and internal capsule; corpus callosum; frontal, temporal, and occipital horns and body of the lateral ventricles; hippocampal formation and fornix

MAJOR MOTOR AND SENSORY PATHWAYS

Upper motor neuron pathway: motor cortex-corona radiata-corticobulbar & corticospinal tracts in internal capsule, cerebral peduncle, pons, pyramid and lateral column

Basal ganglia: substantia nigra-caudate-putamen-globius pallidus-ventral lateral nucleus of the thalamus

Cerebellar outflow pathway: dentate nucleus-superior cerebellar peduncle-(decussation)-ventral lateral nucleus-precentral gyrus

Visual pathway: receptor cells-bipolar cells-ganglion cells-retinal nerve fibers-optic nerve-chiasm-(crossed & uncrossed fibers)-optic tract-lateral geniculate body-optic radiations-calcarine cortex

Auditory pathway: hair cells in cochlea-sprial ganglion-auditory nerve-ventral and dorsal cochlear nuclei-(crossed & uncrossed fibers)-superior olivary nucleus-nucleus
of the lateral lemniscus-inferior collicus-brachium of the inferior colliculus-medial geniculate body-temporal cortex (Heschl’s gyrus)

Vestibular pathway: hair cells in ampullae of semicircular canals-vestibular ganglion-vestibular nerve-vestibular nuclei-ocular pathways & floccular-nodular lobes

Pain & temperature pathway: skin receptors-spinal nerve-dorsal root ganglion-dorsal root-neurons of dorsal horn-decussation-lateral spinothalamic tract-ventral posteriolateral nucleus of the thalamus-postcentral gyrus

Proprioceptive pathway: joint, Golgi tendon & Pacinian receptors-spinal nerve-dorsal root ganglion-dorsal root-fasciculus gracilis & cuneatus-(decussation)-medial lemniscus-ventral posteriolateral nucleus-postcentral gyrus
Neuroepidemiology

The natural history of neurologic disease, particularly chronic disorders, is an area of increasing importance to the overall delivery of health care. The frequency, course and severity of neurologic disorders have been addressed by population-based rates (Kurtz, Ann Neuro 16:265-277, 1984). Annual incidence rates, prevalence and average duration in years per 100,000 for 61 neurological disorders were measured. The annual incidence for disease and injury to the nervous system was 2.5% of the population (2,500 per 100,000). For 55 of these conditions the prevalence was 9.5% or 9500 per 100,000 population. If headache, trauma, neurologic complications from alcoholism and vertebrogenic pain were excluded, 1.1% of the population would develop a neurologic condition each year, and 3.6% of the general population would still require neurologic attention at any one time.

To familiarize the student with the occurrence of neurologic diseases in the United States, the neuroepidemiology of various common or important neurological disorders from the above-mentioned report is found in the following table (* rates/100,000 population).

<table>
<thead>
<tr>
<th>Neurological Disorder</th>
<th>Ann. Incidence*</th>
<th>Prevalence*</th>
<th>Yrs. Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seizure disorder</td>
<td>50</td>
<td>650</td>
<td>13</td>
</tr>
<tr>
<td>Febrile seizures</td>
<td>50</td>
<td>100</td>
<td>2</td>
</tr>
<tr>
<td>Single seizures</td>
<td>20</td>
<td>60</td>
<td>3</td>
</tr>
<tr>
<td>Parkinsonism</td>
<td>20</td>
<td>200</td>
<td>10</td>
</tr>
<tr>
<td>Huntington’s disease</td>
<td>.4</td>
<td>5</td>
<td>14</td>
</tr>
<tr>
<td>Wilson’s disease</td>
<td>.1</td>
<td>1</td>
<td>12</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>3</td>
<td>60</td>
<td>35</td>
</tr>
<tr>
<td>Motor neuron disease</td>
<td>2</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>.4</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>Meningitis</td>
<td>15</td>
<td>5</td>
<td>.3</td>
</tr>
<tr>
<td>Encephalitis</td>
<td>15</td>
<td>10</td>
<td>.7</td>
</tr>
<tr>
<td>Brain abscess</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Migraine</td>
<td>250</td>
<td>2000</td>
<td>40</td>
</tr>
<tr>
<td>Sleep disorders</td>
<td>15</td>
<td>300</td>
<td>20</td>
</tr>
<tr>
<td>Acute cerebrovascular disease</td>
<td>150</td>
<td>600</td>
<td>4</td>
</tr>
<tr>
<td>Transient ischemic attacks</td>
<td>30</td>
<td>150</td>
<td>5</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>15</td>
<td>50</td>
<td>3</td>
</tr>
<tr>
<td>Dementias</td>
<td>50</td>
<td>250</td>
<td>5</td>
</tr>
<tr>
<td>Polyneuropathies</td>
<td>40</td>
<td>20</td>
<td>.5</td>
</tr>
</tbody>
</table>
Guillain-Barre syndrome       2     1     .5
Cervical pain syndrome       20    60    3
Lumbrosacral pain syndrome   150   500   3
Malignant brain tumor        5     5     1
Metastatic brain tumor       15    15    1
Benign brain tumor           10    60    6
Brain injury                200   800   4

The Neurological Examination

The neurological examination is an essential part of every routine physical examination. Its goals are to: 1) establish the functional integrity of the central and peripheral nervous system or 2) define a diffuse disorder or localize focal disturbances. Pertinent features of the history and examination are then used to characterize any pathologic dysfunction. The following are examples. Acute or subacute confusion and disorientation result from a diffuse alteration, usually bilateral, in the cerebral cortex or its subcortical projections. Common causes of this form of altered mental status are metabolic disturbances (impaired oxygenation, low blood sugar, kidney or liver failure), toxins, drugs or bilaterally impaired cerebral perfusion from hypotension. More chronic changes in mentation suggest a slowly expanding tumor or degenerative disorder. Increasing left-sided weakness and depression of consciousness following a blow to the head are indicative of a right hemispheric disorder by a subdural hematoma. Focal back pain and leg weakness in a patient with carcinoma of the lung occurs from spinal cord compression by an epidural metastasis. For sure, localization under the bony protection about the central nervous system is more indirect and dependent upon the constellation of findings than is examination of other organ systems. Thus, the skill involved and reliance upon the neurological examination is important to acquire.

After the locus and nature of the neurological disorder are determined, decisions are more intelligently made concerning further diagnostic testing to confirm the initial impression. Established neurological findings also serve as a baseline for the progress of subsequent therapeutic maneuvers. Lastly, the facility, rapidity and cost effectiveness of a thorough neurological examination should not be overlooked in this age of reliance upon technology.
Methodology

The History

The neurological history alone is the basis of more than three-quarters of clinical diagnoses in neurology. Symptoms of nervous system dysfunction are reviewed in the same systematic, orderly fashion that the examination is performed. Beginning with the head and ending with the toes, inquiry is directed to the function of the cerebral cortex, cranial nerves, cerebellum, motor and sensory systems. The duration, mode of onset (slow, rapid), functional significance, aggravations or relief of the symptom are sought. An insidious onset and progressive worsening of a symptom complex suggests compression of a focal area of the nervous system by a tumor or other mass. Particular attention is given to the character and localization of pain. The distribution of neurological symptoms is also important.

The Examination

An organized, habitual approach to the neurological examination assures thoroughness and allows constant dispatch with ever-increasing skill. Although especially careful attention should be given to symptomatic areas of the nervous system implicated by the history, the order of beginning with the head and proceeding toward the extremities should be maintained. For general practice it is efficient to incorporate the neurological examination into the mechanics of the general physical examination which begins with the vital signs. Critical areas and connecting pathways of the nervous system as well as major vessels and internal organs are also examined. The location of pain should be carefully scrutinized as to location, character, date of onset, duration, precipitants and modes of relief. To be clear and complete, and avoid potentially important omissions, it is important to separately record six major subdivisions of the neurological examination, i.e., mental status, cranial nerves, cerebellum, motor, sensory and reflexes. When there are symptomatic complaints relevant to a specific subdivision, the examination should be more detailed within that subdivision. When appropriate examination of the head and vertebrae and great vessels of the neck should be detailed.
I. Mental Status

This examination begins with the initial patient contact. An altered mental status is often apparent by inspection of the patient’s appearance or after introductory conversation about the patient’s identity (e.g., occupation, residence, family) or presenting complaints. A change in cognitive function is foretold by the presenting complaints or the failure to maintain previous levels of achievement (e.g., job or school performance).

Formal assessment of the mental status begins with a judgment about the level of consciousness. Is the patient alert? If not, is the patient obtunded (continuously responsive to the environment after being aroused by some form of stimulation), stuporous (requiring continuous stimulation to maintain responsiveness to the environment), or comatose (unresponsive to the environment despite vigorous stimulation). If alert, is the use of language correct? This may be done by the ease of conversation in the history. But if there is any question about the use of language or, especially in patients with suspected left hemispheric injury, a formal examination of speech should be performed. This includes remarks about the seven major parameters of speech: fluency (rate of speech), articulation, repetition, naming (especially parts of objects), comprehension (e.g. ability to follow one, two or three stage commands), reading and writing. (Language studies suggest that fluency and articulation are programmed in Broca’s area in the opercular region of the frontal lobe. Comprehension of auditory sounds is deciphered in the posterior one-third of the superior temporal gyrus (Wernicke’s area). Repetition is effected by the connection between Wernicke’s and Broca’s region via the arcuate fasciculus. Reading and, to some extent writing, is determined by the angular gyrus which is located at the termination of the superior temporal gyrus. If language is appropriate, are thoughts organized and coherent? Is the patient confused, delusional or hallucinating? Is attention span limited (determined by spelling five-letter words backwards, e.g. “world” or recalling seven recited digits forward or five backwards? Is the patient oriented to time and place? If there is any suggestion of an abnormal mental status by the history, cognitive function should be further examined with respect to memory, especially immediate versus remote memory (recall of three objects five minutes later, or four hidden objects in four places ten
minutes later versus recall of past Presidents, significance of December 7, 1941). Ability to calculate is determined by requested double digit additions or subtractions or serial backward subtraction of 7’s from 100. Abstract thinking is estimated by an analysis of likenesses, e.g. how is a dog like a cat, fork like a spoon, tree like a bush or apple like a pear? Or how are proverbs interpreted. General fund of knowledge relevant to current political, climatic, commercial or sporting events should also be obtained. Inquiry about previous education and occupational history is advised to compare the expected degree of accomplishment with the current state. Notations about the patient’s emotional condition or affect (i.e. euphoric, anxious, sad, hostile) are also useful.

Attention to an abnormal mental status is essential to all phases of patient management. This awareness immediately includes the degree of cooperation and attentiveness to the sensory and motor portions of the neurological examination.

II. Cranial Nerves

Details regarding the history and examination of vision, ocular motility, mastication, facial sensation and movement, hearing, swallowing and speech are found in the section of the syllabus devoted to the “Examination of the Cranial Nerves.”

III. Cerebellum

Abnormalities in the control of motor functions constitute the major signs of cerebellar function. Attention is given to the pattern of speech, stability of the trunk and fluidity of limb movements. Generally, tests are made of the station (Romberg), gait (tandem walk), upper limbs (rapid alternating movements of the hands [dysdiadochokinesia] and finger-to-nose maneuver [dysmetria]), and lower extremities (heel-to-shin maneuver [dysmetria]). In more severe disease of the cerebellum, hypotonia and pendular reflexes are also present.

IV. Motor System

Complaints relevant to the motor system are those of weakness, uncoordination, abnormal tone (“stiffness”), postures or spontaneous movements. The examination is principally concerned with bulk, tone, strength and coordination of movements. Examination of the motor system is often carried out with the general examination of the extremities. Sustained opening of the jaw (pteryoid power), symmetry of facial
movements and neck flexor and extensor strength (often impaired in disorders affecting the lower motor neuron or muscle) are not to be omitted.

In the course of the evaluation, the patient is often asked to move to a table or chair. The spontaneity, fluidity, balance and ease of the gait should be carefully observed. The power of the flexors (anterior tibialis) and extensors (gastrocnemius and soleus) of the feet are quickly determined by the ability to walk on the heels and toes.

Bulk is evaluated by both inspection and palpation. Muscle tenderness is noted if present.

The examiner determines muscle tone by passively moving the resting extremity. Flaccidity is often encountered with complete paralysis, and hypotonicity is found with acute paresis of any cause, cerebellar disorders and muscle diseases. Spastic hypertonicity is increased tone that suddenly diminishes at a point one-half to two-thirds through the range of passive movement. The feel to the examiner is similar to that of opening a clasp knife. “Clasp knife” hypertonicity is commonly encountered with pyramidal system disorders and often elicited with passive supination or extension of the forearm. Cogwheel rigidity is a form of hypertonicity in which passive range of motion reveals a steady small-amplitude increase and release of muscle tone. This occurs with dopamine deficiency within the basal ganglia from Parkinson’s disease. Paratonic rigidity is a sustained, plastic increase in tone that is not accompanied by any release. It is found with degenerative disorders, especially those involving the frontal lobe. An early form of this rigidity is noted by “counter movements” or Gegenhalten. In this form of abnormal tone, an increase in tone is encountered with the speed and force of the examiner’s movement. Slower and less forceful movements are met by a lesser degree of rigidity.

To evaluate motor power properly, the function of major muscle groups of each limb are determined. This involves mostly the action of flexion and extension about the various joints. However, pronators or supinators of the forearm, invertors or evertors of the foot, or abductors and adductors of the arm, thumb or leg are included. Positioning of the limb and the degree of force used to determine weakness varies among physicians. Placing the limb in the position of greatest mechanical advantage to the muscle group
being assessed is advised. Force is then exerted from a similarly strong muscle group of
the examiner. Exertion is steadily applied in order to avoid unexpected strain or jerking
that causes the patient to release the muscle. The distribution of muscle weakness is
carefully evaluated for a pattern consistent with a segmental, peripheral nerve or
proximal versus distal distribution.

Coordination of the motor system is mostly evaluated with tests of cerebellar function.
Notations regarding slowness or non-intentional movements are most appropriate, i.e.,
tremors, myoclonic jerks (sudden, single contractions), choreiform (quick,
semipurposeful) movements, dystonic postures, tics (repetitive, stereotyped movements),
or myotonia (continued, involuntary muscle contraction).

V. Sensory System

The sensory examination requires an attentive and discriminating patient. Sensory
deficits are often accompanied by complaints of anesthesia (complete sensory loss),
hypalgesia (partial sensory loss), paresthesias (tingling, numbness) or dysesthesias
(painful, aberrant sensations). One peculiar sensory complaint that indicates a
myelopathic process is a sudden, brief or shock-like numbness radiating from the neck,
down the spine and out the extremities. This especially occurs if the neck is flexed and is
termed L’hermitte’s sign. Often a cervical tumor, demyelinating disorder or post-
irradiation process is present. If the patient is drowsy, incoherent, demented or aphasic,
he or she may be unable to understand what the examiner wishes to examine. Only
withdrawal of a limb to deep or superficial pain may be determinable, so the examiner
must proceed to another part of the examination. The sensory examination may be
performed during the general examination of the extremities, but should include
examination of the trunk (T1-T8), abdomen (T8-L1), buttocks (S2-S5) and posterior head
(C2-C3), especially if there is any indication of a high spinal cord lesion.

The chief sensory modalities are superficial pain (usually tested with a pin),
temperature (as to a distinctly cool instrument), light touch (to a finger or wisp of cotton),
position sense (at distal joints) and vibration (tuning fork is applied to a bony surface).
Usually the patient is asked to compare sensations on one side of the body to the other.
In spinal cord disorders, sensation is compared in dermatomes above and below the
suspected level of cord dysfunction. The latter establishes a “sensory level” for spinal cord pathology. It is sometimes helpful to have the patient compare in monetary terms areas of normal to diminished sensation, e.g., “a dollar’s worth” versus “25, 50 or 75 cents”. Focal deficits in any limb should be carefully studied for a pattern that coincides with a specific dermatome versus a peripheral nerve deficit. Distal sensory loss is common in polyneuropathies and usually involves the lower extremities because they have the longest peripheral nerves. Conduction in these longer nerves is necessarily sustained for a longer time, making these nerves more sensitive to toxic, metabolic or degenerative disturbances within the nerve. With peripheral neuropathies, there is often a graded, ill-defined border compared to the sharp borders of a stocking or glove-like distribution of sensory loss found in hysterical patients.

VI. Reflexes

Superficial reflexes are those segmental motor responses that follow stimulation of a specific dermatome. The epigastric (T6-T9), mid-abdominal (T9-T11) and hypogastric responses (T11-L1) are the most useful and are elicited by a light stimulus placed on the skin and drawn toward the stimulus. The anal reflex assess S3-S5 by determining external anal sphincter tone following a noxious stimulus (pin or pinch) to the skin adjacent to the anus or perineum. Other reflexes dependent upon sacral segments are the less commonly used cremasteric and bulbocavernous reflexes; details regarding these tests are found in the texts referenced in this handbook.

Tendon reflexes are the biceps (C5-C6, musculocutaneous nerve), brachioradialis (C5-C6, radial nerve), triceps (C7-C8, radial nerve), knee (L2-L4, femoral nerve) and ankle (L5-S2, posterior tibial nerve). These reflexes are activated by sudden lengthening of the muscle which activates receptors in the muscle spindle. The action potential is carried on Ia fibers through the dorsal root ganglion and via the afferent dorsal root into the dorsal horn. The fiber terminates directly on an alpha motor neuron in the anterior horn. Depolarization results in contraction of the muscle fibers in this neuron’s motor unit. The discharge of a large number of anterior horn cells produces the normal deep tendon reflex. Reflexes on the right are compared to those on the left, and a judgment is rendered regarding the reaction elicited, i.e., absent, hyporeflexic, normal or hyperreflexic (i.e. 0, 1+, 2+, 3+). An absent reflex is considered only after reinforcement
techniques (squeezing a fist, clenching the teeth) have been attempted. An absent reflex implies disruption of the reflex arc. Hyperactive reflexes occur with loss of higher or supranuclear inhibition of the reflex arc. Clonus, a repetitive contraction and release of agonist and antagonist muscles, is often but not invariably a pathologic form of hyperreflexia (4+). It is elicited by a sudden passive stretch of a muscle.

Pathological reflexes are important, particularly the extensor plantar response or Babinski sign which is characterized by extension of the great toe with flexion and fanning of the remaining toes. A stimulus, usually a noxious one, is applied to the lateral plantar surface at the heel, drawn forward to the ball of the foot and moved across the sole toward the great stimulation of the foot (Chaddock), deep pressure applied on the shin below the knee and run down to the ankle (Oppenheim) and abduction of the little toe (Stransky). The pathological response to a noxious stimulus of extension rather than flexion of the great toe results from the release of the anterior motor horn cells at L5 from the usual inhibition of the corticospinal tract. Thus, a pathologic disturbance in the corticospinal pathway is implied. Trace the pathway eliciting the Babinski sign, i.e., from the evoked sensory impulse on the plantar surface through the nerves to the spinal cord and back via the nerve activating the contraction of the extensor hallucis longus.

**Formulation**

Once the examination is completed, the examiner needs to review the findings. Is the examination complete? Does it include all the major subsystems of the nervous system: mental status, cranial nerves, motor, sensory, cerebellar and reflex responses? Was sufficient attention given to the focus in the nervous system suggested by the symptoms?

Further introspection includes answers to the following. Are the findings focal, multifocal or diffuse? Do the motor and reflex findings indicate a supranuclear or infranuclear disturbance? Is the locus of pathology above or below the foramen magnum? Are motor and sensory findings lateralized to the same side? Do the cranial nerve or cerebellar signs indicate a posterior fossa process? Have complaints of pain about the head or vertebral column been thoroughly examined? Is there bony tenderness over the focus of pain? Is the neck stiff? If there is low back pain, was a straight leg
raising test performed (stretching irritable or compressed nerves by elevating the lower limb at the hip with the knee extended until pain is reproduced)?

**Recording**

The examination should be dated, and the time listed.

All data should be expressed in clearly written sentences. Abbreviations and notations should be avoided.
Examination of the Cranial Nerves

INTRODUCTION

Because of their location along the ventral surface of the brain and base of the skull, examination of the cranial nerves is critical in determining the extent of many neurological disorders or in identifying dysfunction of a single cranial nerve. Recognition of the cranial nerve lesion also provides valuable clinical evidence for a pathological process above the foramen magnum. Furthermore, it enables the clinician to concentrate attention to the anatomic pathway of the cranial nerve and contiguous structures. The usual clinical approach to cranial nerve deficits is to divide or “compartmentalize” the cranial nerve into its natural anatomic divisions or compartments, i.e. parenchymatous, subarachnoid, intradural, interosseous, and extracranial. Thus, in the clinical setting the history, examination, and appropriate clinical tests also focus on neighborhood signs or symptoms that result from disease of structures within these compartments.

In the following pages, we summarize the function, historical abnormality, and examination of each separate cranial nerve. The anatomical origin and termination of the cranial nerves are reviewed with emphasis placed on the cranial nerve per se (bracketed in notes), as it extends along the base of the skull and its interosseous course. Common disorders involving each nerve are cited to illustrate the relevance of cranial nerve examination. In all instances the right and left cranial nerves are tested separately.

CN I: Olfactory nerve

1.) Function: smell
2.) History: anosmia
3.) Examination: non-stringent odor applied to nostril, i.e. vanilla, clove, soap
4.) Pathway: [nasal mucosa, cribriform plate of ethmoid bone] to olfactory bulb
5.) Disorders: nasal disease, traumatic avulsion, subfrontal tumors, midline developmental anomalies (e.g. holoprosencephaly)

CNII: Optic nerve

1.) Function: vision
2.) History: dimness and blurriness of vision, often in central portions of the visual field, occasionally in its periphery

3.) Examination:
   a. Visual acuity; best corrected near and/or far vision
   b. Color plate discrimination
   c. Swinging flashlight test for Marcus Gunn pupil (indicates positive optic nerve deficit when the optical media are clear and the retina is grossly normal. The positive sign is the paradoxical dilation of the pupil as a bright penlight is swung from the normal eye to the one affected by optic nerve disease).
   d. Ophthalmoscopy

   It is generally recommended for neurologists to dilate the pupils of the patients they examine. This can be accomplished preferentially with 1% tropicamide. To obtain a satisfactory and thoughtful inspection of the fundus it is equally important that both the patient and examiner are comfortable. If the patient’s eye is positioned lower than the examiner’s, it is often helpful to tilt the patient’s head and ocular gaze upwards, and vice versa. To stabilize the retina during the examination, the patient is asked to fixate on a distant object and then hold that position. The eye is approached about 30° into the temporal field. As the funduscope is drawn near the cornea, and if the media are clear (if not, the red reflex will not be present), the optic disc head can be sighted in its usual position, 15° into the nasal retina from the posterior pole in the horizontal raphe. Then, the vessels and the macula are examined.

   1.) Optic disc: round or oval, flat, optic cup in center, orangish hue.

   Contrary to what is commonly said, the normal optic nerve not completely “sharp”. Its borders show the irregularity cost by the nerve fiber bundles. Completely sharp discs are usually atrophic. Conditions that cause and attrition of ganglion cell nerve fibers degenerate, the capillary bed is lost in the nerve head turns pale as light reflects off of the remaining nerve fibers in the lamina cribosa. The paleness is sometimes a matter of degree and needs to be distinguished from normal temporal pallor on occasions.
Swelling of the optic nerve head results from any condition which obstructs axoplasmic flow in ganglion cell nerve fibers. Hence optic disc edema may occur ocularly and accompanied by impaired visual function from inflammatory or ischemic diseases within the eye. More important to the standard examination is the presence of bilateral papilledema from increased intracranial pressure. The swelling in the optic disk apparently results from an increase in subarachnoid fluid pressure as it envelops the optic nerve from the cranial cavity to the globe of the eye. This increased pressure tamponades the flow of axoplasm, which then accumulates behind the lamina cribosa at the optic disc. Since impaired axoplasmic flow does not disrupt electrical conduction along the nerve membrane, visual function remains normal until long-standing choke of the nerve interferes with blood supply to the disc head. The characteristic appearance of papilledema includes: opacity of the peripapillary nerve fiber with blurring of the disc margins; hyperemia of the disc; engorgement of retina veins and loss of spontaneous venous pulsations; elevation of the disc; flame shaped hemorrhages in the nerve fiber layer; exudates.

2.) Vessels: inferior and superior central retinal artery and vein (3:4 ratio); venous pulsations usually seen in vein near cup.

3.) Macula: temporal to disc, fovea in center is red; inspect nearby retina for hemorrhages and exudates.

e. Visual fields

This is the area of vision seen by one eye from 90° temporally, 60° nasally, 60° superiorly, and 80° inferiorly. Its center is the point of foveal fixation in the region of keenest visual resolution (20/20 by the Snellen method). About 15° temporally and horizontally is the blind spot, a 5° by 8° region of absolute blindness that corresponds to the optic disc in the nasal retina. As one moves from the fovea peripherally, the
receptive field (number of receptors to each ganglion cell) increases in size and visual resolution drops so that it is around 20/70 at the blind spot and 20/200 in the far periphery. Test targets of defined size, shape and color are customarily used to test visual resolution in the visual field rather than numbers or letters of various sizes. Any depression in the normal field to well-defined test targets is regarded as a scotoma.

The technique of confrontation is used by bedside examiners to test visual fields and, if correctly performed, is capable of detecting most of the visual field defects occurring from disease within the neural portion of the visual system. The patient is asked to cover one eye and gaze into the opposite pupil of the examiner. As he maintains fixation, he is asked to acknowledge perception of an image which is brought into the edge of the field, i.e. hand, finger, head of a pin. By placing such objects in equivalent portions of the temporal and nasal field, the patient is asked to judge the similarity in brightness and clarity of the two presented objects. Thus partial or relative scotomas are detected.

Formal visual field testing with precisely defined targets and screens in quiet rooms with standard illumination and projection screens is needed for complete quantification of the visual field defect, to follow the progress of treatment of diseases or to detect small scotomas. The tangent screen is a flat surface that is 2 m square and assesses the central portions of the visual field, 25° at 2 m and 50° if the patient is at 1 m. Since the major portion of the optic nerve transmits signals from the fovea and macula, the tangent screen is quite adequate in evaluating most visual field defects of neurological origin. For full assessment of the field, a curved surface must be used. Full field, perimetry (Goldman fields) is required for many retinal disorders.

The recognition of the location of a visual field defect is dependent upon the pattern or configuration of the scotoma. A monocular defect confines the disorder to the pre-chiasmal portion of the visual system. Defects which fall along the vertical midline indicate a deficit within or posterior to the chiasm. Defects which are similar and lateralized to the leftward or rightward portions of the field are termed homonymous. If precisely the same, the defects are referred to as congruous, but if their edges do
not correspond, they are referred to as incongruous. As a rule incongruous homonymous defects occur in disorders of the geniculocalcarine radiations as the fibers pass through the temporal or parietal lobes, whereas congruence homonymous defects occur where the crossed and uncrossed chiasmal projections are more nearly aligned, the occipital lobe.

Recall that the optical axis lies within the lens of the eye, so that images in the upper field are projected on the lower retina, nasal field to the temporal retina, etc. Fibers in the lower and upper retina remain respectively in their inferior and superior positions as they traverse the visual system. The nasal retinal fibers, however, decussate in the chiasm and join the opposite temporal fibers in the optic tract.

4.) Pathway: retinal ganglion cells; [optic disc, intraorbital, canalicular (optic canal), intracranial to chiasm]; tract to lateral geniculate body.

5.) Disorders: retinal infarction (central retinal artery occlusion); glaucoma; ischemic optic neuritis; optic neuritis (inflammation with demyelination); optic nerve compression (aneurysm, tumor); papilledema due to increased intracranial pressure.

Arcuate scotoma: This is a defect in the nerve fibers running from the temporal retina to the optic disc. Central acuity is preserved. This type of defect occurs in glaucoma which is associated with an enlarged optic cup and elevated intraocular tension.
Enlarged blind spots: This occurs in patients with raised intracranial pressure and bilateral papilledema. The swelling of the optic disc displaces adjacent retina. Headaches and other signs and symptoms of intracranial mass may be present.

Altitudinal scotoma (lower): This occurs with infarction of the upper retina or optic disc. It may be associated with visible cholesterol emboli in the eye or an audible bruit in the ipsilateral carotid that is a common source of such emboli. This defect may also occur with ischemic infarction of the optic disc.
Central scotoma: This occurs with disease of the macula or optic nerve. If in the retrobulbar portion of the nerve, the fundus appears normal. Obviously, central acuity is impaired.

Bilateral centrocecal scotomas: Cecal refers to involvement of the blind spot. Central visual acuity is impaired. This is the type of defect occurring in alcoholic amblyopia. The lesion is retrobulbar so the fundus appears normal initially, but optic atrophy eventually develops with retrograde degeneration of the optic nerve fibers.
Junctional scotomas: Refers to involvement of the optic nerve (central scotoma OS) proximally on the left at its junction with the chiasm (bitemporal defect). This is a common finding in patients with pituitary tumors in which symptoms of pituitary insufficiency or excess hormone secretion occur. Bitemporal defects of chiasmal disease also occur with hypothalamic tumors.

Upper quadrantanopia: This is the typical defect of temporal lobe disease, often a glioma. There may be an associated psychomotor seizure disorder. Incongruity is most apparent in temporal lobe defects due to the wide splaying of Meyer’s loop in the temporal radiations. This type of visual field deficit is often referred to as the “pie in the sky”.
**Lower quadrantanopia**: This defect occurs with parietal lobe disease. It may be associated with visual and/or tactile special defects or other parietal lobe signs. Incongruity is less pronounced in parietal lesions since the upper radiations are apparently more compact than the lower temporal ones.

**Homonymous socotomas**: This is a congruous defect indicative of an occipital (upper, left) lobe lesion. The small discrete deficit implies infarction in the territory of a small branch of the calcarine artery. The infarct may result from emboli from an atheromatous vertebrobasilar artery tree.
**CN III: Oculomotor nerve**

1.) Function: ocular movement; elevation of the lid; regulation of light passing through the retina; accommodation

2.) History: horizontal or oblique diplopia; ptosis; blurry vision at near

3.) Examination: move the eye horizontally, up and in, up and out, down and out; measure the interpalpebral fissure; observe the pupillary reaction to light and a near stimulus; measure near acuity.

4.) Pathway: oculomotor nucleus, fast sickles in midbrain,[subarachnoid space to dura in cavernous sinus to superior orbital fissure] upper and lower divisions in orbit. Edinger-Westphal nucleus to CN III trunk to ciliary nerves to globe to iris (sphincter pupillae muscle) and angle (ciliary muscle).

5.) Disorders: aneurysms, trauma, herniation (uncal compression) the seas, tumors (pituitary, meningioma in dura, chordoma, metastatic) or infarction as with diabetes (pupil is spared).

**CN IV: Trochlear nerve.**

1.) Function: intorsion, movement of the eye down and in during adduction.

2.) History: vertical diplopia

3.) Examination: head tilt test: diplopia lessons when the head is tilted away from the involved eye so as to place the ear on the shoulder; ipsilateral tilting makes the displacement of images greater. Compare the position of the eyes with gaze down and in: a lesser degree of depression indicates the weak eye.

4.) Pathway: cochlear nucleus to exit the dorsal inferior surface of the midbrain to [edge of tentorium to the cavernous sinus, superior orbital fissure, orbit]

5.) Disorders: trauma to head or orbital rim (trochlear process); infarction; tumor

**CN V: Trigeminal nerve**

1.) Function: a.) Facial, nasal and oral sensation, b.) Mastication

2.) History: numbness or anesthesia; jaw deviation; weakened chewing

3.) Examination: a.) Touch, pain, coolness of V1. V2, V3; corneal reflex; wisp of cotton placed at limbus and moved over cornea, b.) Palpation of temporalis and
masseter muscles with forceful draw closure; jaw deviation with jaw opening (deviates ipsilateral to weak pterygoid); weakness of jaw opening and displacement to side of weak pterygoid.

4.) Pathway: dermatomes of V1 (superior orbital fissure), V2 (foramen rotundum), V3 (foramen ovale); cavernous sinus to trigeminal ganglion; [trigeminal root] two main sensory nucleus (touch) and spinal trigeminal nucleus (pain and temperature). Motor nucleus in pons, [motor root] to V3 to masticatory muscles.

5.) Disorders: cerebello-pontine tumor: extension of nasopharyngeal tumor to V1, V2, V3; herpes zoster (V1).

**CN VI: Abducent nerve**

1.) Function: abduction of the globe of the eye
2.) History: horizontal diplopia, especially with distant gaze
3.) Examination: horizontal eye movement
4.) Pathway: abduct nucleus, fascicles in pons, [subarachnoid space to posterior cavernous sinus to superior orbital fissure to orbit]
5.) Disorders: post-viral neuropathy; infarction (diabetic); tumor (primary or metastatic, nasopharyngeal tumor especially); aneurysm; trauma; increased intracranial pressure.

**CN VII: Facial nerve**

1.) Function: facial movements; lacrimation; salivation (maxillary glands); taste; external ear sensation; hyperacusis
2.) History: loss of eye closure and smile, slurred speech, food sticks about lower gums, dry and burning eye
3.) Examination: test for asymmetric wrinkling of forehead, elevation of the eyebrow, retraction of the corner of the mouth when smiling or grimacing; test for ability to forcefully closed eyelids, purse lips, and contract platysma. Be certain to understand the clinical significance between a supranuclear facial paresis (lower face weakness most prominent) and a nuclear lesion (upper and lower face equally affected).
4.) Pathway: [CN VII nucleus, genu, root in CP angle, internal auditory canal and facial canal in petrus bone, stylomastoid foramen, parotid gland] to facial muscles; taste buds to lingual nerve (V3), to chorda tympani to geniculate ganglion to [nervous intermedius] to nucleus tractus solitarius in medulla; superior salivatory nucleus [nervous intermedius] to greater petrosal nerve to pterygopalatine ganglion to lacrimal gland.

5.) Disorders: Bell’s palsy; trauma; cerebellopontine angle tumors; sarcoidosis

**CN VIII: Acoustic nerve**

1.) Function: hearing and balance

2.) History: tinnitus, deafness, vertiginous episodes

3.) Examination: a.) watch test; Rinne test (normal if AC>BC, abnormal with external or middle ear disease); Weber test (vibrating fork on vertex, normally heard equally in both ears, hear ipsilaterally with external or middle ear disease, contralaterally with cochlear ear disease) b.) positional nystagmus via head rotation and postural tilting; peripheral vertigo (rotatory nystagmus is brief, prolonged nystagmus suggests lesion in cerebellovestibular connections.

4.) Pathway: hair cells in organ of Corti, [rootlets to spiral ganglion, root in IAC] to cochlear nuclei in pons; hair cells of semicircular canals, utricle, saccule, [rootlets to vestibular ganglion, CN VII in IAC] to vestibular nuclei in pons

5.) Disorders: acoustic neuromas; idiopathic neuritis

**CN IX: Glossopharyngeal nerve**

1.) Function: taste (posterior 1/3 of tongue); salivation; stylopharyngeal muscle

2.) History: none specifically

3.) Examination: gag response after touching posterior pharyngeal wall (not reliable as CN IX test for response is shared by CN X)

4.) Pathway: taste and motor fibers pass in [CN IX via jugular foramen] inferior salivatory nucleus to CN IX to optic ganglion to parotid gland]
CN X: Vagus nerve

1.) Function: swallowing; phonation; pulmonary, cardiac and visceral innervation
2.) History: nasal voice and hoarseness; if bilateral, dysphagia and regurgitation
3.) Examination: a.) palate deviates away from paralysed side; vocal cord does not oppose, b.) loss of ipsilateral carotid sinus reflex
4.) Pathway: nucleus ambiguous to [CN X via jugular foramen] to palatal, pharyngeal and laryngeal muscles
5.) Disorders: jugular foramen tumors; ALS

CN XI: Accessory nerve

1.) Function: head rotation and elevation of the shoulder
2.) History: difficulty in elevation of the shoulder and turning the head
3.) Examination: palpate sternocleidomastoid muscle; test for elevation and retraction of shoulders and rotating head.
4.) Pathway: ventral horn C 1-5 [rootlets enter foramen magnum pass through jugular foramen] to SCM and trapezius muscles
5.) Disorders: jugular foramen tumors

CN XII: Hypoglossal nerve

1.) Function: articulation and swallowing
2.) History: dysarthria
3.) Examination: look for atrophy or fasciculations ipsilaterally; upon protrusion tongue deviates ipsilateral to nerve lesion due to unopposed action of contralateral genioglossus muscle
4.) Pathway: hypoglossal nucleus, [root passes through hypoglossal canal] to tongue muscle
5.) Disorders: metastatic tumors to hypoglossal nerve
SAINT LOUIS UNIVERSITY
SCHOOL OF MEDICINE
DEPARTMENT OF NEUROLOGY

NEUROLOGICAL CHECK SHEET

PATIENT:........................SEX:......HOSPITAL:......AGE:......DATE:......

SYMBOLS: Normal: *Abnormal; O Absent; +Exaggerated; ++Greatly exaggerated; NE Not examined

A. Mental Status and Higher Cerebral Functions

Handedness: R......L......Occupation:......Highest education level:.............
Where:..................Appearance, behavior, cooperation:......................Describe level
of consciousness:........Orientation: Time:......Place:......Person:......Memory:
Past:......Recent:......Retention and recall: Digits: Forward:..............Backward:
3 objects:......General information:......Calculations:......Proverbs and similarities:
Judgement and insight:......Mood and activity:......Hallucinations
and delusions:......Language function: Articulation, phonation:......Volume,
dysarthric features: slurring, other disorders of rhythm, pitch, and tonal inflection:......
Fluency and correctness of spontaneous language:......................
Syllabic articulation:......Echolalia:......Naming on visual presentation:......
Comprehension of spoken language:......Repetition: Syllables:......Words:......
Phrases:......Reading:......Writing (include sample):......Revisualization:......
Capacity (use of language and comprehension of the intent of what is said to
him):......Competence: Sentence construction:......Linguistic transformation:......
Apraxia: Constructional:......Dressing:......Ideational:......Ideomotor:......
Linguistic:......Echopraxia:......Agnosia: Modality:......Special features, e.g. for
faces:......Neglect:......Denial:......

B. Cranial Nerves

I. Smell: R......L......Substance used:............................

II. Visual acuity and reading fields, fundi, and perception (include horizontal
bisection, Bender VII, clock face):..........

III. Pupil size: R......M......M......Reaction to light: R......L......Accommodation:

IV. R......L......Foveal fissures: R......L......Exophthalmos:......Eye move-
ments: Gaze: R......L......Up......Down......Individual muscles:......Conver-
gence:......Red-glass test if diplopia present:......Nystagmus and direction:......
Ppesis: R......L......Optokinetic nystagmus:......

V. Strength and bulk of masticatory muscles: R......L......Jaw jerk:..........
Sensation on face: R......L......Corneal reflex: R......L......

VII. Facial movements: Volitional: R......L......Taste:......Stimulus used:......

VIII. Ophthalmoscopic examination: R......L......Recognition for whispered words or quiet
sounds: R......L......Schwabach, Weber, Rinne tests: R......L......Fork frequency:
R......L......Caloric testing: R......L......Positional vertigo:.............

IX. Palatal movement:......Swallowing:......Phonation:......Gag reflex:......
X. Laryngoscopic examination:.................

XI. Strength and bulk of sternomastoid, trapezius muscles: R......L......Head
posture:..........

XII. Inspection of tongue for atrophy, fasciculations: R......L......Protrusion
of tongue:......Rapid movements: R......L......Tremor:.............

C. Motor (Make table where applicable; specify for upper, lower, limbs, trunk)

Skilled acts:......Posture:......Gait:......Power:......Bulk:......Tone:......
Involuntary movements:......Associated movements:......Fasciculations:......
Convulsions:......Myoclonus:......Straight leg raising:......Ability to roll over,
rise from bed, chair:......
PATIENT: .................................. DATE: .................................. NEUROLOGICAL CHECK SHEET

D. Sensation (Use sensory chart)

Subjective complaints: ....... Coordination: ....... Pinprick: ....... Touch: ............
Fingertips: ....... Two-point discrimination: ....... Tactile localization: ....... Number
writing: ....... Appreciation of texture, size: ....... Shape: ....... Double simultaneous
Muscle tenderness: ....... Achilles pain: .......

E. Coordination: ....... Station-Romberg-tandem gait: ....... Finger-nose: Eyes open: .......  
Closed: ....... Alternating movements: ....... Heel-shin: ....... Toe to object: .......

F. Reflexes

<table>
<thead>
<tr>
<th>R&gt; = &lt;L</th>
<th>Brachio-</th>
<th>R&gt; = &lt;L</th>
<th>Biceps</th>
<th>R&gt; = &lt;L</th>
<th>Cremasteric</th>
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<tbody>
<tr>
<td>Jaw</td>
<td>Brachial</td>
<td>Grasp</td>
<td>Radialis</td>
<td>Biceps</td>
<td>or bulbo-</td>
</tr>
<tr>
<td>Grasp</td>
<td>Radialis</td>
<td>Avoid</td>
<td>Finger</td>
<td>Abdominal</td>
<td>cavernous</td>
</tr>
<tr>
<td>Suck</td>
<td>Flexor</td>
<td>Suck</td>
<td></td>
<td></td>
<td>Semi-</td>
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<td>Biceps</td>
<td>Knee</td>
<td>Biceps</td>
<td></td>
<td></td>
<td>Membranous</td>
</tr>
<tr>
<td>Triceps</td>
<td>Ankle</td>
<td></td>
<td></td>
<td></td>
<td>Plantar</td>
</tr>
</tbody>
</table>

Note any other reflex elicited: ........................................

G. Sphincters: Bladder; Retina: ....... Voiding: ....... Incontinence: ....... Bowel:  
Restraining: ....... Evacuation: ....... Incontinence: ....... Sweating, piloerection: .......  
Skin and Limb temperature: ....... Sexual function (erection, ejaculation): .......  
Vasodepressor and cardioacceleratory responses: ....... Pulse rate: ....... Blood
pressure: Lying: ....... Sitting 5 minutes: ....... Standing 2 minutes: ....... Valsalva
pulse: Resting: ....... Pulse at end of maneuver: .......

H. Head, neck, and spine

Signs of meningeal irritation: Rigidity: ....... Brudzinski: ....... Kernig: .......  
Cranium: OF circumference: ....... Shape: ....... Signs of trauma: ....... Tenderness: .......  
Spine: Configuration: ....... Tenderness: ....... Movements: ....... Extracranial Cerebral

I. Endocrine: Body type: ....... Sexual development: ....... Thyroid: .......

Instructions:

1. This form to be used on all patients.
2. Attach patient's handwriting sample, drawing of clock face with numerals, copy of Bender figure VII *, and bisection of horizontal line.
3. This is not a substitute for a written out, thoroughly documented description of the result of your examination, but is a guide. The abnormal findings are to be described fully on a blank sheet of history paper to follow this form in the record. The object of the check sheet is to note quickly normal findings. At the end of the description of the neurological and general medical findings, the examiner is to propose an anatomical localization and pathophysiological inference, and define the PROBLEM which the patient presents, the probable diagnosis, and the plan for solving the patient's problem. If problems are multiple they should be arranged in order of their significance.

* Bender figure VII

EXAMINER: .................................. DATE: ..................................
INSTRUCTIONS FOR ADMINISTRATION OF MINI-MENTAL STATE EXAMINATION

ORIENTATION

(1) Ask for the date. Then ask specifically for parts omitted, e.g., "Can you also tell me what reason it is?" One point for each correct.
(2) Ask in turn "Can you tell me the name of this hospital?" (town, county, etc.). One point for each correct.

REGISTRATION

Ask the patient if you may test his memory. Then say the names of 3 unrelated objects, clearly and slowly, about one second for each. After you have said all 3, ask him to repeat them. This first repetition determines his score (0-3), but keep testing these until he can repeat all 3, up to 6 trials. If he does not eventually learn all 3, recall cannot be meaningfully tested.

ATTENTION AND CALCULATION

Ask the patient to begin with 100 and count backwards by 7. Stop after subtractions (93, 86, 79, 72, 65). Score the total number of correct answers.

If the patient cannot or will not perform this task, ask him to spell the word "word" backwards. The score is the number of letters in correct order. E.g., does - 5, does - 3.

RECALL

Ask the patient if he can recall the 3 words you previously asked him to remember. Score 0 - 3.

LANGUAGE

Naming: Show the patient a wrist watch and ask him what it is. Repeat for pencil. Score 0 - 2.

Repetition: Ask the patient to repeat the sentence after you. Allow only one trial. Score 0 or 1.

2-stage command: Give the patient a piece of plain blank paper and repeat the command. Score 1 point for each part correctly executed.

Reading: On a blank piece of paper print the sentence "Close your eyes", in letters large enough for the patient to see clearly. Ask him to read it and do what it says. Score 1 point only if he actually closes his eyes.

Writing: Give the patient a blank piece of paper and ask him to write a sentence for you. Do not dictate a sentence; it is to be written spontaneously. It must contain a subject and verb and be sensible. Correct grammar and punctuation are not necessary.

Copying: On a clean piece of paper, draw intersecting pentagons, each side about 1 inch, and ask him to copy it exactly as it is. All 10 angles must be present and 2 must intersect to score 1 pint. Tracing and rotation are ignored.

Estimate the patient's level of sensorium along a continuum, from alert on the left to coma on the right.

The density of shading and separation should indicate the severity of the sensory loss.

MARK:
- Position Sense (at joints tested)
- Vibration Sense (at prominences tested)

Examiner:

Normal
Reduced
Absent
Bloodborne Pathogen Exposure
Saint Louis University School of Medicine

Medical students come into contact with patients and with blood or other body fluids from patients. An exposure is defined as a percutaneous injury, mucous membrane contact, or non-intact skin contact with bodily fluids. Percutaneous injuries may include a needle stick or a cut with a sharp object. Risks vary and may be substantial and health care services are based on the nature of the hazards, the intensity and frequency of exposure, and overall risk.

If you are exposed to a bloodborne pathogen, you should take the following steps.

If the incident occurs at
  o SLU Hospital during business hours, report immediately to SLU Employee Health.
  
  o SLU Hospital outside of regular business hours, report immediately to the SLU emergency room and then follow up with SLU Employee Health on the next business day.
  
  o An affiliated hospital, report immediately to that hospital’s emergency room, report it to Employee Health at the affiliated hospital, and then follow up with SLU Employee Health on the next business day.
  
  o A community preceptor site, report it immediately to the preceptor and report immediately to SLU Employee Health.

Exposure requires filing a Report of Injury with SLU Employee Health and with the Office of Student Affairs in the School of Medicine.

Employee Health
Saint Louis University
3655 Vista Avenue – West Pavilion Suite 116
Saint Louis, MO  63110
Phone:  314.268.5499
Fax:    314.268.5537

Office hours: 7:30 am to 4:00 pm
Walk in services are offered 7:30am to 11am OR 1pm to 3:30pm excluding holidays.