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KV Krishna Das

Clinical Medicine

A Textbook of Clinical Methods and Laboratory Investigations

As per the Competency Based Medical Education Curriculum (NMC)



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Competency Table

Number	*COMPETENCY* *The student should be able to:*	*Core Y/N*	*Suggested learning methods*	*Suggested assessment methods*	*Chapter number*	*Page number*
IM1.1	Describe and discuss the epidemiology, pathogenesis clinical evolution and course of common causes of heart disease including—rheumatic/valvular, ischemic, hypertrophic inflammatory	Y	Lecture, small group discussion	Written/viva voce	14	202–203
IM1.4	Stage heart failure	Y	Lecture, small group discussion	Written/viva voce	14	203
IM1.11	Perform and demonstrate a systematic examination based on the history that will help establish the diagnosis and estimate its severity including—measurement of pulse, blood pressure and respiratory rate, jugular venous forms and pulses, peripheral pulses, conjunctiva and fundus, lung, cardiac examination including palpation and auscultation with identification of heart sounds and murmurs, abdominal distension and splenic palpation	Y	Bedside clinic, DOAP session	Skill assessment	15 12 14 2	216–242 148–161 203 23–61
IM1.12	Demonstrate peripheral pulse, volume, character, quality and variation in various causes of heart failure	Y	Bedside clinic, DOAP session	Skill assessment		
IM1.13	Measure the blood pressure accurately, recognize and discuss alterations in blood pressure in valvular heart disease and other causes of heart failure and cardiac tamponade	Y	Bedside clinic, DOAP session	Skill assessment	15 2	242 23–61
IM1.17	Order and interpret diagnostic testing based on the clinical diagnosis including 12 lead ECG, chest radiograph, blood cultures	Y	Bedside clinic, DOAP session	Skill assessment	16	245
IM1.18	Perform and interpret a 12 lead ECG	Y	Bedside clinic, DOAP session	Skill assessment	16	245
IM1.19	Enumerate the indications for and describe the findings of heart failure with the following conditions including—2D echocardiography, brain natriuretic peptide, exercise testing, nuclear medicine testing and coronary angiogram	N	Lecture, small group discussion, bedside clinic	Skill assessment	16	263
IM1.20	Determine the severity of valvular heart disease based on the clinical and laboratory and imaging features and determine the level of intervention required including surgery	Y	Small group discussion, lecture, bedside clinic	Written/skill assessment	16	245–272

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM1.26	Develop document and present a management plan for patients with heart failure based on type of failure, underlying etiology	Y	Bedside clinic, skill assessment, small group discussion	Bedside clinic/skill assessment/ written	17	273–276
IM2.1	Discuss and describe the epidemiology, antecedents and risk factors for atherosclerosis and ischemic heart disease	Y	Lecture, small group discussion	Written/viva voce	14	202–203
IM2.6	Elicit document and present an appropriate history that includes onset evolution, presentation risk factors, family history, comorbid conditions, complications, medication, history of atherosclerosis, IHD and coronary syndromes	Y	Bedside clinic, DOAP session	Skill assessment	15	206–216
IM2.7	Perform, demonstrate and document a physical examination including a vascular and cardiac examination that is appropriate for the clinical presentation	Y	Bedside clinic, DOAP session	Skill assessment	15 2	216–225 23–61
IM2.8	Generate document and present a differential diagnosis based on the clinical presentation and prioritize based on "cannot miss", most likely diagnosis and severity	Y	Bedside clinic, DOAP session	Skill assessment	2	23–61
IM2.10	Order, perform and interpret an ECG	Y	Bedside clinic, DOAP session	Skill assessment	16	245
IM2.11	Order and interpret a chest X-ray and markers of acute myocardial infarction	Y	Bedside clinic, DOAP session	Skill assessment	16	245–272
IM2.13	Discuss and enumerate the indications for and findings on echocardiogram, stress testing and coronary angiogram	Y	Lecture, small group discussion	Written/viva voce	16	245–272
IM2.21	Observe and participate in a controlled environment an ACLS program	N	DOAP session	NA	17	274
IM2.22	Perform and demonstrate in a mannequin BLS	Y	DOAP session	Skill assessment	17	273
IM3.5	Perform, document and demonstrate a physical examination including general examination and appropriate examination of the lungs that establishes the diagnosis, complications and severity of disease	Y	Bedside clinic, DOAP session	Skill assessment	12	148–161
IM3.7	Order and interpret diagnostic tests based on the clinical presentation including—CBC, chest X-ray PA view, Mantoux, sputum gram stain, sputum culture and sensitivity, pleural fluid examination and culture, HIV testing and ABG	Y	Bedside clinic, DOAP session	Skill assessment	13	178–191
IM3.8	Demonstrate in a mannequin and interpret results of an arterial blood gas examination	Y	Bedside clinic, DOAP session	Skill assessment	13 2	178–191 23–61
IM3.9	Demonstrate in a mannequin and interpret results of a pleural fluid aspiration	Y	DOAP session	Skill assessment	13	178–191

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM3.11	Describe and enumerate the indications for further testing including HRCT, viral cultures, PCR and specialized testing	Y	Bedside clinic, DOAP session	Skill assessment	13	178–191
IM3.14	Perform and interpret a sputum gram stain and AFB	Y	DOAP session	Skill assessment	13	178–191
IM4.10	Perform a systematic examination that establishes the diagnosis and severity of presentation that includes—general skin mucosal and lymph node examination, chest and abdominal examination (including examination of the liver and spleen)	Y	Bedside clinic, DOAP session	Skill assessment	2	23–61
IM4.11	Generate a differential diagnosis and prioritize based on clinical features that help distinguish between infective, inflammatory, malignant and rheumatologic causes	Y	Bedside clinic, DOAP session	Written/viva voce	2	23–61
IM4.12	Order and interpret diagnostic tests based on the differential diagnosis including—CBC with differential, peripheral smear, urinary analysis with sediment, chest X-ray, blood and urine cultures, sputum gram stain and cultures, sputum AFB and cultures, CSF analysis, pleural and body fluid analysis, stool routine and culture and QBC	Y	Bedside clinic, skill assessment	Skill assessment	2	23–61
IM5.3	Describe and discuss the pathologic changes in various forms of liver disease	Y	Lecture, small group discussion	Written/viva voce	8	127–130
IM5.4	Describe and discuss the epidemiology, microbiology, immunology and clinical evolution of infective (viral) hepatitis	Y	Lecture, small group discussion	Written/viva voce	8	127–130
IM5.5	Describe and discuss the pathophysiology and clinical evolution of alcoholic liver disease	Y	Lecture, small group discussion	Written/viva voce	8	127–130
IM5.6	Describe and discuss the pathophysiology, clinical evolution and complications of cirrhosis and portal hypertension including ascites, spontaneous bacterial peritonitis, hepatorenal syndrome and hepatic encephalopathy	Y	Lecture, small group discussion	Written/viva voce	8	127–130
IM5.7	Enumerate and describe the causes and pathophysiology of drug induced liver injury	Y	Lecture, small group discussion	Written/viva voce	8	127–130
IM5.8	Describe and discuss the pathophysiology, clinical evolution and complications cholelithiasis and cholecystitis	Y	Lecture, small group discussion	Written/viva voce	8	127–130
IM5.9	Elicit document and present a medical history that helps delineate the etiology of the current presentation and includes clinical presentation, risk factors, drug use, sexual history, vaccination history and family history	Y	Bedside clinic, DOAP session	Skill assessment	8 5, 6	127–130 80–103

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM5.10	Perform a systematic examination that establishes the diagnosis and severity that includes nutritional status, mental status, jaundice, abdominal distension ascites, features of portosystemic hypertension and hepatic encephalopathy	Y	Bedside clinic, DOAP session	Skill assessment	8 5, 6 6	127–130 80–103
IM5.11	Generate a differential diagnosis and prioritize based on clinical features that suggest a specific etiology for the presenting symptom	Y	Bedside clinic, DOAP session	Skill assessment/ viva voce	8	127–130
IM5.12	Choose and interpret appropriate diagnostic tests including—CBC, bilirubin, function tests, hepatitis serology and ascitic fluid examination in patient with liver diseases	Y	Bedside clinic, DOAP session	Skill assessment	9 7	132–135 107–123
IM5.13	Enumerate the indications for ultrasound and other imaging studies including MRCP and ERCP and describe the findings in liver disease	Y	Bedside clinic, small group discussion	Viva voce/ written	9 10	132–135 136
IM5.14	Outline a diagnostic approach to liver disease based on hyperbilirubinemia, liver function changes and hepatitis serology	Y	Bedside clinic, small group discussion	Viva voce/ written	9	132–135
IM5.15	Assist in the performance and interpret the findings of an ascitic fluid analysis	Y	DOAP session	documen-tation in log book	9	132–135
IM6.11	Enumerate the indications and describe the findings for CT of the chest and brain and MRI	N	Small group discussion, lecture, bedside clinic	Written/viva voce	16	269
IM6.15	Demonstrate in a model the correct technique to perform a lumbar puncture	Y	Simulation	Skill assessment	34	585
IM7.1	Describe the pathophysiology of autoimmune disease	Y	Lecture, small group discussion	Written/viva voce	18	279–280
IM7.3	Classify cause of joint pain based on the pathophysiology	Y	Lecture, small group discussion	Written/viva voce	18	279–280
IM7.4	Develop a systematic clinical approach to joint pain based on the pathophysiology	Y	Lecture, small group discussion	Written/viva voce	19	282–306
IM7.5	Describe and discriminate acute, subacute and chronic causes of joint pain	Y	Lecture, small group discussion	Written/viva voce	19	282–306
IM7.6	Discriminate, describe and discuss arthralgia from arthritis and mechanical from inflammatory causes of joint pain	Y	Lecture, small group discussion	Written/viva voce	19	282–306
IM7.7	Discriminate, describe and discuss distinguishing articular from periarticular complaints	Y	Lecture, small group discussion	Written/viva voce	19	282–306

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM7.8	Determine the potential causes of join pain based on the presenting features of joint involvement	Y	Lecture, small group discussion	Written/viva voce	19	282–306
IM7.9	Describe the common signs and symptoms of articular and periarticular diseases	Y	Lecture, small group discussion	Written/viva voce	19	282–306
IM7.10	Describe the systemic manifestations of rheumatologic disease	Y	Lecture, small group discussion	Written/viva voce	19	282–306
IM7.11	Elicit document and present a medical history that will differentiate the etiologies of disease	Y	Bedside clinic, DOAP session	Skill assessment	19	282–306
IM7.12	Perform a systematic examination of all joints, muscle and skin that will establish the diagnosis and severity of disease	Y	Bedside clinic, DOAP session	Skill assessment	19	282–306
IM7.13	Generate a differential diagnosis and prioritize based on clinical features that suggest a specific etiology	Y	Bedside clinic, small group discussion	Skill assessment/ written	19	282–306
IM7.14	Describe the appropriate diagnostic work up based on the presumed etiology	Y	Bedside clinic, small group discussion	Skill assessment/ written	20	309–317
IM7.15	Enumerate the indications for and interpret the results of—CBC, anti-CCP, RA, ANA, DNA and other tests of autoimmunity	Y	Bedside clinic, small group discussion	Skill assessment/ written	20	309
IM7.17	Enumerate the indications and interpret plain radiographs of joints	Y	Bedside clinic, small group discussion	Skill assessment/ written	20	314
IM8.10	Perform a systematic examination that includes—an accurate measurement of blood pressure, fundus examination, examination of vasculature and heart	Y	Bedside clinic, DOAP session	Skill assessment	14 2	203 23–61
IM8.11	Generate a differential diagnosis and prioritize based on clinical features that suggest a specific etiology	Y	Bedside clinic, DOAP session	Skill assessment	2	23–61
IM8.13	Enumerate the indications for and interpret the results of—CBC, urine routine, BUN, Cr, electrolytes, uric acid, ECG	Y	Small group discussion	Skill assessment/ written/viva voce	16	245
IM8.17	Perform and interpret a 12 lead ECG	Y	DOAP session	Documentation in log book/skills station	16	245
IM9.1	Define, describe and classify anemia based on red blood cell size and reticulocyte count	Y	Lecture, small group discussion	Written/viva voce	23	335–340
IM9.2	Describe and discuss the morphological characteristics, etiology and prevalence of each of the causes of anemia	Y	Lecture, small group discussion	Written/viva voce	24	358

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM9.3	Elicit document and present a medical history that includes symptoms, risk factors including GI bleeding, prior history, medications, menstrual history, and family history	Y	Bed side clinic, DOAP session	Skill assessment	22 24 5, 6	325–332 358 80–103
IM9.4	Perform a systematic examination that includes—general examination for pallor, oral examination, DOAP session of hyper dynamic circulation, lymph node and splenic examination	Y	Bedside clinic, DOAP session	Skill assessment	22 2	325–332 23–61
IM9.5	Generate a differential diagnosis and prioritize based on clinical features that suggest a specific etiology	Y	Bedside clinic, DOAP session	Skill assessment/ written	22	325–332
IM9.6	Describe the appropriate diagnostic work up based on the presumed etiology	Y	Bedside clinic, DOAP session	Skill assessment/ written	23	333–357
IM9.7	Describe and discuss the meaning and utility of various components of the hemogram	Y	Lecture, small group discussion	Written/viva voce/skill assessment	23	333–357
IM9.8	Describe and discuss the various tests for iron deficiency	Y	Lecture, small group discussion	Written/viva voce/skill assessment	23	333–357
IM9.9	Order and interpret tests for anemia including hemogram, red cell indices, reticulocyte count, iron studies, B12 and folate	Y	Bedside clinic, DOAP session	Skill assessment/ written	23	333–357
IM9.10	Describe, perform and interpret a peripheral smear and stool occult blood	P	Bedside clinic, DOAP session	Skill assessment/ written	23	333–357
IM9.11	Describe the indications and interpret the results of a bone marrow aspirations and biopsy	Y	Lecture, small group discussion	Written/viva voce/skill assessment	23	333–357
IM9.12	Describe, develop a diagnostic plan to determine the etiology of anemia	Y	Lecture, small group discussion	Written/viva voce/skill assessment	24	358
IM10.1	Define, describe and differentiate between acute and chronic renal failure	Y	Lecture, small group discussion	Written/viva voce	28	399–403
IM10.2	Classify, describe and differentiate the pathophysiologic causes of acute renal failure	Y	Lecture, small group discussion	Written/viva voce	28	399–403
IM10.5	Describe and discuss the etiology of CRF	Y	Lecture, small group discussion	Written/viva voce	28	399–403
IM10.6	Stage chronic kidney disease	Y	Lecture, small group discussion	Written/viva voce	28	399–403

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM10.12	Elicit document and present a medical history that will differentiate the etiologies of disease, distinguish acute and chronic disease, identify predisposing conditions, nephrotoxic drugs and systemic causes	Y	Bedside clinic, DOAP session	Skill assessment	29	405–413
IM10.13	Perform a systematic examination that establishes the diagnosis and severity including determination of volume status, presence of edema and heart failure, features of uremia and associated systemic disease	Y	Bedside clinic, DOAP session	Skill assessment	14 29	203 405–413
IM10.14	Generate a differential diagnosis and prioritize based on clinical features that suggest a specific etiology	Y	DOAP session, small group discussion	Skill assessment/ written/viva voce	29	405–413
IM10.15	Describe the appropriate diagnostic work up based on the presumed etiology	Y	DOAP session, small group discussion	Skill assessment/ written/viva voce	30	414–430
IM10.16	Enumerate the indications for and interpret the results of—renal function tests, calcium, phosphorus, PTH, urine electrolytes, osmolality, Anion gap	Y	DOAP session, small group discussion	Skill assessment/ written/viva voce	30	414–430
IM10.17	Describe and calculate indices of renal function based on available laboratories including FENa (Fractional excretion of sodium) and CrCl (Creatinine clearance)	Y	DOAP session, small group discussion	Skill assessment/ written/viva voce	30	414–430
IM10.18	Identify the ECG findings in hyperkalemia	Y	DOAP session, small group discussion	Skill assessment/ written/viva voce	30	414–430
IM10.19	Enumerate the indications and describe the findings in renal ultrasound	N	Lecture, small group discussion	Written/viva voce	30	414–430
IM10.20	Describe and discuss the indications to perform arterial blood gas analysis—interpret the data	Y	DOAP session	Documentation in log-book	30	414–430
IM11.2	Describe and discuss the epidemiology and pathogenesis and risk factors and clinical evolution of type 1 diabetes	Y	Lecture, small group discussion	Written/viva voce	25	366
IM11.3	Describe and discuss the epidemiology and pathogenesis and risk factors economic impact and clinical evolution of type 2 diabetes	Y	Lecture, small group discussion	Written/viva voce	25	366
IM11.4	Describe and discuss the genetic background and the influence of the environment on diabetes	N	Lecture, small group discussion	Written/viva voce	25	367

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Number	*COMPETENCY The student should be able to:*	*Core Y/N*	*Suggested learning methods*	*Suggested assessment methods*	*Chapter number*	*Page number*
IM11.7	Elicit document and present a medical history that will differentiate the etiologies of diabetes including risk factors, precipitating factors, lifestyle, nutritional history, family history, medication history, co-morbidities and target organ disease	Y	Bedside clinic, DOAP session	Skill assessment	26	372–375
IM11.8	Perform a systematic examination that establishes the diagnosis and severity that includes skin, peripheral pulses, blood pressure measurement, fundus examination, detailed examination of the foot (pulses, nervous system findings and deformities and injuries)	Y	Bedside clinic, DOAP session	Skill assessment	26	372–375
IM11.11	Order and interpret laboratory tests to diagnose diabetes and its complications including—glucose, glucose tolerance test, glycosylated hemoglobin, urinary microalbumin, ECG, electrolytes, ABG, ketones, renal function tests and lipid profile	Y	Bedside clinic, DOAP session	Skill assessment	27	285
IM11.12	Perform and interpret a capillary blood glucose test	Y	Bedside clinic, DOAP session	Skill assessment	27	385
IM11.13	Perform and interpret a urinary ketone estimation with a dipstick	Y	Bedside clinic, DOAP session	Skill assessment	27	385
IM12.1	Describe the epidemiology and pathogenesis of hypothyroidism and hyperthyroidism including the influence of iodine deficiency and autoimmunity in the pathogenesis of thyroid disease	Y	Lecture, small group discussion	Written/viva voce	25	366
IM12.2	Describe and discuss the genetic basis of some forms of thyroid dysfunction	N	Lecture, small group discussion	Written/viva voce	25	367
IM12.3	Describe and discuss the physiology of the hypothalamopituitary-thyroid axis, principles of thyroid function testing and alterations in physiologic function	Y	Lecture, small group discussion	Short notes	26	369–375, 380
IM12.5	Elicit document and present an appropriate history that will establish the diagnosis cause of thyroid dysfunction and its severity	Y	Bedside clinic	Skill assessment/ short case	26	369–375
IM12.6	Perform and demonstrate a systematic examination based on the history that will help establish the diagnosis and severity including systemic signs of thyrotoxicosis and hypothyroidism, palpation of the pulse for rate and rhythm abnormalities, neck palpation of the thyroid and lymph nodes and cardiovascular findings	Y	Bed side clinic, DOAP session	Skill assessment	26	369–375, 380
IM12.7	Demonstrate the correct technique to palpate the thyroid	Y	Bedside clinic, DOAP session	Skill assessment	26	372–375

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM12.9	Order and interpret diagnostic testing based on the clinical diagnosis including CBC, thyroid function tests and ECG and radio iodine uptake and scan	Y	Bedside clinic, DOAP session	Skill assessment	27	383–384, 388
IM12.11	Interpret thyroid function tests in hypo- and hyperthyroidism	Y	Bedside clinic, lab	Skill assessment	27	388
IM13.9	Demonstrate in a mannequin the correct technique for performing breast exam, rectal examination and cervical examination and pap smear	Y	Bedside clinic	Skill assessment/ short case	26 2	380 23–61
IM14.7	Perform, document and demonstrate a physical examination based on the history that includes general examination, measurement of abdominal obesity, signs of secondary causes and comorbidities	Y	Bedside clinic, skills lab	Skill assessment	2	23–61
IM15.1	Enumerate, describe and discuss the etiology of upper and lower GI bleeding	Y	Lecture, small group discussion	Short note/ viva voce	5, 6	80–103
IM15.4	Elicit and document and present an appropriate history that identifies the route of bleeding, quantity, grade, volume loss, duration, etiology, comorbid illnesses and risk factors	Y	Bedside clinic	Skill assessment	24	358
IM15.5	Perform, demonstrate and document a physical examination based on the history that includes general examination, volume assessment and appropriate abdominal examination	Y	Bedside clinic, skills lab	Skill assessment	5, 6	80–103
IM15.7	Demonstrate the correct technique to perform an anal and rectal examination in a mannequin or equivalent	Y	DOAP session	Skill assessment	5, 6	80–103
IM15.9	Choose and interpret diagnostic tests based on the clinical diagnosis including complete blood count, PT and PTT, stool examination, occult blood, liver function tests, *H. pylori* test	Y	Bedside clinic, DOAP session, small group discussion	Skill assessment/ short note/ viva voce	7 23	107–123 344–354
IM15.10	Enumerate the indications for endoscopy, colonoscopy and other imaging procedures in the investigation of upper GI bleeding	Y	Lecture, small group discussion	Written/viva voce	7	107–123
IM16.4	Elicit and document and present an appropriate history that includes the natural history, dietary history, travel , sexual history and other concomitant illnesses	Y	Bedside clinic, skills lab	Skill assessment	5, 6	80–103
IM16.5	Perform, document and demonstrate a physical examination based on the history that includes general examination, including an appropriate abdominal examination	Y	Bedside clinic, skills lab	Skill assessment	5, 6	80–103

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM16.8	Choose and interpret diagnostic tests based on the clinical diagnosis including complete blood count, and stool examination	Y	Bedside clinic, skills lab, small group discussion	Skill assessment/ short note/ viva voce	7	107–123
IM16.9	Identify common parasitic causes of diarrhea under the microscope in a stool specimen	Y	DOAP session	Skill assessment	7	107–123
IM16.11	Enumerate the indications for stool cultures and blood cultures in patients with acute diarrhea	Y	Lecture, small group discussion	Written/viva voce	7	107–123
IM16.12	Enumerate and discuss the indications for further investigations including antibodies, colonoscopy, diagnostic imaging and biopsy in the diagnosis of chronic diarrhea	Y	Lecture, small group discussion	Written/viva voce	7	107–123
IM17.1	Define and classify headache and describe the presenting features, precipitating factors, aggravating and relieving factors of various kinds of headache	Y	Lecture, small group discussion	Short note/ viva voce	33	561
IM17.2	Elicit and document and present an appropriate history including aura, precipitating aggravating and relieving factors, associated symptoms that help identify the cause of headaches	Y	Bedside clinic, small group discussion	Bedside clinic/skill assessment	33	561
IM17.4	Perform and demonstrate a general neurologic examination and a focused examination for signs of intracranial tension including neck signs of meningitis	Y	Bedside clinic, small group discussion	Bedside clinic/skill assessment	32	452–555
IM17.5	Generate document and present a differential diagnosis based on the clinical features, and prioritize the diagnosis based on the presentation	Y	Bedside clinic, small group discussion	Bedside clinic/skill assessment	33	562–574
IM17.6	Choose and interpret diagnostic testing based on the clinical diagnosis including imaging	Y	Lecture, small group discussion, bedside clinic	Skill assessment	34	580–598
IM17.7	Enumerate the indications and describe the findings in the CSF in patients with meningitis	Y	Small group discussion, bedside clinic	Skill assessment	34	585
IM17.8	Demonstrate in a mannequin or equivalent the correct technique for performing a lumbar puncture	Y	DOAP session	Skill assessment	34	585
IM17.9	Interpret the CSF findings when presented with various parameters of CSF fluid analysis	Y	Small group discussion, bedside clinic	Skill assessment	34	585
IM18.1	Describe the functional and the vascular anatomy of the brain	Y	Lecture, small group discussion	Written/viva voce	31	436–442

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM18.3	Elicit and document and present an appropriate history including onset, progression, precipitating and aggravating relieving factors, associated symptoms that help identify the cause of the cerebrovascular accident	Y	Bedside clinic	Skill assessment	33	562–574
IM18.5	Perform, demonstrate and document physical examination that includes general and a detailed neurologic examination as appropriate, based on the history	Y	Bedside clinic, DOAP session	Skill assessment	32 31	452–555 436–445
IM18.6	Distinguish the lesion based on upper vs lower motor neuron, side, site and most probable nature of the lesion	Y	Bedside clinic, DOAP session	Skill assessment	33	562–574
IM18.8	Describe and distinguish, based on the clinical presentation, the types of bladder dysfunction seen in CNS disease	Y	Small group discussion, bedside clinic	Written/viva voce	32	548
IM18.9	Choose and interpret the appropriate diagnostic and imaging test that will delineate the anatomy and underlying cause of the lesion	Y	Bedside clinic, DOAP session, small group discussion	Written/viva voce/skill assessment	34	580–598
IM19.1	Describe the functional anatomy of the locomotor system of the brain and controlling parts of locomotor system	Y	Lecture, small group discussion	Written/viva voce	31	436–442
IM19.7	Choose and interpret diagnostic and imaging tests in the diagnosis of movement disorders	Y	Bedside clinic, small group session	Skill assessment/ written/viva voce	20	315–317
IM22.1	Enumerate the causes of hypercalcemia and distinguish the features of PTH vs non-PTH mediated hypercalcemia	N	Lecture, small group discussion	Written/viva voce	26	369–375
IM22.2	Describe the etiology, clinical manifestations, diagnosis and clinical approach to primary hyperparathyroidism	N	Lecture, small group discussion	Written/viva voce	26	369–375
IM22.4	Enumerate the components and describe the genetic basis of the multiple endocrine neoplasia syndrome	N	Lecture, small group discussion	Written/viva voce	25	368
IM23.4	Enumerate the indications for enteral and parenteral nutrition in critically-ill patients	Y	Lecture, small group discussion	Written/viva voce	2	23–61
IM24.1	Describe and discuss the epidemiology, pathogenesis, clinical evolution, presentation and course of common diseases in the elderly	Y	Lecture, small group discussion	Written/viva voce	11 18 36	141 280 632–637
IM24.2	Perform multidimensional geriatric assessment that includes medical, psycho-social and functional components	Y	Bedside clinic, DOAP session	Skill assessment	36	632–637

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Number	*COMPETENCY* *The student should be able to:*	*Core Y/N*	*Suggested learning methods*	*Suggested assessment methods*	*Chapter number*	*Page number*
IM24.3	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of acute confusional states	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.4	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of vascular events in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24 5	Describe and discuss the etiopathogenesis clinical presentation identification, functional changes, acute care, stabilization, management and rehabilitation of depression in the elderly	Y	Lecture Small group discussion	Written/viva voce	36	632–637
IM24.6	Describe and discuss the etiopathogenesis causes, clinical presentation, presentation identification, functional changes, acute care, stabilization, management and rehabilitation of dementia in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.7	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of personality changes in the elderly	N	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.8	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of osteoporosis in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.9	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of CVA in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.10	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of COPD in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.11	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of the elderly undergoing surgery	Y	Lecture, small group discussion	Written/viva voce	36	632–637

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM24.12	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of degenerative joint disease	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.13	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of falls in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.14	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of common fractures in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.15	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of vision and visual loss in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.16	Describe and discuss the principles of physical and social rehabilitation, functional assessment, role of physiotherapy and occupational therapy in the management of disability in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.17	Describe and discuss the etiopathogenesis, clinical presentation, identification, functional changes, acute care, stabilization, management and rehabilitation of hearing loss in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.18	Describe the impact of the demographic changes in aging on the population	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.19	Enumerate and describe the social problems in the elderly including isolation, abuse, change in family structure and their impact on health	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.20	Enumerate and describe social interventions in the care of elderly including domiciliary services, rehabilitation facilities, old age homes and state interventions	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.21	Enumerate and describe ethical issues in the care of the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637
IM24.22	Describe and discuss the etiopathogenesis, clinical presentation, complications, assessment and management of nutritional disorders in the elderly	Y	Lecture, small group discussion	Written/viva voce	36	632–637

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM26.19	Demonstrate ability to work in a team of peers and superiors	Y	Bedside clinic, DOAP session	Skill assessment	1	3–13
IM26.20	Demonstrate ability to communicate to patients in a patient, respectful, non-threatening, non-judgmental and empathetic manner	Y	Bedside clinic, DOAP session	Skill assessment	1	3–13
IM26.21	Demonstrate respect to patient privacy	Y	Bedside clinic, DOAP session	Skill assessment	1	3–13
IM26.22	Demonstrate ability to maintain confidentiality in patient care	Y	Bedside clinic, DOAP session	Skill assessment	1	3–13
IM26.23	Demonstrate a commitment to continued learning	Y	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.24	Demonstrate respect in relationship with patients, fellow team members, superiors and other healthcare workers	Y	Bedside clinic, DOAP session	Skill assessment/ viva voce	1	3–13
IM26.25	Demonstrate responsibility and work ethics while working in the healthcare team	Y	Bedside clinic, DOAP session	Skill assessment/ viva voce	1	3–13
IM26.26	Demonstrate ability to maintain required documentation in health care (including correct use of medical records)	Y	Small group discussion	Skill assessment/ viva voce	1 59	3–13 871–885
IM26.27	Demonstrate personal grooming that is adequate and appropriate for healthcare responsibilities	Y	Small group discussion	Skill assessment	1	3–13
IM26.28	Demonstrate adequate knowledge and use of information technology that permits appropriate patient care and continued learning	Y	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.29	Communicate diagnostic and therapeutic options to patient and family in a simulated environment	Y	Bedside clinic, DOAP session	Skill assessment/ viva voce	1	3–13
IM26.30	Communicate care options to patient and family with a terminal illness in a simulated environment	Y	Bedside clinic, DOAP session	Skill assessment/ viva voce	1	3–13
IM26.31	Demonstrate awareness of limitations and seeks help and consultations appropriately	Y	Bedside clinic, DOAP session	Skill assessment/ viva voce	1	3–13
IM26.32	Demonstrate appropriate respect to colleagues in the profession	N	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.33	Demonstrate an understanding of the implications and the appropriate procedures and response to be followed in the event of medical errors	N	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.34	Identify conflicts of interest in patient care and professional relationships and describe the correct response to these conflicts	Y	Small group discussion	Skill assessment/ viva voce	1	3–13

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
IM26.35	Demonstrate empathy in patient encounters	Y	Bedside clinic, DOAP session	Skill assessment/ viva voce	1	3–13
IM26.36	Demonstrate ability to balance personal and professional priorities	N	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.37	Demonstrate ability to manage time appropriately	Y	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.38	Demonstrate ability to form and function in appropriate professional networks	N	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.39	Demonstrate ability to pursue and seek career advancement	N	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.40	Demonstrate ability to follow risk management and medical error reduction practices where appropriate	N	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.41	Demonstrate ability to work in a mentoring relationship with junior colleagues	N	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.42	Demonstrate commitment to learning and scholarship	N	Small group discussion	Skill assessment/ viva voce	1	3–13
IM26.49	Administer informed consent and appropriately address patient queries to a patient being enrolled in a research protocol in a simulated environment	Y	Bedside clinic, DOAP session	Written/viva voce	1	3–13
INTEGRATIONS						
OG12.1	Define, classify and describe the etiology and pathophysiology, early detection, investigations; principles of management of hypertensive disorders of pregnancy and eclampsia, complications of eclampsia	Y	Lecture, small group discussion, bedside clinics	Written/viva voce/skill assessment	37–40	643–667
OG12.2	Define, classify and describe the etiology, pathophysiology, diagnosis, investigations, adverse effects on the mother and fetus and the management during pregnancy and labor, and complications of anemia in pregnancy	Y	Lecture, small group discussion, bedside clinics	Written/viva voce/skill assessment	37–40	643–667
OG12.3	Define, classify and describe the etiology, pathophysiology, diagnosis, investigations, criteria, adverse effects on the mother and fetus and the management during pregnancy and labor, and complications of diabetes in pregnancy	Y	Lecture, small group discussion, bedside clinics	Written/viva voce/skill assessment	37–40	643–667
OG12.4	Define, classify and describe the etiology, pathophysiology, diagnosis, investigations, criteria, adverse effects on the mother and foetus and the management during pregnancy and labor, and complications of heart diseases in pregnancy	Y	Lecture, small group discussion, bedside clinics	Written/viva voce/skill assessment	37–40	643–667

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
OG12.5	Describe the clinical features, detection, effect of pregnancy and impact of the disease on pregnancy complications and management in pregnancy of urinary tract infections disease and impact of the disease on pregnancy complications and management in pregnancy of liver disease	Y	Lecture, small group discussion, bedside clinics	Written/viva voce/skill assessment	37–40	643–667
OG12.7	Describe and discuss screening, risk factors, management of mother and newborn with HIV	Y	Lecture, small group discussion, bedside clinics	Written/viva voce/skill assessment	37–40	643–667
DR9.1	Classify, describe the epidemiology, etiology, microbiology pathogenesis and clinical presentations and diagnostic features of leprosy	Y	Lecture, small group discussion	Written /viva voce	41–42	671–688
DR9.2	Demonstrate (and classify based on) the clinical features of leprosy including an appropriate neurologic examination	Y	Lecture, small group discussion	Bedside clinic session/skill assessment	41–42	671–688
DR9.4	Enumerate, describe and identify lepra reactions and supportive measures and therapy of lepra reactions	Y	Lecture, small group discussion	Written /viva voce	41–42	671–688
DR10.1	Identify and classify syphilis based on the presentation and clinical manifestations	Y	Bedside clinic	Skill assessment	43	690–691
DR10.6	Describe the etiology, diagnostic and clinical features of non-syphilitic sexually transmitted diseases (chancroid, donovanosis and LGV)	Y	Lecture, small group discussion	Written /viva voce	43	690–691
PS3.7	Enumerate and describe common organic psychiatric disorders, magnitude, etiology and clinical features	Y	Lecture, small group discussion	Written/viva voce	44–45	697–708
PS3.8	Enumerate and describe the essential investigations in patients with organic psychiatric disorders	Y	Lecture, small group discussion	Written/viva voce	46	712
PS10.1	Enumerate and describe the magnitude and etiology of somatoform, dissociative and conversion disorders	Y	Lecture, small group discussion	Written/viva voce	44–45	697–708
PS10.2	Enumerate, elicit, describe and document clinical features in patients with somatoform, dissociative and conversion disorders	Y	Bedside clinic, DOAP session	Skill assessment	44–45	697–708
PS10.3	Enumerate and describe the indications and interpret laboratory and other tests used in somatoform, dissociative and conversion disorders	Y	Bedside clinic, DOAP session	Skill assessment	46	712
PS12.2	Enumerate, elicit, describe and document clinical features in patients with psychosomatic disorders	Y	Bedside clinic, DOAP session	Skill assessment	44–45	697–708

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Number	COMPETENCY The student should be able to:	Core Y/N	Suggested learning methods	Suggested assessment methods	Chapter number	Page number
PS12.3	Enumerate and describe the indications and interpret laboratory and other tests of psychosomatic disorders	Y	Bedside clinic, DOAP session	Skill assessment	46	712
PS16.1	Enumerate and describe common psychiatric disorders in the elderly including dementia, depression and psychosis	Y	Lecture, small group discussion	Written/viva voce	44–45	697–708
CM6.1	Formulate a research question for a study of your choice	Y	Small group discussion, lecture, DOAP sessions	Written/viva voce/skill assessment	56–57	837–852
CM6.2	Describe and discuss the principles and demonstrate the methods of collection, classification, analysis, interpretation and presentation of statistical data	Y	Small group discussion, lecture, DOAP sessions	Written/viva voce/skill assessment	56–57	837–852
CM6.3	Describe, discuss and demonstrate the application of elementary statistical methods including test of significance in various study designs	Y	Small group discussion, lecture, DOAP sessions	Written/viva voce/skill assessment	56–57	837–852
CM6.4	Enumerate, discuss and demonstrate common sampling techniques, simple statistical methods, frequency distribution, measures of central tendency and dispersion	Y	Small group discussion, lecture, DOAP sessions	Written/viva voce/skill assessment	56–57	837–852
CM7.1	Define epidemiology and describe and enumerate the principles, concepts and uses	Y	Small group discussion, lecture	Written/viva voce	56–57	837–852
CM7.2	Enumerate, describe and discuss the modes of transmission and measures for prevention and control of communicable and non-communicable diseases	Y	Small group discussion, lecture	Written/viva voce	56–57	837–852
CM7.3	Enumerate, describe and discuss the sources of epidemiological data	Y	Small group discussion, lecture	Written/viva voce	56–57	837–852
CM7.4	Define, calculate and interpret morbidity and mortality indicators based on given set of data	Y	Small group discussion, DOAP sessions	Written/skill assessment	56–57	837–852
CM7.5	Enumerate, define, describe and discuss epidemiological study designs	Y	Small group discussion, lecture	Written /viva voce	56–57	837–852
CM7.6	Enumerate and evaluate the need of screening tests	Y	Small group discussion, DOAP sessions	Written/skill assessment	56–57	837–852
CM8.3	Enumerate and describe disease-specific National Health Programs including their prevention and treatment of a case	Y	Small group discussion, lecture	Written/viva voce	58	856–865
CM8.5	Describe and discuss the principles of planning, implementing and evaluating control measures for disease at community level bearing in mind the public health importance of the disease	Y	Small group discussion, lecture	Written/viva voce	58	856–865

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CHAPTER

Investigations in Neurological Diseases

AS Girija, KV Krishna Das

Chapter Outline

- General Investigations
- Neurological Investigations
- Lumbar Puncture
- CSF Examinations
- Special Neurological Investigations
- Electroencephalography
- Magnetoencephalography
- Nerve Conduction Studies
- Evoked Potentials
- Electromyography
- Muscle Enzymes
- Radiological Studies
- Computed Tomographic Scan
- Magnetic Resonance Imaging
- Positron Emission Tomography
- Echoencephalography
- Imaging Studies of Muscle
- Duplex Doppler Scanning
- Histopathological Studies
- Genetic Studies in Neurology

INTRODUCTION

These investigations may be broadly classified into the following varieties:

a. *General investigations:* These are to be undertaken by the family physician at first examination by itself. These give clues to the involvement of the nervous system in systemic diseases, such as diabetes mellitus, syphilis or malaria.

b. *Specific investigations:* Investigations, such as lumbar puncture, radiography of skull and spine, ultrasonography, computed tomography, and magnetic resonance imaging have to be done by the internist, mainly under hospital setting. These help to diagnose conditions, such as meningitis, seizures, tumors, abscesses, subdural hematomas, bony lesions of the skull, and others.

c. *Specialized investigations:* Investigations, such as angiography, myelography, electrophysiological studies, such as electroencephalography (EEG), electromyography (EMG), isotopic tests, and biopsy studies which help the neurologist to arrive at the final diagnosis.

GENERAL INVESTIGATIONS

These investigations include the following: Urinalysis for albumin, sugar, phenylalanine, other aminoacidurias, constituents of bile, porphobilinogen, and microscopic abnormalities, such as excess of erythrocytes and casts.

Biochemical Tests

1. *Blood glucose level*: Hypoglycemia, generally with blood levels below 50 mg/

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dL, gives rise to neuroglycopenia, which manifests as clouding of consciousness, convulsions, and coma. When blood glucose level goes above 400 mg/dL, the chances to develop hyperosmolar coma increase.

2. *Blood urea, uric acid, creatinine:* These are elevated in renal failure. Uremia leads to several neurological complications and neuropsychiatric abnormalities.
3. *Blood ammonia level:* It is elevated in hepatic failure, which may be the cause of neuropsychiatric complications in acute or chronic liver disease.
4. Abnormal increase of *amino acids* in urine is found in several metabolic disorders, e.g., *phenylketonuria.*
5. Reduction in the blood levels of *ceruloplasmin* and elevated levels of *serum copper* in blood and tissues is a diagnostic finding in hepatolenticular degeneration (*Wilson's disease*).
6. *Muscle enzymes*, such as creatine phosphokinase and aldolase are increased in muscle dystrophies.

Several neurological abnormalities can be diagnosed by biochemical investigations. The reader should refer to monographs on neurology for a complete list.

Hematology

Hemoglobin Level

Anemia leads to headache, giddiness, throbbing in the head, syncope, and retinal changes, such as hemorrhage and papilledema. These are more pronounced when the hemoglobin level drops below 6 g/dL. These clear up when the hemoglobin level is restored to normal.

Total and Differential Leukocyte Counts

Acute leukemias, particularly acute lymphatic leukemia leads to neurological involvement which may present clinically with the picture of meningitis, raised intracranial tension, convulsions, retinal hemorrhages, tumor formation, and various forms of paralysis. In children with rapidly progressing neurological abnormalities, neuroleukemia should be kept in mind.

Neutrophil leukocytosis may accompany bacterial meningitis. Lymphocytosis in the presence of meningitis may suggest viral etiology or tuberculosis.

Platelets

Thrombocytopenia from any cause may lead to intracranial hemorrhage if the platelet count falls below 20,000/cmm. In fulminant purpura and disseminated intravascular coagulation, intracranial bleeding is a fatal complication.

Coagulation Tests

Both therapeutic anticoagulation and natural coagulopathies may be complicated by intracranial bleeds. In such cases, parameters to assess the hemostatic profile, e.g., bleeding and clotting time, prothrombin time, partial thromboplastin time, assays of coagulation factors, studies of platelet number and function, and excessive fibrinolysis are to be undertaken.

Hypercoagulable States

Thrombocytosis, thrombocythemia, hyperviscosity states, dehydration, and congenital deficiency of naturally occurring antithrombotic substances, such as protein C and protein S lead to venous and arterial thrombosis in the CNS. Management of these cases is the realm of the hematologist. Several hematological diseases, such as sickle cell anemia and leukemias lead to neurological syndromes.

Serological Tests

Both syphilis and AIDS are common to produce neurological complications and, therefore, routine testing is indicated in a suspected case—VDRL in the CSF for syphilis and Elisa and Western blot tests for AIDS.

Parasitology

Falciparum malaria, trypanosomes, and cysticerci are common to involve the CNS. Malaria and trypanosomiasis can be diagnosed by blood smear examination. Cysticercosis can be diagnosed by serological tests and the presence of these cysts in the brain, which will be revealed by CT scan, but better by MRI.

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NEUROLOGICAL INVESTIGATIONS

Lumbar Puncture

Lumbar puncture (LP) is performed to obtain samples of cerebrospinal fluid (CSF) for analysis, introduce therapeutic or investigational agents into the lumbar subarachnoid space, and for measuring CSF pressure. It is an invasive procedure. With the advent of CT and MRI scans, many neurological centers have reduced the number of lumbar punctures, especially when diagnostic information can be obtained otherwise and when the procedure is likely to be risky. However, general physicians have to perform this procedure in many patients with neurological disorders since it helps to arrive at the diagnosis and it can be performed even in moderately equipped hospitals. At times the procedure can be hazardous and therefore, great care has to be taken in patient selection and performance of the procedure. In centers where facility for computed tomogram (CT) is available, it is better to make a preliminary assessment of the intracranial pathology before undertaking lumbar or cisternal punctures. When judiciously employed and skillfully performed, it is one of the most valuable diagnostic procedures in neurology. Hence, every trainee in internal medicine has to be conversant with this procedure.

Indications for LP

1. To confirm the diagnosis of meningitis, encephalitis, neurosyphilis, and other infections when clinical picture is suggestive.
2. To rule out or confirm subarachnoid hemorrhage when computerized tomography imaging facilities are not available, and when meningitis has to be excluded.
3. Suspected involvement of CNS in leukemia and lymphoma.
4. Guillain-Barré syndrome, multiple sclerosis, some cases of peripheral neuropathy, and transverse myelitis.
5. Idiopathic intracranial hypertension to document the exact CSF pressure using a manometer.
6. Normal pressure hydrocephalus as a therapeutic procedure.
7. To introduce contrast agents, such as iodinated dyes into the subarachnoid space during imaging procedures, such as CT myelogram which is done rarely as MRI studies are now increasingly done.
8. To introduce therapeutic agents into CSF, e.g., antibiotics for meningitis, antileukemic drugs in acute lymphatic leukemia, and others.
9. To administer spinal anesthesia.
10. As a therapeutic measure to reduce intracranial pressure in idiopathic intracranial hypertension, neonatal intraventricular hemorrhage, and hydrocephalus complicating meningitis.

Contraindications for LP

1. Sepsis at the site of lumbar puncture. The infection may spread into CSF and lead to meningitis.
2. Possibility of raised intracranial pressure, particularly due to intracranial space occupying lesions. If CSF is released from below in the presence of raised intracranial tension, downward herniation of brain occurs leading to pressure effects and reduction in blood supply of the brainstem, resulting in respiratory paralysis and death. This is known as "coning effect." The common structures that herniate are the uncus of the temporal lobe through the tentorium cerebelli and cerebellar tonsils into the foramen magnum.
3. Possibility of spinal cord compression: Sudden release of CSF pressure from below may worsen cord compression in cases of spinal tumors, edema or infection of the cord, and others.
4. Hypersensitivity to the drugs and anesthetic agents employed.

Note: Absence of papilledema does not always exclude increased intracranial pressure caused by intracranial space occupying lesion. Therefore, if the latter condition is suspected clinically, a CT scan of the head should be done prior to LP.

However, idiopathic intracranial hypertension is one condition where measurement

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of CSF pressure is the confirmatory test and even in the presence of severe papilledema, if CT scan rules out space occupying lesion, LP has to be performed. In idiopathic intracranial hypertension, coning of the brainstem is rare. In all difficult and potentially dangerous situations, it is preferable to perform LP only in centers where neurosurgical help is available to tackle the postlumbar puncture complications. It should be remembered that conditions, such as meningitis and encephalitis where delay in specific treatment may lead to mortality or severe morbidity, LP should not be delayed, since CSF examination is the gold standard of diagnosis. If there is any contraindications or LP cisternal puncture should be done to collect CSF. As it is an invasive procedure planning regarding amount, of CSF to be collected, the tests to be done should be noted and a small quantity should be kept reserved for new tests if needed on follow-up.

Procedure of LP

Explain the procedure to the patient and relatives and get informed consent. The LP should be done under strict aseptic precautions as for a minor surgical procedure.

i. *Position of the patient:* ***Make the patient lie on the edge of a hard bed on his left side*** with his back perpendicular to the bed. Keep him in "fetal" position, i.e., knees drawn up toward the chest and the head flexed, so as to widen the interspinous spaces maximally.
   Preparation of skin at the puncture site: Expose his back from mid thoracic to midgluteal region. The skin is thoroughly cleaned in turn with spirit, iodine, and spirit or povidone iodine. Drape the area with sterile towels, exposing only the area to be punctured.

ii. *Localization of the puncture site:* LP is done usually through the interspinous space between L3-L4 vertebrae. In infants, the L4-L5 space is selected due to low placement of the conus medullaris. The L3-L4 space can be easily identified as it lies just below the line joining the highest points on the iliac crests. Select this interspace and mark it.

iii. *Local anesthesia:* It is produced by injecting about 2 mL of 2%, lignocaine solution, first into the skin and then into deeper tissues. Start the LP only after ensuring that the spot is anesthetized.

iv. *Insertion of* LP *needle:* The LP needle is a special hollow needle about 8 cm long with a sharp, short bevel at its tip and provided with its stylet. LP needles are available in sizes of 9, 18, 20, and 21 Gauge. The higher the number, the smaller the bore and length of the needle. For an average sized adult, 18 or 19 Gauge needle should be used. For children, 20 or 21 Gauge is to be preferred.
   Stretch the skin over the site of puncture at the same time feeling for the interspace with the tip of your left thumb. Insert the LP needle containing the stylet keeping the bevel parallel to the long axis, so that the needle tip only splits the longitudinal fibers of ligamentum flavum and the dura mater, without cutting them. Push the needle firmly inward keeping it exactly in the midline at right angles to the body curvature and with a slight cephalic inclination.
   The ***position and direction of the needle movement are crucial to get successful LP.*** If the needle gets deflected or encounters bony resistance, withdraw the needle up to the subcutaneous tissue and reintroduce it at a different angle. Do not change the direction of the needle when it is midway in the deeper tissues. When properly introduced to a depth of about 4.5 cm a slight resistance will be felt due to ligamentum flavum. A feeling of "giving way" indicates puncture of this ligament, the dura mater, and entry of the needle tip into subarachnoid space. At this stage, remove the stylet. When LP is successful, the CSF flows out as the stylet is removed. Allow only a few drops of the fluid to escape and reinsert the stylet immediately. The patient is made to lie relaxed with his legs extended, head straight, and

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breathing freely. If no CSF emerges, slowly withdraw the needle 1 to 2 mm by rotating it. This will help to clear any obstruction to the needle tip, and start CSF flow. If the patient feels a sudden shooting pain in one lower limb, it indicates that a sensory root has been touched by the needle tip, and that the needle tip is too far away from the midline.

If no CSF emerges, refix the stylet, advance the needle a bit more, and then remove the stylet to see CSF flow. If you feel that you have entered the subarachnoid space by the "giving way" feeling on the needle, but still no CSF is coming out, perform Queckenstedt's test to raise spinal CSF pressure and promote flow.

If this is also unsuccessful, withdraw the needle up to the subcutaneous tissue and reintroduce it in a different direction. If two or three attempts fail, try the procedure through the adjacent interspinous space or defer the procedure to a subsequent occasion. A failed LP is most often due to faulty technique. If the patient is restless and has behavior problem, he may be sedated with intravenous phenergan or in severe cases parenteral haloperidol 5 mg may be given as a last resort.

An alternate position to do LP is with the patient sitting up with his legs hanging by the side of the couch, and leaning forward over a pillow held firmly in front of his abdomen, so that his face touches his thighs. In this position, the interspinous spaces are widened maximally. Once the LP needle has reached the subarachnoid space and CSF comes out, the patient is made to lie in the lateral decubitus. CSF pressure is measured and further procedures done in this position.

Sometimes when the CSF pressure is too low, as in severe dehydration, the fluid may not come out spontaneously. In such cases, perform Queckenstedt's test to get more fluid. If the CSF is very thick due to purulent inflammation, it may not flow out through a narrow needle.

v. *Measurement of CSF pressure:* In all cases where abnormalities of CSF pressure are suspected, manometry should be done to measure the CSF pressure. The patient should be in the lateral decubitus with the head and sacrum at the same level and the legs fully extended, and breathing freely. The LP manometer is a calibrated glass tube with 1 mm bore, 30 cm long and provided with a reservoir at its top. It is connected to the LP needle through an adapter, allowing only minimal CSF to escape during the procedure.

Normally, the opening pressure is about 60 to 150 mm of CSF, with 1 to 2 mm oscillation, synchronous with each pulse, and 1 to 2 cm fluctuation with each respiration. The pressure as seen in the manometer is recorded.

Pressures between 130 to 200 mm of CSF are borderline increases and above 200 mm are definitely abnormal. Fallacies occur if the patient is tense and not fully relaxed or the head is flexed, thereby the jugular veins are compressed. These factors should be corrected before taking the final reading. If the pressure is genuinely elevated, disconnect the manometer and withdraw the LP needle after inserting the stylet. The CSF obtained from the manometer can be used for further tests.

Even while doing LP note the rate of flow of CSF, its color and consistency and the responses to Queckenstedt's test. If possible, collect the first few drops directly on glass slides for examining the cellular content and for Gram staining in the case of purulent CSF or opalescent CSF.

Queckenstedt's test: This test is employed to determine whether there is spinal block, i.e., any block between the intracranial and lumbar subarachnoid space. In conditions, such as spinal arachnoiditis, spinal block may develop.

With the patient lying in the lateral decubitus, and CSF flowing freely through the LP needle or during spinal manometry, compress the internal jugular vein of one side at the root of the neck for about 10 seconds. This hinders venous return from the intracranial venous sinuses and thus elevates intracranial pressure. This rise is reflected in the lumbar subarachnoid space as a rise in

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pressure in the manometer or more rapid flow of CSF. On releasing the compression in the neck, the CSF flow returns to the original rate. This test is hardly used now as MRI is routinely done in suspected cases of cord compression due to tumors which was the most common cause of low-pressure CSF.

If the lateral or sigmoid venous sinus of one side is occluded by thrombus, there will not be rise of intracranial pressure on compressing the jugular vein on that side. Queckenstedt's test is contraindicated in the presence of raised intracranial pressure.

Collection of CSF: While performing LP, the CSF should be collected for the following tests, as per the clinical indications. In all about 5 mL of CSF may be collected in three sterilized containers.

CSF EXAMINATIONS

Microscopic Examination (Table 34.1)

1. *Cell count:* Normal CSF is clear, such as water, colorless, and contains only up to 2 to 5 lymphocytes per cmm. Pleocytosis is the presence of excessive number of leukocytes. When more than 200 cells/cmm are present, the fluid may become opalescent. Presence of more than 1000 cells/cmm makes it frankly turbid and purulent. Purulent CSF is suggestive of bacterial meningitis and the predominant cells are neutrophils. Morphology of the cells can be made out by wet-staining with methylene blue. Absolute count can be done as is done for WBC in a Fuchs-Rosenthal counting chamber. CSF pleocytosis may be due to lymphocytes, granulocytes, or mixed. Presence of excess cells is the most definite diagnostic evidence of meningitis. In infiltration of the central nervous system by leukemia, lymphoma, and carcinomas, the corresponding abnormal cells appear in CSF.
2. *Gram staining:* This is the most easy and readily available rapid method to diagnose the presence of bacterial meningitis and identify the organism. In all cases of purulent meningitis, this should be done. Morphological identification of the organism *(Pneumococcus, Streptococcus, Meningococcus, H. influenzae,* and *E. coli)* helps to institute specific antibiotic therapy without delay. Acid fast staining of the cobweb formed in the CSF in TB

TABLE 34.1: Diagnostic features of CSF in the common forms of meningitis.

Test	*Normal*	*Pyogenic*	*TB*	*Meningitis Viral*	*Fungal*	*Syphilitic*
Appearance	Clear colorless	Turbid	Clear or slightly opalescent	Clear	Clear	Clear
Pressure	60–150 mm of CSF	Raised	Raised	Raised	Raised	Raised or Normal
Proteins	20–40 mg/dL	500–1000 mg/dL	300–400 mg/dL	180–200 mg/dL	up to 200 or more	80–100 mg/dL
Sugar	40–80 mg/dL	Below 40	Below 40	Normal	Reduced	Normal
Chlorides	720–750 mg/dL	Reduced	Considerably reduced	Normal	Reduced	Normal
Cells	0–5/cmm, all lymphocytes	1000/cmm or more, mostly neutrophils	150–200/cmm or more, mostly lymphocytes	100–200/cmm or more mostly lymphocytes	100–200/cmm or more mostly lymphocytes	100–200/cmm or more mostly lymphocytes
Culture	Sterile	Organisms isolated	*Mycobacterium* in 20–30%	Viruses may be isolated	Fungi can be grown	*Treponema* can be demonstrated or grown by special techniques
VDRL	Negative	Negative	Negative	Negative	Negative	Positive

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meningitis may reveal mycobacteria. India ink staining reveals *Cryptococcus*.

Biochemical Tests

Protein content, glucose, chlorides, immunoglobulins, and several others can be estimated. Normal CSF contains 20 to 40 mg/dL proteins, 60 to 80 mg/dL glucose, and chlorides depending on the serum chloride level.

Proteins: Moderate to high rise in protein content occurs in inflammatory conditions, such as meningitis and encephalitis. Maximum elevation occurs in spinal block and Guillain-Barré syndrome. In infective lesions, the protein content and cell count rise together, whereas in spinal block and Guillain-Barré syndrome, proteins increase without corresponding rise in cells (albuminocytological dissociation).

Glucose: CSF glucose levels vary directly with the blood glucose level. Generally, the CSF glucose is 20 to 30 mg below the blood glucose levels, if the specimens are taken at the same time. Disproportionate fall in CSF glucose occurs in all types of bacterial meningitis. In tuberculous meningitis, substantial reduction of CSF glucose is a very valuable diagnostic clue which helps to arrive at the provisional diagnosis early. In viral meningitis, encephalitis and neuroleukemia in which lymphocytes may be seen in excess, the CSF glucose is not diagnostically altered.

Chlorides: The CSF chloride levels vary directly with the blood chloride levels. Low CSF chloride levels occur in dehydration and electrolyte depletion. It is not of specific diagnostic importance.

Immunoglobulins: Identification and estimation of IgG may help in the diagnosis of multiple sclerosis, sarcoidosis, and connective tissue diseases. Normally, IgG in CSF is 6 to 12% of the total proteins. Values above 20% are abnormal. Electrophoresis of CSF may reveal oligoclonal bands in the gamma globulin region in multiple sclerosis.

Special studies of CSF—Estimation of PCR test for tuberculosis, Herpes simplex infection, autoimmune antibodies, e.g., NMDA receptor antibodies, etc., may be needed based on clinical diagnosis.

Note: Normal CSF does not show any coagulum or clot on standing. In tuberculous meningitis, the CSF may show a "cobweb" like precipitate on keeping at room temperature for 12 to 24 hours. Staining of the cobweb may show acid fast bacilli in 20 to 30% of cases of tuberculous meningitis.

Frank coagulation of CSF on standing occurs when the protein content is very high. This may occur in spinal block and Guillain-Barre syndrome. The combination of xanthochromia (yellow color), spontaneous clotting, and albuminocytological dissociation of CSF is called Froin's syndrome which occurs in spinal block.

Microbiological Test

CSF has to be sent for culture of bacteria, fungi, and viruses. Etiological diagnosis of infective lesion can be made by this method. Cultures for *M. tuberculosis* take about 4 to 6 weeks to grow. Therefore, newer methods, such as immunofluorescence or DNA techniques, such as polymerase chain reaction (PCR) have been devised.

For demonstrating *Cryptococcus* in CSF, a simple method is to mix equal amounts of CSF and India ink; and examine the wet preparation under high power of the microscope. Cryptococci are seen as refractile dots with a clear circular zone around.

Several other tests are available which are to be employed by the neurologist under particular circumstances.

Blood Stained CSF

This may be due to injury to one of the veins during LP or it may be due to subarachnoid hemorrhage. These can be easily distinguished by the points given in **Table 34.2**. In subarachnoid hemorrhage, the erythrocytes break down releasing bile pigments which stain the CSF yellow. This is seen about 6 hours after the bleed.

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Complications of LP

1. *Coning of the brainstem:* This should be suspected if:
   a. The patient becomes less alert
   b. The pupils start dilating
   c. The respiration becomes shallow and slower.
   To avoid this, the patient should be made to lie prone soon after LP. If coning is suspected, immediately give 20% mannitol 200 mL intravenously rapidly and summon the help of a neurologist or neurosurgeon.
2. *Postlumbar puncture headache:* This is due to disturbance of CSF hemodynamics and leak of CSF into extradural space. This is minimized by using the smallest bore needle for LP. The headache stops spontaneously over hours. Sometimes, it may be prolonged and distressing. Analgesics may help, so also rehydration of the patient and reassurance.
3. Infection and secondary meningitis are a rare, but grave complication. This should be avoided by strict aseptic precautions.
4. Chemical meningitis may result from irritation by the drug introduced into the CSF.

TABLE 34.2: Differentiation in CSF between traumatic bloody tap and subarachnoid hemorrhage.

Findings	*Traumatic blood staining*	*Subarachnoid hemorrhage*
1. Observation of CSF on flowing from the LP needle or if it is collected in three successive tubes	Tends to become clearer	Uniform blood staining
2. CSF is kept at room temperature for 1 hour	The blood clots	Does not clot, but the erythrocytes settle to the bottom. In SA hemorrhage defibrination takes place
3. Supernatant fluid observed after centrifuging for 2 minutes at 3000 RPM, soon after obtaining the CSF	Supernatant is colorless	Supernatant is xanthochromic due to the presence of bile pigment

Alternative Methods to Obtain CSF

When examination of the CSF is mandatory and absolutely necessary to establish the diagnosis, but where the LP fails repeatedly, CSF can be obtained by cisternal puncture or cerebral ventricular puncture.

SPECIAL NEUROLOGICAL INVESTIGATIONS

	Tests for structural integrity	*Tests for functional integrity*
Brain	Computed tomography (CT) Magnetic resonance Imaging (MRI) Cerebral angiography (conventional) Digital subtraction angiography (DSA) MR angiography Radioisotope scan Echoencephalography Transcranial Doppler	Electroencephalography (EEG) Evoked potential (EP) study Brain electrical activity mapping (BEAM) Single photon emission computed tomography (SPECT) Positron emission tomography (PET)
Brain biopsy		
Spinal cord	CT scan MRI scan	Evoked potentials CT myelography
Nerve	Nerve biopsy	Nerve conduction velocity (NCV) study, evoked potentials
Muscle	Muscle biopsy	Electromyography (EMG)
Neuromuscular junction		Muscle enzymes-CPK and LDH Repetitive, electrical nerve stimulation

General Considerations

Many diseases of the nervous system demand specialized investigations to know the structural and functional abnormalities and to help in the diagnosis, management, follow-up, and prognosis. Since many of these investigations require costly equipment and experienced personnel, these are available only in a few places in India. Therefore, there is great need to select the appropriate

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investigation in a cost-effective manner. The following account gives a brief description of various investigations available with their indications, advantages, and limitations. Any investigation in a given patient will give either normal results or reveal some abnormalities. The interpretation of these results also has to be done in the light of the clinical settings.

ELECTROENCEPHALOGRAPHY

Electroencephalography (EEG) is a specialized neurological investigation which involves recording of the spontaneous electrical activity of the brain from the scalp. This is a safe, noninvasive, relatively inexpensive, painless, easily repeatable procedure to know the functional status as opposed to the CT or MRI scan which reveals the structural integrity of the brain. Since the EEG findings in many of the cerebral disorders are nonspecific, its diagnostic value is confined to a few conditions, such as epilepsies, encephalitides, metabolic, toxic, anoxic, and drug-induced encephalopathies and in the study of sleep disorders.

The modern EEG machine has 8 to 16 channels to record the brain activity simultaneously from many areas of the scalp. Electrodes, which are usually silver chloride disks are applied to the scalp, by means of an adhesive material which also provides good electrical contact between the electrode and the skin, after the hair and the scalp are cleaned of oil and grease. The placement of these electrodes on the scalp is standardized internationally (10–20% system). The tracing is recorded digitally with computerized technique for about 30 to 60 minutes during the awake state, while the patient comfortably lies in a quiet room. The patient need not fast for this test nor miss his medication, including the anticonvulsants. Ideally, the first EEG may be taken after sleep deprivation in suspected cases of epilepsy before starting treatment. The best time is in the immediate postictal period or within 2 weeks of an episode of seizure. In many laboratories, hyperventilation for 3 minutes and photic stimulation at different rates with a flickering light (strobe) are used as provocating procedures.

The EEG tracing is studied to note the frequency, amplitude, morphology, and topography of the brain electrical activity. Four types of frequency bands of EEG are encountered. They are: (i) delta (< 4 Hz activity), (ii) theta (4–8 Hz), (iii) alpha (8–13 Hz), and (iv) beta (> 13 Hz) bands.

The EEG of a normal adult in the awake and resting state with eyes closed consists of alpha rhythm of 30 to 60 microvolts over the posterior regions of the head. In children, the frequency is slower in the theta or delta range, depending on the age.

The abnormalities in the EEG may be in the form of asymmetrical activity, faster or slower frequency activity, low or high amplitude activity, or abnormal wave forms, such as sharp waves, slow waves, and/or spikes. Localized or focal abnormalities, though highly significant, cannot reliably distinguish an underlying pathological lesion, such as tumor, abscess, hematoma, or infarct.

EEG is of greatest value in seizure disorders not only to document the interictal subclinical seizure activity but also to classify the epilepsy as partial or generalized, depending on whether the seizure activity is focal or generalized. Sporadic spikes, sharp waves, spike and slow wave complexes or 3 Hz spike, and wave complexes in petit mal epilepsy are some of the EEG patterns, which are diagnostic **(Figs. 34.1A and B)**. However, it should be mentioned here that a normal interictal EEG does not exclude epilepsy because in only 50 to 60% of these patients EEG will be abnormal.

The characteristic periodic EEG complexes in subacute sclerosing panencephalitis and Jacob Cruetzfeldt disease, the triphasic waves in hepatic coma and the electrocerebral silence in brain death are some of the other diagnostic EEG patterns.

Video Encephalography

Video encephalography is a more useful technique to detect intermittent seizure activity and distinguish between organic and functional diseases. It is invaluable in the presurgical evaluation in intractable epilepsy.

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Figs. 34.1A and B: Normal EEG and EEG in seizure.

MAGNETOENCEPHALOGRAPHY

Unlike EEG, magnetoencephalography (MEG) detects the magnetic fields emanating from the brain tissue. It is much more sensitive than EEG in detecting deep seated small pathological lesions in the brain. However, it is very expensive and hence is not easily available for clinical studies.

NERVE CONDUCTION STUDIES

Nerve conduction study is employed to test the integrity of a peripheral nerve. The presence or absence of denervation, the site of nerve block if any, and nature of the lesion, such as demyelination or axonal lesions can be detected easily even if the patient is not cooperative. Hence, this test can be employed in infants, children, and in comatose patients.

Nerve conduction velocity studies (NCV) are of two types:

i. Motor nerve conduction velocity (MNCV) study
ii. Sensory nerve conduction velocity (SNCV) study.

In motor nerve conduction studies, a simulating surface electrode is placed on the

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skin overlying a peripheral nerve and a brief electric shock, slightly greater than that required to produce a maximal contraction of the innervated muscle (supramaximal stimulus) is given to produce muscle contraction. The action potential generated by the contracting muscle is picked up by recording electrodes placed on the skin over the muscle and this is displayed on a cathode-ray oscilloscope for analysis. The action potential of the muscle occurs a few milliseconds after the stimulus is delivered (stimulus artifact). The time taken for the stimulus impulse to travel along the nerve and the neuromuscular junction from the stimulation artifact to the beginning of the evoked muscle action potential is called latency, and it is measured in milliseconds. The duration and amplitude of the evoked muscle action potential are also measured.

For determining nerve conduction velocity, the same nerve is stimulated at two points along its course. The difference in the latency times of these two stimuli gives the conduction time between the two points of stimulation. The distance between these two sites is measured and the NCV calculated as meters/second. Normal is 50 to 60 m/sec.

NCV studies are valuable (i) to detect demyelination in the peripheral nerves where the NCV will be significantly slowed down and (ii) to detect axonal lesion where the NCV may be within normal limits but the evoked muscle action potential will be of abnormally low amplitude and longer duration. NCV study also helps to localize the site of pathology in the peripheral nerves.

Using special techniques, it is also possible to study electrophysiologically the integrity of the proximal portion of the nerve roots and the reflex arc.

Routinely, motor nerve conduction studies are performed on median and ulnar nerves in the upper limbs and the lateral popliteal nerves in the lower limbs.

Sensory nerve conduction studies are performed by stimulating the sensory nerves distally and picking up the nerve action potentials proximally. Being very small in amplitude, these potentials have to be amplified to a greater degree and averaged through a computer to make them more discernible. The common sites selected for distal stimulation are the index finger for the median nerve, the little finger for the ulnar nerve, and below and in front of the ankle for the sural nerve **(Table 34.3)**.

TABLE 34.3: The normal values for the commonly studied nerves in adults.

Nerve	*Distal latency (milli-seconds)*	*Amplitude (peak to peak)*	*Maximum conduction velocity (meters/sec)*
Ulnar (motor)	3.0	5 mv	50
Ulnar (sensory)	3.2	5 μv	51
Median (motor)	3.8	5 mv	50
Median (sensory)	3.5	10 μv	51
Radial (motor)	2.9	7 mv	52
Radial (sensory)	3.3	15 μv	50
Peroneal (motor)	5.0	4 mv	44
Tibial (motor)	5.1	5 mv	43
Sural (sensory)	4.4	5 μv	40
H-reflex latency	35	—	—
F-wave latency		—	—
Upper limbs	30	—	—
Lower limbs	50	—	—

Note: However, it should be remembered that the normal values may vary from laboratory to laboratory because of differences in the techniques used.

Normal Values for Nerve Conduction

Nerve conduction velocities at birth are about 50% of normal adult values, increasing rapidly to about 75% by 1 year and reach adult values by about 4 to 5 years of age. There is gradual slowing of conduction velocities as the age advances (3% decline per decade after 30 years) with a steep decline after the age of 60 years. Upper extremity nerve conduction velocities are generally 10 to 15% faster than those of the lower extremity nerves. Sensory nerve conduction time is about 5% faster than motor conduction time.

For diagnosing neuromuscular disorders, such as myasthenia gravis, a peripheral nerve, such as median or ulnar nerve or the brachial plexus at the Erb's point in the neck is stimulated

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by delivering a series of supramaximal stimuli and the evoked response is recorded from the corresponding muscles. A significant decrease in the amplitude of the third or fourth evoked muscle response as compared to the first response suggests myasthenia gravis. This phenomenon is known as decremental response. In Eaton-Lambert syndrome, there is significant increase in action potential with repeated stimulation.

EVOKED POTENTIALS

It is possible to study the functional integrity of the sensory pathways from the periphery to the cerebral cortex by stimulating the peripheral sensory nerves or their receptors. Electrical potentials (EPs) which are generated in the central nervous system are called evoked potentials. Those commonly employed include:

1. *Visual evoked potentials* (VEPs), elicited by stimulating the rods and cones in the retina by a flash of light, or more commonly by a checker board pattern on a TV screen.
2. *Auditory evoked potentials* (AEPs) elicited by stimulating the ears by clicks through head phones.
3. *Somatosensory evoked potentials* (SSEPs) produced by stimulating the peripheral nerves with electrical current.

The evoked electrical potentials, which are of very low amplitude, are averaged by a computer and amplified by special equipment. These amplified potentials are picked up by surface electrodes on the scalp or over the spine in the case of SSEP. By knowing the normal time taken for the impulse to travel from the site of stimulation to the cortex (latency), it is possible to find out in a given patient, whether his EP latencies are normal or not. If abnormal, it is also possible to localize the site of abnormality in the sensory pathways. The advantage of these studies is that they do not depend on subjective sensations by the patient and also they can be performed in comatose individuals, in infants, children, and uncooperative adults under sedation.

ELECTROMYOGRAPHY

Electromyography (EMG) is an investigation in which the electrical activity of muscle is recorded through a needle electrode inserted into the muscle and it is displayed on a cathode ray oscilloscope and played over a loud speaker for simultaneous visual and auditory analysis. Usually, this is performed by the neurologist and is associated with some amount of discomfort to the patient because of multiple needle insertions into the muscle.

Basically, it is carried out to know whether a muscle is normal or shows evidence of myopathy or neurogenic lesion, both of which can lead to muscle weakness and wasting. A normal muscle is electrically silent when it is at complete rest.

When it contracts minimally voluntarily, the action potentials of motor units that are activated appear on the oscilloscope which have a certain configuration, amplitude, and duration. With more and more vigorous voluntary contraction, the number of these action potentials increases and during maximal contraction which recruits all the motor units for contraction, these potentials merge with each other to give full recruitment pattern.

When the muscle contraction ceases, again the muscle becomes electrically silent.

When a muscle becomes weak due to a primary muscle disease, such as myopathy, its EMG pattern differs from that of a normal muscle. When the affected muscle is at rest, there will be spontaneous electrical activity in the form of fibrillation potentials. During minimal voluntary contraction, the generated action potentials will be of lower amplitude and shorter duration as compared to a normal muscle and during maximal contraction, the recruitment potentials will be full. As against this, a muscle which is weak due to a neurogenic lesion, i.e., its neural connection to the anterior horn cells of the spinal cord is affected, it shows spontaneous electrical activity at rest in the form of fibrillation and fasciculation potentials and during minimal voluntary contraction, the action potentials will be of larger amplitude and longer duration

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than normal and during maximal contraction, the recruitment potentials will be less than normal.

Thus, EMG helps to differentiate muscle weakness due to a primary muscle disease from that due to a nerve or anterior horn cell disease. But it does not indicate the causes or etiology of the diseases for which other supplementary investigations are required. Many a time, it is possible to distinguish primary myopathy from neuropathy clinically, but occasionally, it is difficult. EMG is most valuable to distinguish such cases. Characteristic EMG pattern occurs in myotonic disorders (dive bomber sound) and EMG also helps to differentiate true myotonia from pseudomytonia and true weakness from hysterical weakness. Muscle enzyme levels, such as creatine phosphokinase (CPK) and lactic dehydrogenase (LDH) in the serum may transiently increase, following an EMG investigation because of the needle trauma to muscles. Therefore, the blood samples for estimation of these enzymes should be collected before performing the EMG.

MUSCLE ENZYMES

There are several enzymes in the muscles which will be released into circulation whenever there is injury to the muscles. Of these, **creatinine kinase (CK)** is the most important one. The CK is an enzyme found primarily in skeletal muscles, heart, and brain with concentration in skeletal muscles being more than three times that in the heart and brain. There are three forms of CK isoenzymes—MM (skeletal muscles), MB (cardiac muscle), and BB (brain). Normal adult skeletal muscle contains about 95% MM and 5% MB. The normal serum CK concentration is <130 U/L.

Muscle damage due to any cause significantly elevates CK levels in the serum. It should be remembered that normal exercise, an IM injection, or even EMG examination with needle electrodes may elevate CK levels as much as three- to eight-fold which may take 24 to 48 hours to return to normal level.

Marked CK elevation occurs only in polymyositis or dermatomyositis, some types of muscular dystrophies, and hypokalemic myopathy. Serum CK is also elevated in acute myocardial infarction, collagen vascular disease, burns, chest trauma, status epilepticus, brain infarction, hyperthermia, hypothyroid myopathy, alcoholic myopathy, and after surgery. Certain drugs, such as lovastatin, clofibrate, and aminocaproic acid may elevate serum CK level.

An elevation of serum CK is a much more sensitive determinant of muscle damage than elevation of any other muscle enzyme (e.g., LDH, aldolase).

RADIOLOGICAL STUDIES

Plain X-rays

Plain X-rays of the skull and the spine are useful mostly in traumatic lesions. Their ready availability in almost all parts of India is of considerable advantage.

Skull X-rays detect osteolytic lesions of the skull vault as is seen in metastatic deposits and multiple myeloma, osteosclerotic changes as in Paget's disease, and fractures due to head trauma, infections, such as osteomyelitis, bony erosions of the petrous temporal bone in acoustic neuroma and enlargement of the foramina at the bases of the skull. Increased intracranial tension can be diagnosed by beaten silver appearance of skull vault, separation of cranial sutures in children, and erosion of the dorsum sella.

Enlargement of the pituitary fossa caused by a pituitary tumor, thickening of the skull vault in acromegaly, and abnormal calcification in tuberous sclerosis and Sturge-Weber syndrome can all be detected by skull X-rays. Use of skull X-rays has become limited with use of CT and MRI.

X-rays of the vertebral column are indicated whenever lesions are suspected in the vertebrae, spinal cord, spinal roots, or the adjacent tissues. Routinely, both anteroposterior (AP) and lateral views are obtained. In the cervical and lumbar regions, additional right and left oblique views are required because the intervertebral foramina are seen well only in the oblique pictures.

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While ordering for X-ray of the spine, the particular region of interest should be specified so that this portion can be focused properly in the picture.

Knowledge of the relationship between vertebral bodies and spinal segments helps to specify the vertebrae to be imaged and correlate the neurological defect with the vertebral lesion.

Contrast Radiography

Cerebral Arteriography

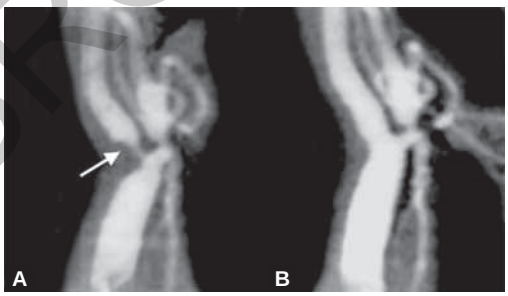
This is the technique by which the cerebral blood vessels are radiologically visualized. Its use has drastically come down with the introduction of CT. However, it is the most important investigation whenever an abnormality in the cerebral vessels is suspected, such as aneurysm, arteriovenous malformation, and arteritis **(Figs. 34.2 and 34.3)**.

It is also indicated in certain brain tumors, such as meningiomas, to assess their vascularity and in patients with TIA due to occlusive carotid artery disease, prior to their surgical management. Cerebral arteriography can be performed either by direct puncturing of the carotid or vertebral arteries in the neck percutaneously or by catheterization of these vessels. In the latter method, which is the preferred one, a catheter is introduced into one of the peripheral arteries, such as the femoral or brachial and it is passed up into the root of the aorta and then selectively into the carotid or vertebral artery on either side. After positioning the catheter tip in the desired vessels, radio-opaque iodinated contrast agent is injected under pressure into the catheter and a rapid series of X-rays is taken while injecting the contrast agent.

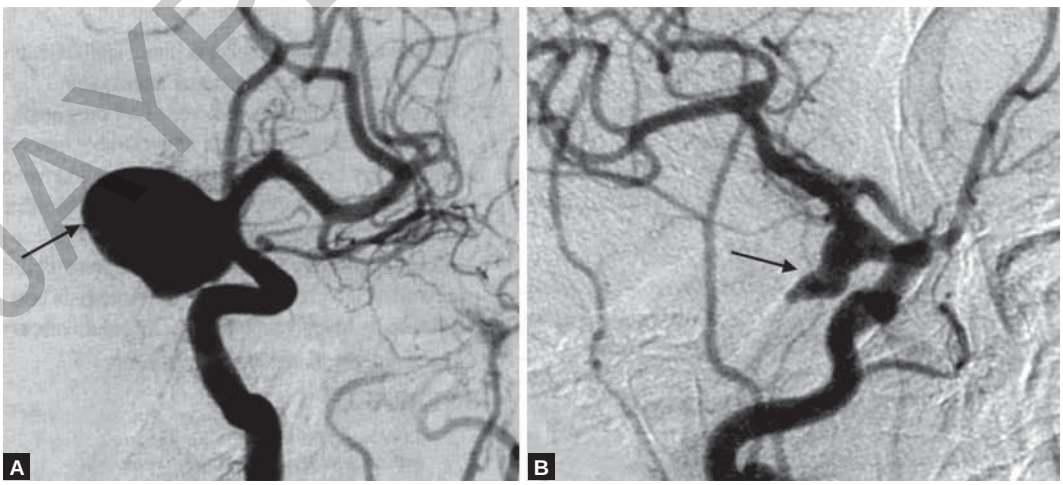
This is time-consuming and invasive procedure with a definite risk of minor and major complications requires hospitalization of the patient.

Digital Subtraction Angiography

Digital subtraction angiography (DSA) is an advancement over the conventional cerebral angiography. In this procedure, the contrast agent is administered intravenously and a series of X-ray images are obtained which are digitized and subjected to computer subtraction for enhanced images of the blood



Figs. 34.2A and B: Internal carotid artery stenosis, arteriogram (A) the narrowing—arrow; (B) after correction.



Figs. 34.3A and B: Carotid angiography. A and B show Berry aneurysm—arrows.

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vessels. This can be done as an outdoor procedure. Although the intravenous injection of contrast permits satisfactory visualization of the bigger vessels in the neck, for visualization of intracranial blood vessels, it is far from satisfactory. For this purpose, selective intraarterial contrast injection is still required.

Ventriculography

This procedure, once employed in the past, mainly for investigating intraventricular and posterior fossa tumors and hydrocephalus, has now been replaced by CT and MRI.

Pneumoencephalography

In pneumoencephalography (PEG) procedure, air is injected into the lumbar subarachnoid space and it is manipulated to enter the cerebral ventricles and subarachnoid space and has become obsolete due to its unawareness and untoward side effects.

Myelography

This procedure is no longer done. Plain X-rays of spine are done after injection of radio opaque dye intrathecally for diagnosis of cord compression. Since the introduction of CT and MRI, myelography is required only for a much smaller number of patients, as greater information can be obtained by combining CT with myelography (CT-myelography).

Complications

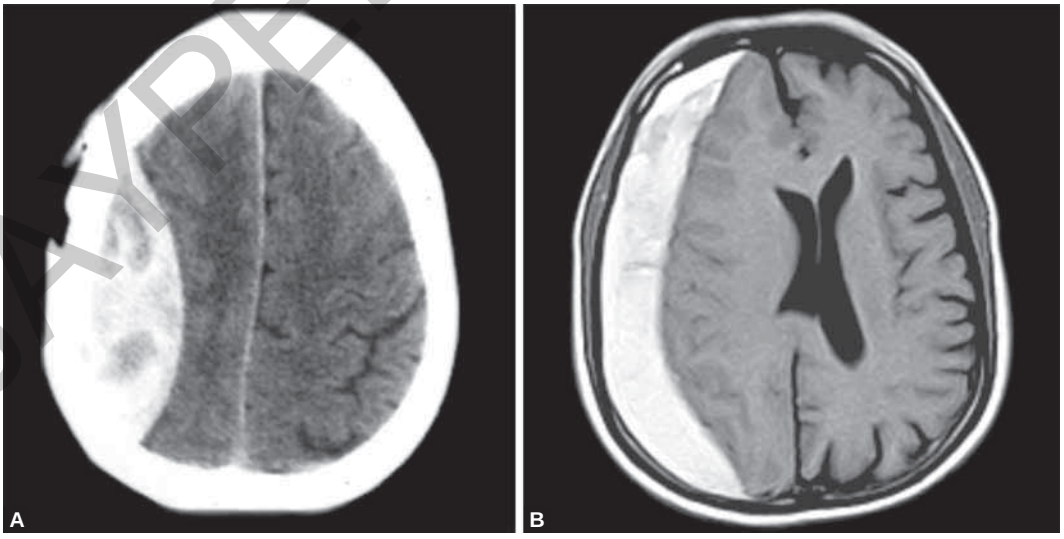
Angiography can produce nausea and vomiting due to reaction to drugs.

Radioiodinated Serum Albumin Cisternography

By injecting 99 technetium-tagged serum albumin into the lumbar subarachnoid space, its flow into the intracranial subarachnoid space and the ventricles is monitored and imaged sequentially over a period of time, to visualize its distribution. Radioiodinated serum albumin (RISA) cisternography was mainly performed in suspected cases of normal pressure hydrocephalus and CSF rhinorrhea. This is also replaced now by CT and MRI.

COMPUTED TOMOGRAPHIC SCAN

In general index include "**Computed Tomographic in Neurological Disorders**". At present, computed tomographic (CT) scan has become a part of routine investigations in the evaluation of many neurological problems. In most of the towns and cities in India, CT scan centers have come up. The cost is relatively high, ranging from ` 1500 to ` 3000 depending upon the part imaged **(Figs. 34.4 to 34.6A)**.



Figs. 34.4A and B: CT scan showing (A) extradural and (B) subdural hematomas. *Note:* The hematoma compressing the brain tissue and leading to pressure effects, i.e., distortion of the ventricles and shift of midline structures.

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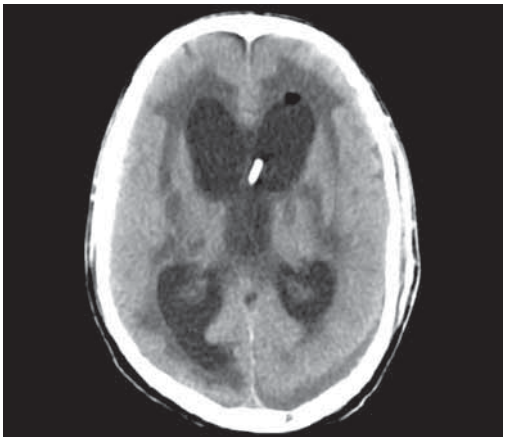

Fig. 34.5: CT scan showing hydrocephalus.
Note: The dilated ventricles and thinned cortex.

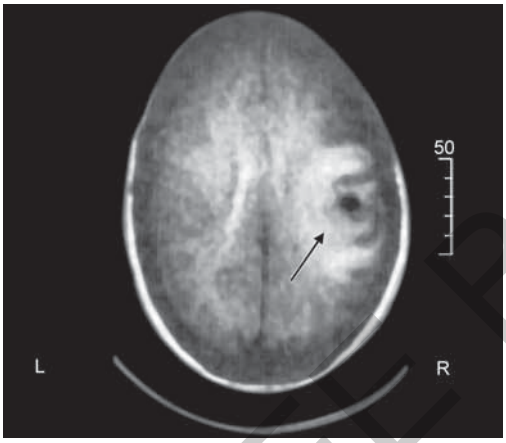

Fig. 34.6A: CT scan. Tuberculoma right parietal lobe (*see* arrow). Male aged 26 years with epilepsy. Completely cleared with therapy.

Indications

The CT scan of the head is indicated and essential for diagnosis and management of the following conditions:

A patient with:

1. Focal neurological deficit of relatively recent onset and of undetermined cause, especially vascular accidents, hemorrhages, thrombosis, or embolism.
2. Features of definite or suspected raised intracranial pressure.
3. Altered sensorium or mental status of undetermined cause.
4. Focal seizures of recent onset.
5. Late onset seizures.
6. Subarachnoid hemorrhage.
7. Head trauma.
8. Suspected intracranial mass lesion.
9. Parasitic diseases of the brain, e.g., cysticercosis, hydatid cyst.

CT scan is also the procedure of choice in patients in whom MRI is contraindicated due to the presence of implanted cardiac pacemakers, mechanical heart valves, or magnetizable intracranial metal clips.

Although a normal CT is sometimes reassuring to the patient as well as to the clinician, the practice of ordering CT scan indiscriminately should be discouraged. Under no circumstances, CT should be considered as a substitute to clinical examination. *CT scanning delivers a high dose of irradiation which should be avoided, if possible.*

Limitations: While CT imaging of the supratentorial structures in the brain is quite good, its use in the evaluation of the posterior fossa structures, such as brainstem and the cerebellum is limited because of the beam-hardening artifacts of adjacent bones. For the same reason, CT for the evaluation of spinal cord diseases is less than satisfactory. The other limitations of CT are its inability to reveal very small lesions, isodense lesions (lesion density same as that of normal brain), and cerebral infarcts in the first 24 to 48 hours of their occurrence. Hence, a normal CT especially if without contrast does not ensure that all possible structural pathologies have been excluded. In such situations, MRI may reveal abnormalities.

Technical modifications in CT with special studies, such as CT angio and perfusion CT are used in the acute management of stroke.

MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) offers better visualization of the anatomy of the brain and the spinal cord because of its high resolution. This is also a safe, rapid, and noninvasive procedure, such as CT, but it is more expensive (` 4000 to ` 6000 per study). The advantages of MRI over CT scan are **(Figs. 34.6B, 34.6C, and 34.7)**:

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