Updates in Clinical Neurology

Professor Leila Rinatovna Akhmadeeva

March 5, 2022

My patient #1

UpToDate®

myasthenia gravis

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Overview of the treatment of myasthenia gravis

Author: Shawn J Bird, MD
Section Editor: Jeremy M Shefner, MD, PhD
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Contributor Disclosures

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Jan 2022.** | This topic last updated: **Mar 29, 2021.**

Commonly used therapies for myasthenia gravis

	Time to onset of effect*	Time to maximal effect*
Symptomatic therapy		
Pyridostigmine	10 to 15 minutes	2 hours
Chronic immunotherapies		
Prednisone	2 to 3 weeks	5 to 6 months
Azathioprine	~12 months	1 to 2 years
Mycophenolate mofetil	6 to 12 months	1 to 2 years
Cyclosporine and tacrolimus	~6 months	~12 months
Rapid immunotherapies		
Plasmapheresis	1 to 7 days	1 to 3 weeks
Intravenous immune globulin	1 to 2 weeks	1 to 3 weeks
Surgery		
Thymectomy	1 to 10 years	1 to 10 years

* Estimated times are rough guidelines based upon clinical experience in myasthenia gravis.

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Drugs that may unmask or worsen myasthenia gravis

Drugs that may unmask or worsen myasthenia gravis
Anesthetic agents
Neuromuscular blocking agents[¶]
Antibiotics
Aminoglycosides[¶] (eg, gentamicin, neomycin, tobramycin)
Fluoroquinolones (eg, ciprofloxacin, levofloxacin, norfloxacin)
Ketolides[◇] (eg, telithromycin)
Macrolides (eg, azithromycin, clarithromycin, erythromycin)
Cardiovascular drugs
Beta blockers (eg, atenolol, labetalol, metoprolol, propranolol)
Procainamide
Quinidine
Other drugs
Anti-PD-1 monoclonal antibodies (eg, nivolumab and pembrolizumab)
Botulinum toxin
Chloroquine
Hydroxychloroquine
Magnesium
Penicillamine
Quinine

Drugs usually well tolerated in myasthenia gravis but occasionally associated with an exacerbation*
Anesthetic agents
Inhalation anesthetics (eg, isoflurane, halothane)
Local anesthetics[Δ] (eg, lidocaine, procaine)
Antibiotics and antiviral agents
Antiretroviral agents (eg, ritonavir)
Clindamycin
Metronidazole
Nitrofurantoin
Tetracyclines (eg, doxycycline, tetracycline)
Vancomycin
Antiseizure medications
Carbamazepine
Ethosuximide
Gabapentin
Phenobarbital
Phenytoin
Antipsychotics and other psychiatric drugs
Butyrophenones (eg, haloperidol)
Lithium
Phenothiazines[§] (eg, chlorpromazine, prochlorperazine)
Glucocorticoids[¥]
Dexamethasone
Methylprednisolone
Prednisone
Ophthalmic drugs
Betaxolol
Echothiophate
Proparacaine
Timolol
Tropicamide
Other drugs
Cisplatinum
Emetine (Ipecac syrup)
Fludarabine
Glatiramer acetate
HMG CoA reductase inhibitors (statins)
Interferon alpha
Interleukin-2
Iodinated contrast agents
Riluzole

PD-1: programmed death receptor-1; HMG CoA: hydroxymethylglutaryl coenzyme A; IVIG: intravenous immune globulin.
* This is not a complete list of all drugs that may, in individual patients, adversely affect neuromuscular transmission. Refer to UpToDate topics for further information.
¶ Only when necessary in hospitalized patients and with caution for respiratory muscle weakness.
Δ When administered intravenously.
◇ **Contraindicated** in myasthenia gravis.
§ Also used as antiemetics.
¥ Although glucocorticoids are a common treatment for myasthenia gravis, at high doses they may cause a significant exacerbation of myasthenia gravis symptoms during early stages of treatment. For this reason, glucocorticoids should be started in high doses only in hospitalized patients who are receiving concurrent plasmapheresis or IVIG for myasthenic crisis.

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eLIBRARY ID: 46511587 DOI: 10.17513/spno.30878

ЭПИДЕМИОЛОГИЧЕСКИЕ АСПЕКТЫ МИАСТЕНИИ В САНКТ-ПЕТЕРБУРГЕ

КРЮЧКОВА В.В. [1], **АЛЕКСЕЕВА Т.Н.** [1], **ЛОБЗИН С.В.** [2], **РУДЕНКО Д.И.** [3]

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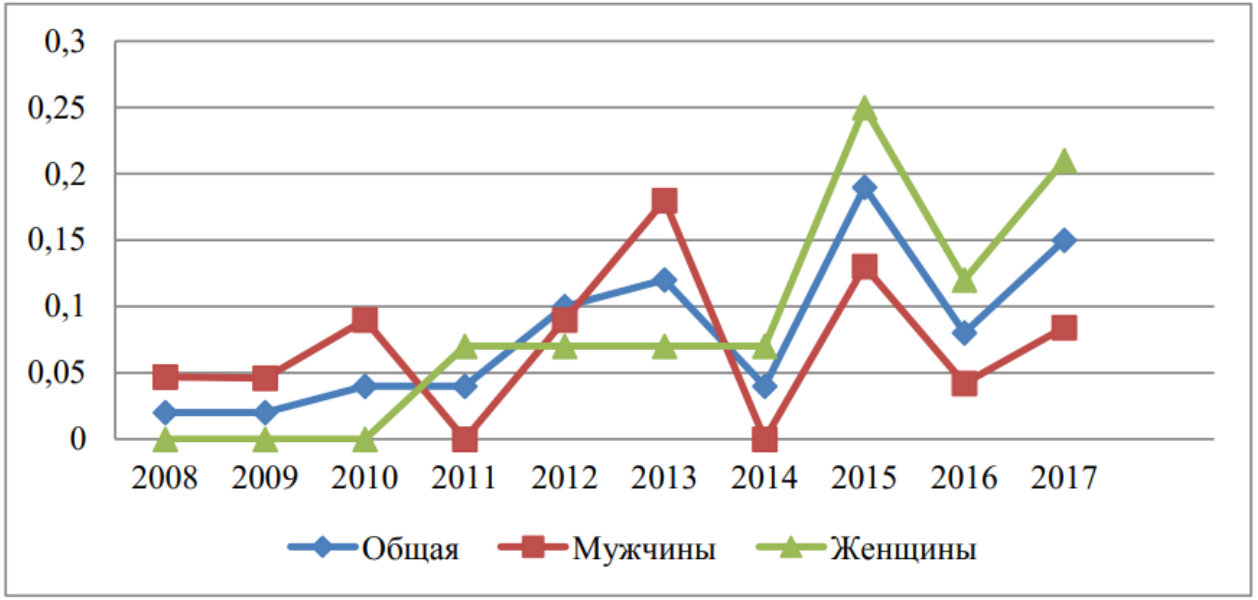

Рис. 3. Смертность среди пациентов с миастенией в Санкт-Петербурге в 2008–2017 гг. на 100 тыс. человек

Patient #2

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mild cognitive impairment treatment

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Mild cognitive impairment: Prognosis and treatment

Author: Ronald C Petersen, MD, PhD
Section Editor: Kristine Yaffe, MD
Deputy Editor: Janet L Wilterdink, MD

Contributor Disclosures

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Nov 2021.** | This topic last updated: **Jul 30, 2021.**

Patient #3

THE CLINICAL NEUROPSYCHOLOGIST, 2016
http://dx.doi.org/10.1080/13854046.2016.1241893

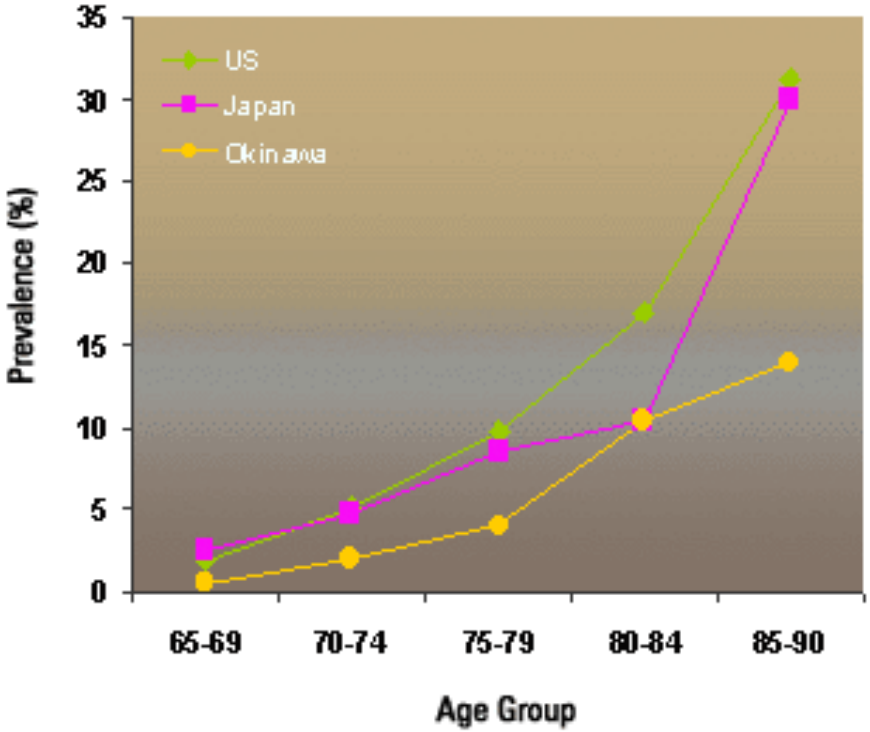
Routledge
Taylor & Francis Group

Rouleau version of the Clock Drawing Test: age- and education-adjusted normative data from a wide Italian sample

Mattia Siciliano[a,b,c], Gabriella Santangelo[a,d], Alfonsina D'Iorio[a], Giuseppe Basile[a], Fausta Piscopo[a], Dario Grossi[a] and Luigi Trojano[a,e]

[a]Department of Psychology, Second University of Naples, Caserta, Italy; [b]Department of Medical, Surgical, Neurological, Metabolic and Aging Sciences, Second University of Naples (SUN), Naples, Italy; [c]Department of Neuroscience, Reproductive and Odontostomatologic Sciences, University "Federico II", Naples, Italy; [d]IDC-Hermitage-Capodimonte, Naples, Italy; [e]Salvatore Maugeri Foundation, Scientific Institute of Telese, Telese Terme, Italy

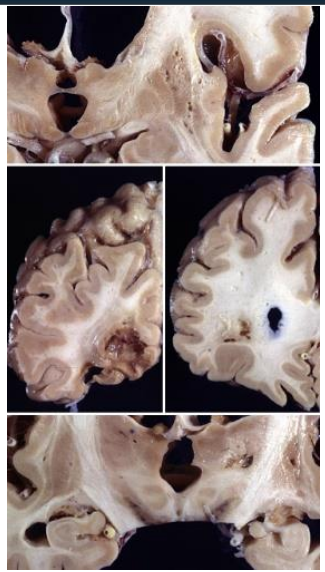
Prevalence of Dementia



Sources: Yamada, M., et al. J Am Geriatr Soc 1999;47:189-95. Kokmen, E., et al. Mayo Clin Proc 1996;71:275-82. Ogura, C., et al. Internatl J Epidemiol 1995;24:373-80.

Dementia – many causes

NEUROLOGY



Practice parameter: Diagnosis of dementia (an evidence-based review): Report of the Quality Standards Subcommittee of the American Academy of Neurology

D. S. Knopman, S. T. DeKosky, J. L. Cummings, H. Chui, J. Corey–Bloom, N. Relkin, G. W. Small, B. Miller and J. C. Stevens

Neurology 2001;56;1143-1153

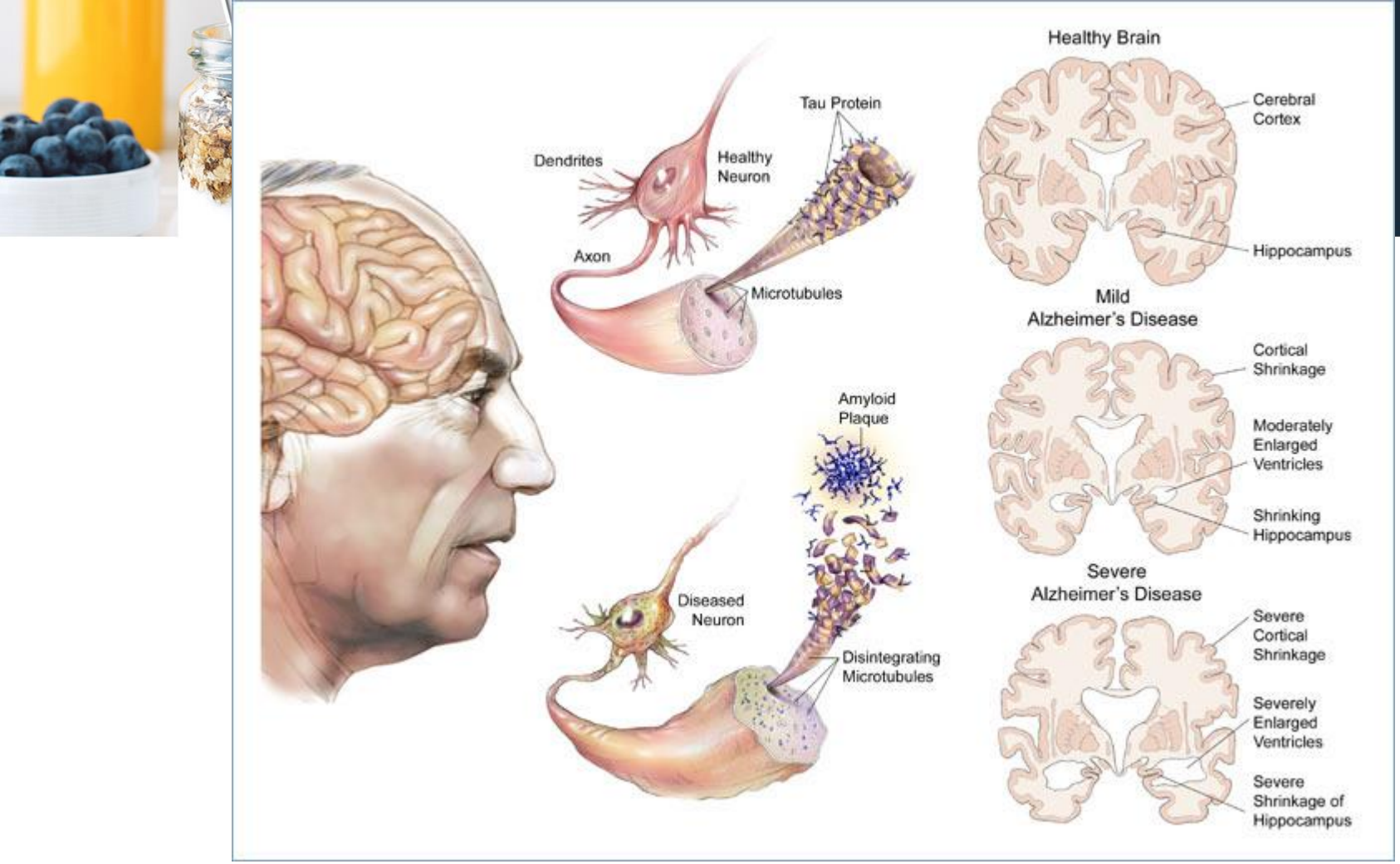
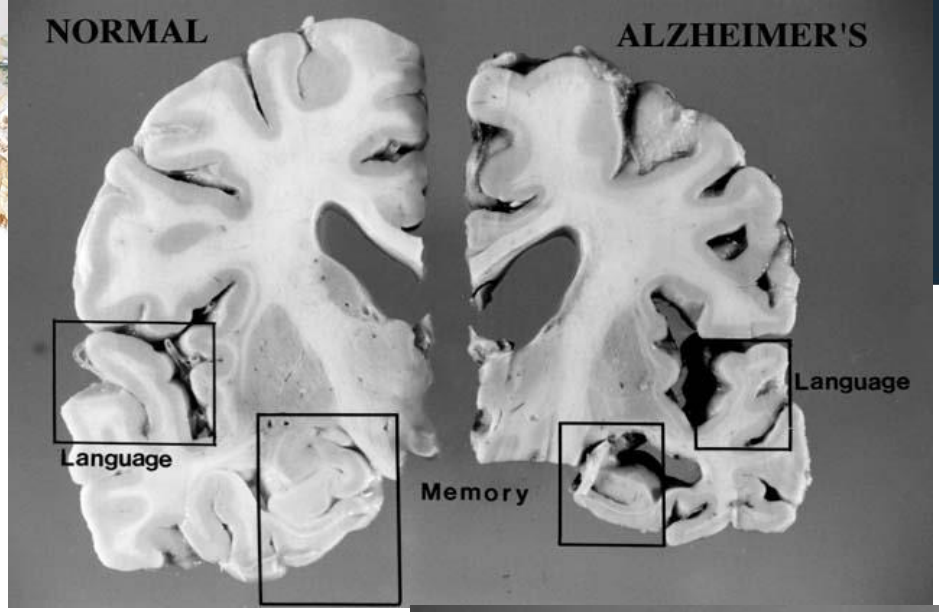
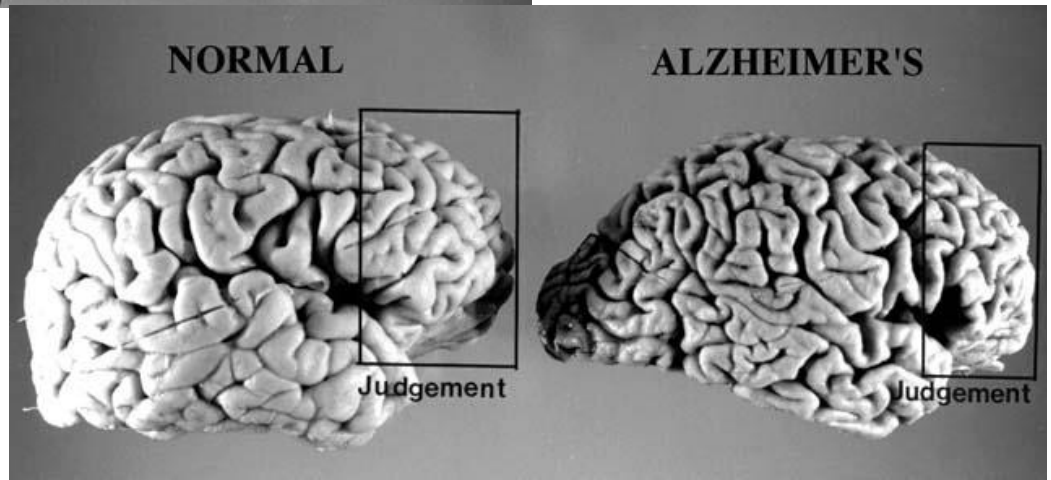


Illustration by Bob Morreale, provided courtesy of the American Health Assistance Foundation

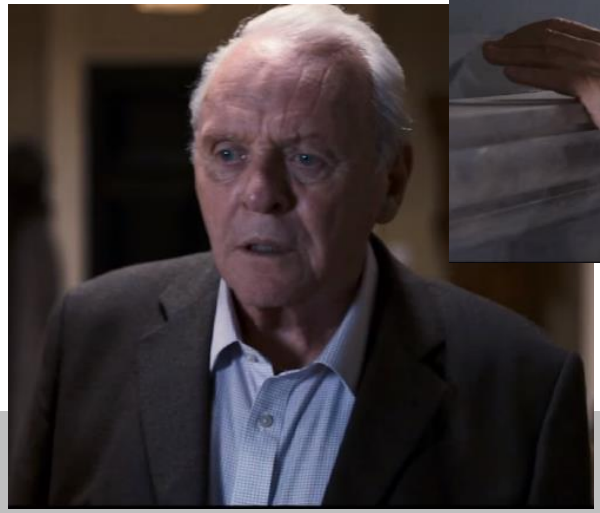


Cerebral atrophy – mostly in hippocampal are in Alzheimer's disease



ICD-11 MMS Chapters v 2020-09

01 Certain infectious or parasitic diseases (1A00-1H0Z)
02 Neoplasms (2A00-2F9Z)
03 Diseases of the blood or blood-forming organs (3A00-3C0Z)
04 Diseases of the immune system (4A00-4B4Z)
05 Endocrine, nutritional or metabolic diseases (5A00-5D46)
06 Mental, behavioural or neurodevelopmental disorders (6A00-6E8Z)
07 Sleep-wake disorders (7A00-7B2Z)
08 Diseases of the nervous system (8A00-8E7Z)
09 Diseases of the visual system (9A00-9E1Z)
10 Diseases of the ear or mastoid process (AA00-AC0Z)
11 Diseases of the circulatory system (BA00-BE2Z)
12 Diseases of the respiratory system (CA00-CB7Z)
13 Diseases of the digestive system (DA00-DE2Z)
14 Diseases of the skin (EA00-EM0Z)
15 Diseases of the musculoskeletal system or connective tissue (FA00-FC0Z)
16 Diseases of the genitourinary system (GA00-GC8Z)
17 Conditions related to sexual health (HA00-HA8Z)
18 Pregnancy, childbirth or the puerperium (JA00-JB6Z)
19 Certain conditions originating in the perinatal period (KA00-KD5Z)
20 Developmental anomalies (LA00-LD9Z)
21 Symptoms, signs or clinical findings, not elsewhere classified (MA00-MH2Y)
22 Injury, poisoning or certain other consequences of external causes (NA00-NF2Z)
23 External causes of morbidity or mortality (PA00-PL2Z)
24 Factors influencing health status or contact with health services (QA00-QF4Z)



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Evaluation of cognitive impairment and dementia

Author: Eric B Larson, MD, MPH
Section Editors: Steven T DeKosky, MD, FAAN, FACP, FANA, Kenneth E Schmader, MD
Deputy Editor: Janet L Wilterdink, MD

Contributor Disclosures

All topics are updated as new evidence becomes available and our peer review process is complete.

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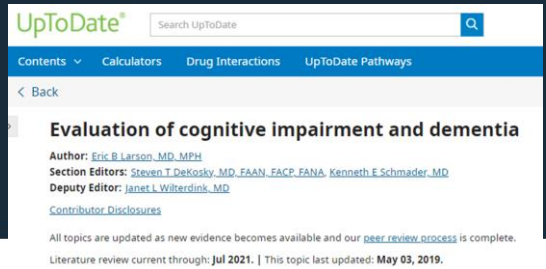
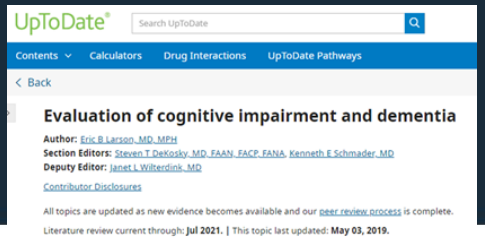


Neuroimaging — Brain imaging, preferably with magnetic resonance imaging (MRI), is indicated in the evaluation of patients with suspected AD [99]. Brain MRI can document potential alternative or additional diagnoses including cerebrovascular disease, other structural diseases (chronic subdural hematoma, cerebral neoplasm, normal pressure hydrocephalus), and regional brain atrophy suggesting frontotemporal dementia (FTD) or other types of neurodegenerative disease. (See "Evaluation of cognitive impairment and dementia", section on 'Neuroimaging'.)

●MRI – Structural MRI findings in AD include both generalized and focal atrophy, as well as white matter lesions. In general, these findings are nonspecific.

The most characteristic focal finding in AD is reduced hippocampal volume or medial temporal lobe atrophy [47,100-103]. Because hippocampal volumes decline in normal aging, however, age-specific criteria are needed [100,101,104]. The finding of hippocampal atrophy provides incremental support for a diagnosis of AD in a patient with a typical clinical presentation, but it is not sufficiently specific to contribute significantly to the accuracy of the diagnosis over the clinical assessment alone [105]. Some studies have suggested that MRI features may predict rate of decline of AD and in the future may guide treatment decisions [85,106]. Hippocampal volumetry using age-corrected norms available from the Alzheimer Disease Neuroimaging Initiative can predict rates of progression of mild cognitive impairment (MCI) to dementia [107]. However, the tools to generate these measurements are not in wide use, nor have these findings been validated in a clinical practice setting.



Diagnosis – AD should be suspected in any older adult with insidious onset, progressive decline in memory, and at least one other cognitive domain leading to impaired functioning. The diagnosis of AD is made in large part by this clinical assessment.

Neuropsychologic testing may provide confirmatory information and aid in patient management. A neuroimaging study should be obtained on every patient suspected of having AD.

In selected cases (eg, those with young age of onset or atypical presentations), other imaging or biomarker tests including 18-F fluorodeoxyglucose positron emission tomography (FDG-PET), cerebrospinal fluid (CSF) testing, or amyloid/tau PET may be helpful, although access and reimbursement for these tests may present challenges. If use of aducanumab is being considered, confirmation of amyloid status is necessary with either amyloid PET or CSF testing.

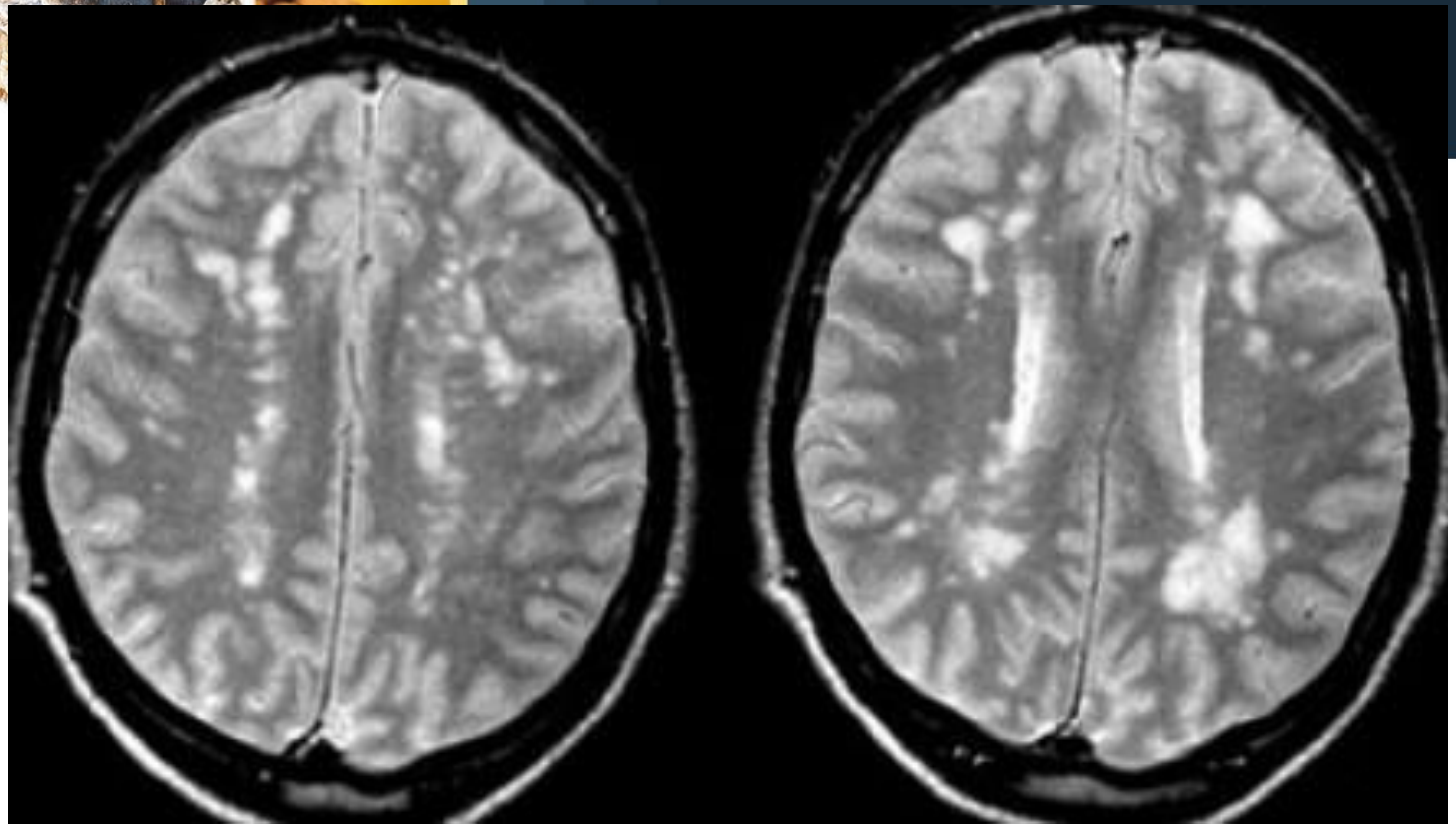
DIFFERENTIAL DIAGNOSIS

The most common disorders considered in the differential diagnosis of AD are vascular dementia and other neurodegenerative dementias. The two most common neurodegenerative dementias after AD are dementia with Lewy bodies (DLB) and frontotemporal dementia (FTD).

●Vascular dementia is caused by either ischemic or hemorrhagic strokes or small vessel cerebrovascular disease. Diagnosis is most specific if there is a stroke-like course of illness, neurologic signs of stroke on examination, and imaging evidence of cerebrovascular disease. However, the course of illness may appear smoothly progressive, and there may be no elementary neurologic signs. Cerebrovascular disease commonly co-occurs with AD. With increasing age, it is more common than not to find both AD and cerebrovascular disease in the brain of a patient with dementia.

●DLB may be the second most common type of degenerative dementia after AD. Clinical features that help distinguish this from AD include prominent early appearance of visual hallucinations, along with parkinsonism, cognitive fluctuations, dysautonomia, rapid eye movement (REM) sleep behavior disorder, and neuroleptic sensitivity.

●FTD is a neuropathologically and clinically heterogeneous disorder characterized by focal degeneration of the frontal and/or temporal lobes. Early alteration of personality, social and emotional behavior, and executive functioning are prominent clinical characteristics of behavioral variant FTD. Primary progressive aphasia (PPA) is a form of FTD in which gradually progressive language impairment is the core feature early in the course. There are three major subtypes, with the semantic and nonfluent variants being associated usually with frontotemporal lobar degeneration (FTLD) pathologies (usually tau or TDP-43). PPA, particularly the logopenic variant, can also be a presentation of AD.



https://www.arthriticchick.com/diagnoses/non-autoimmune-diseases/brain-disorders/chronic-small-vessel-disease-of-the-brain

Patient #4

Metabolic myopathies

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metabolic myopathy

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Approach to the metabolic myopathies

Author: Basil T Darras, MD
Section Editor: Marc C Patterson, MD, FRACP
Deputy Editor: John F Dashe, MD, PhD

Contributor Disclosures

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EMG
Muscle pathology
Muscle MRI
CK level (in blood)

Correlation of metabolic myopathy symptoms and signs with specific biochemical defects

Static symptoms and signs
Acid maltase deficiency
Branching enzyme deficiency
Debranching enzyme deficiency
Carnitine transport defect
LCAD, VLCAD deficiencies
Trifunctional enzyme deficiency
Mitochondrial disorders
Dynamic symptoms and signs
Phosphorylase b kinase deficiency
Myophosphorylase (PPL) deficiency
Phosphofructokinase (PFK) deficiency
Phosphoglycerate kinase (PGK) deficiency
Lactate dehydrogenase (LDH) deficiency
Carnitine palmitoyltransferase II (CPT II) deficiency
Fatty acid oxidation/mitochondrial defects
Static and dynamic symptoms and signs
Myophosphorylase deficiency
PFK, PPL b kinase deficiencies (plus fixed weakness)
Debranching enzyme deficiency (plus dynamic symptoms)
LCAD, VLCAD, SCHAD deficiencies
Trifunctional enzyme deficiency
Multiple mitochondrial DNA deletions

LCAD: long-chain acyl-CoA dehydrogenase; VLCAD: very long-chain acyl-CoA dehydrogenase; SCHAD: short-chain 3-hydroxyacyl-CoA dehydrogenase.

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Patient #5

Nonmotor symptoms of Parkinson disease

Cognitive dysfunction
Psychosis
Mood disorders (depression, anxiety, apathy/abulia)
Sleep disturbances
Fatigue
Autonomic dysfunction (urinary urgency/frequency, constipation, orthostasis, erectile dysfunction)
Olfactory dysfunction
Pain and sensory disturbances
Dermatologic findings (seborrhea)

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Motor features of Parkinson disease

Cardinal manifestations
Tremor
Bradykinesia
Rigidity
Postural instability
Other motor features
Craniofacial
Hypomimia (masked facial expression)
Decreased eye blinking
Speech disturbances (hypokinetic dysarthria, hypophonia)
Dysphagia
Sialorrhea
Visual
Blurred vision
Impaired contrast sensitivity
Hypometric saccades
Impaired vestibuloocular reflex
Impaired upward gaze and convergence
Lid apraxia
Musculoskeletal
Micrographia
Dystonia
Myoclonus
Stooped posture
Camptocormia (severe anterior flexion of the thoracolumbar spine)
Pisa syndrome (subacute axial dystonia with lateral flexion of the trunk, head, and neck)
Kyphosis
Scoliosis
Difficulty turning in bed
Gait
Shuffling, short-stepped gait
Freezing
Festination

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palliative parkinson

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Palliative approach to Parkinson disease and parkinsonian disorders

Authors: David Oliver, BSc, FRCP, FRCGP, Simone Veronese, MD, MSc, PhD
Section Editors: R Sean Morrison, MD, Howard I Hurtig, MD
Deputy Editor: April F Eichler, MD, MPH

Contributor Disclosures

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ADVANCE CARE PLANNING

Goshen Health

It starts with a conversation.

DISCUSS
Begin the conversation

DECIDE
Create a plan

DIRECT
Document your choices

Examples of medications that may cause or exacerbate orthostatic hypotension

Drug group	Mechanism of hypotension and comments
Diuretics ▪ Loop diuretics (eg, furosemide, torsemide) or thiazides	Extracellular fluid volume depletion.
Adrenergic antagonists	
▪ Alpha-1-adrenergic blockers (eg, alfuzosin, tamsulosin, terazosin)	Alpha-1-adrenergic blockers produce vasodilation via direct effect in vascular smooth muscle.
▪ Beta-adrenergic blockers (eg, propranolol)	Beta-adrenergic blockers reduce cardiac output and renin release. May also reduce vascular peripheral resistance.
Alpha-2-adrenergic agonists (eg, tizanidine, clonidine)	Vasodilation via central inhibition of sympathetic efferent activity.
Nitric oxide-mediated vasodilators ▪ Nitroglycerin, hydralazine ▪ Phosphodiesterase-5-inhibitors (eg, sildenafil)	Vasodilation via direct effect in vascular smooth muscle.
Renin-angiotensin system (RAS) inhibitors (eg, lisinopril, valsartan)	Vasodilation via RAS inhibition.
Calcium-channel blockers (eg, verapamil, diltiazem)	Reduction of cardiac output, vasodilation via direct effect in vascular smooth muscle.
Dopamine antagonists ▪ Phenothiazines (eg, chlorpromazine) ▪ Atypical antipsychotics (eg, olanzapine, risperidone, quetiapine)	Vasodilation via central inhibition of sympathetic efferent activity.
Antidepressants (eg, trazodone, amitriptyline)	Vasodilation via central and peripheral inhibition of sympathetic efferent activity through stimulation of adrenergic receptors.
Selective serotonin receptor reuptake inhibitors (eg, paroxetine)	Unknown mechanism, possibly via central and peripheral inhibition of sympathetic efferent activity through stimulation of alpha-2-adrenergic receptors.

Patient #6

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Prevention of cardiovascular disease events in those with established disease (secondary prevention) or at very high risk

Authors: Charles H Hennekens, MD, DrPH, Jose Lopez-Sendon, MD, PhD
Section Editors: Joann G Elmore, MD, MPH, Christopher P Cannon, MD, Juan Carlos Kaski, DSc, MD, DM (Hons), FRCP, FESC, FACC, FAHA
Deputy Editors: Jane Givens, MD, MSCE, Nisha Parikh, MD, MPH

Contributor Disclosures

All topics are updated as new evidence becomes available and our peer review process is complete.

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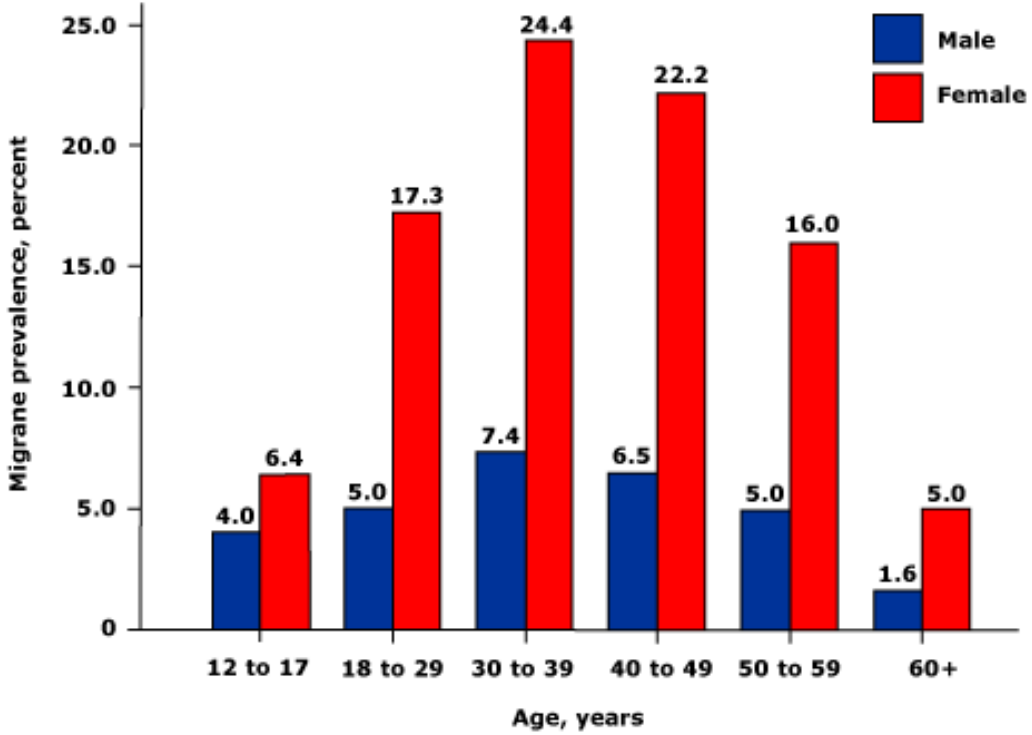
This topic is a broad overview of our approach to the prevention of CVD events in those with established CVD or at very high risk.

Patient #7

Migraine prevalence



Data from: Lipton, RB, Bigal, ME, Diamond, M, et al. Migraine prevalence, disease burden, and the need for preventative therapy. Neurology 2007; 68:343.

› Expert Rev Neurother. 2020 Apr;20(4):313-317. doi: 10.1080/14737175.2020.1730816. Epub 2020 Feb 18.

Migraine, low-dose combined hormonal contraceptives, and ischemic stroke in young women: a systematic review and suggestions for future research

Raffaele Ornello [1], Marianne Canonico [2], Gabriele S Merki-Feld [3], Tobias Kurth [4], Øjvind Lidegaard [5], E Anne MacGregor [6 7], Christian Lampl [8 9], Rossella Elena Nappi [10 11], Paolo Martelletti [12], Simona Sacco [1]

Affiliations + expand

PMID: 32056462 DOI: 10.1080/14737175.2020.1730816

› Am J Obstet Gynecol. 2017 May;216(5):489.e1-489.e7. doi: 10.1016/j.ajog.2016.12.019. Epub 2016 Dec 26.

Use of combined hormonal contraceptives among women with migraines and risk of ischemic stroke

Steven W Champaloux [1], Naomi K Tepper [2], Michael Monsour [1], Kathryn M Curtis [1], Maura K Whiteman [1], Polly A Marchbanks [1], Denise J Jamieson [1]

Affiliations + expand

PMID: 28034652 DOI: 10.1016/j.ajog.2016.12.019

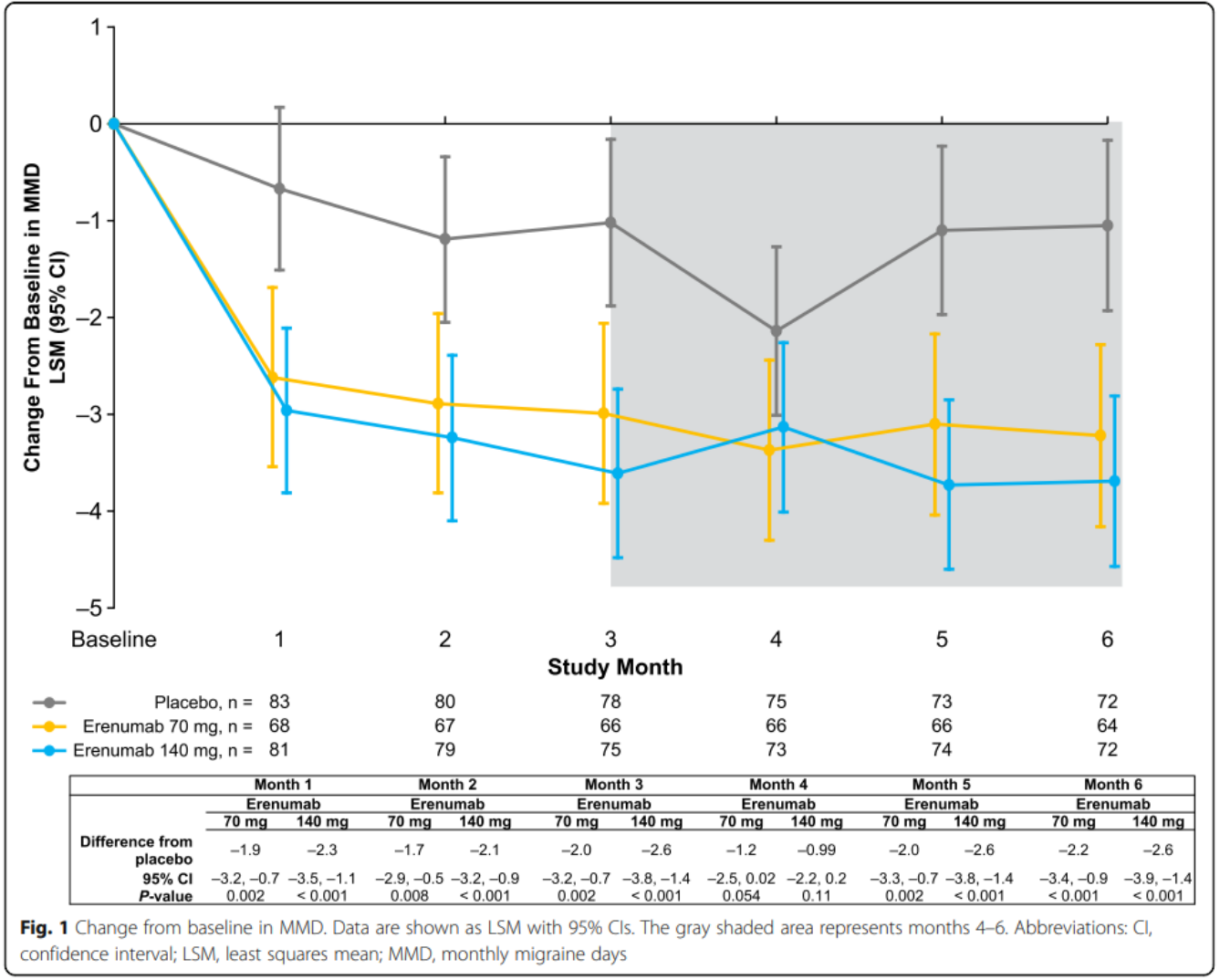
Review › Contraception. 2016 Dec;94(6):630-640. doi: 10.1016/j.contraception.2016.04.016. Epub 2016 May 3.

Safety of hormonal contraceptives among women with migraine: A systematic review

Naomi K Tepper [1], Maura K Whiteman [2], Lauren B Zapata [2], Polly A Marchbanks [2], Kathryn M Curtis [2]

Affiliations + expand

PMID: 27153744 DOI: 10.1016/j.contraception.2016.04.016



	Month 1		Month 2		Month 3		Month 4		Month 5		Month 6	
	Erenumab		Erenumab		Erenumab		Erenumab		Erenumab		Erenumab	
	70 mg	140 mg	70 mg	140 mg	70 mg	140 mg	70 mg	140 mg	70 mg	140 mg	70 mg	140 mg
Difference from placebo	−1.9	−2.3	−1.7	−2.1	−2.0	−2.6	−1.2	−0.99	−2.0	−2.6	−2.2	−2.6
95% CI	−3.2, −0.7	−3.5, −1.1	−2.9, −0.5	−3.2, −0.9	−3.2, −0.7	−3.8, −1.4	−2.5, 0.02	−2.2, 0.2	−3.3, −0.7	−3.8, −1.4	−3.4, −0.9	−3.9, −1.4
P-value	0.002	< 0.001	0.008	< 0.001	0.002	< 0.001	0.054	0.11	0.002	< 0.001	< 0.001	< 0.001

Fig. 1 Change from baseline in MMD. Data are shown as LSM with 95% CIs. The gray shaded area represents months 4–6. Abbreviations: CI, confidence interval; LSM, least squares mean; MMD, monthly migraine days


Pavlovic et al. The Journal of Headache and Pain (2020) 21:95
https://doi.org/10.1186/s10194-020-01167-6
The Journal of Headache and Pain
RESEARCH ARTICLE Open Access

Efficacy and safety of erenumab in women with a history of menstrual migraine

Jelena M. Pavlovic[1,2*], Koen Paemeleire[3], Hartmut Göbel[4], Jo Bonner[5], Alan Rapoport[6], Risa Kagan[7,8], Feng Zhang[9], Hernan Picard[9] and Daniel D. Mikol[9]

who.int/news/item/11-02-2022-who-s-new-international-classification-of-diseases-(icd-11)-comes-into-effect

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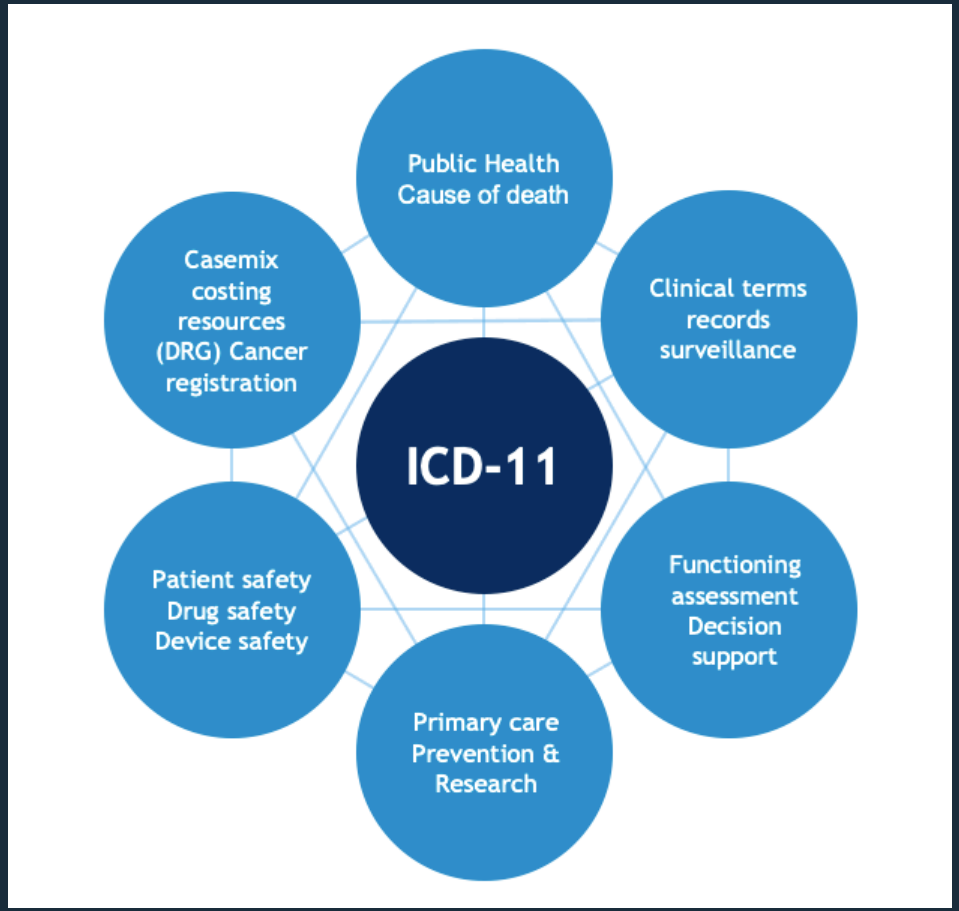
WHO's new International Classification of Diseases (ICD-11) comes into effect

Feb 11, 2022

https://www.who.int/news/item/11-02-2022-icd-11-2022-release



icd.who.int

EN

ICD-11

International Classification of Diseases 11th Revision

The global standard for diagnostic health information

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https://www.who.int/news/item/11-02-2022-who-s-new-international-classification-of-diseases-(icd-11)-comes-into-effect

- **ICD-11 highlights**
- Legally mandated health data standard*
- Comparable statistics with semantic interoperability
- Conceptual framework for all languages and cultures
- Integration of terminology and classification
- Up-to-date clinical and scientific knowledge
- End-to-end digital solution
- Freely available through open license

ICD-11 MMS Chapters v 2020-09

01 Certain infectious or parasitic diseases (1A00-1H0Z)
02 Neoplasms (2A00-2F9Z)
03 Diseases of the blood or blood-forming organs (3A00-3C0Z)
04 Diseases of the immune system (4A00-4B4Z)
05 Endocrine, nutritional or metabolic diseases (5A00-5D46)
06 Mental, behavioural or neurodevelopmental disorders (6A00-6E8Z)
07 Sleep-wake disorders (7A00-7B2Z)
08 Diseases of the nervous system (8A00-8E7Z)
09 Diseases of the visual system (9A00-9E1Z)
10 Diseases of the ear or mastoid process (AA00-AC0Z)
11 Diseases of the circulatory system (BA00-BE2Z)
12 Diseases of the respiratory system (CA00-CB7Z)
13 Diseases of the digestive system (DA00-DE2Z)
14 Diseases of the skin (EA00-EM0Z)
15 Diseases of the musculoskeletal system or connective tissue (FA00-FC0Z)
16 Diseases of the genitourinary system (GA00-GC8Z)
17 Conditions related to sexual health (HA00-HA8Z)
18 Pregnancy, childbirth or the puerperium (JA00-JB6Z)
19 Certain conditions originating in the perinatal period (KA00-KD5Z)
20 Developmental anomalies (LA00-LD9Z)
21 Symptoms, signs or clinical findings, not elsewhere classified (MA00-MH2Y)
22 Injury, poisoning or certain other consequences of external causes (NA00-NF2Z)
23 External causes of morbidity or mortality (PA00-PL2Z)
24 Factors influencing health status or contact with health services (QA00-QE4Z)

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Research Paper

PAIN®

VIDEO

Comparing the ICD-11 chronic pain classification with ICD-10: how can the new coding system make chronic pain visible? A study in a tertiary care pain clinic setting

Nantthasorn Zinboonyahgoon[a], Choopong Luansritisakul[a,*], Sarasate Eiamtanasate[a], Sirikan Duangburong[a], Virachat Sanansilp[a], Beatrice Korwisi[b], Antonia Barke[c], Winfried Rief[b], Rolf-Detlef Treede[d]

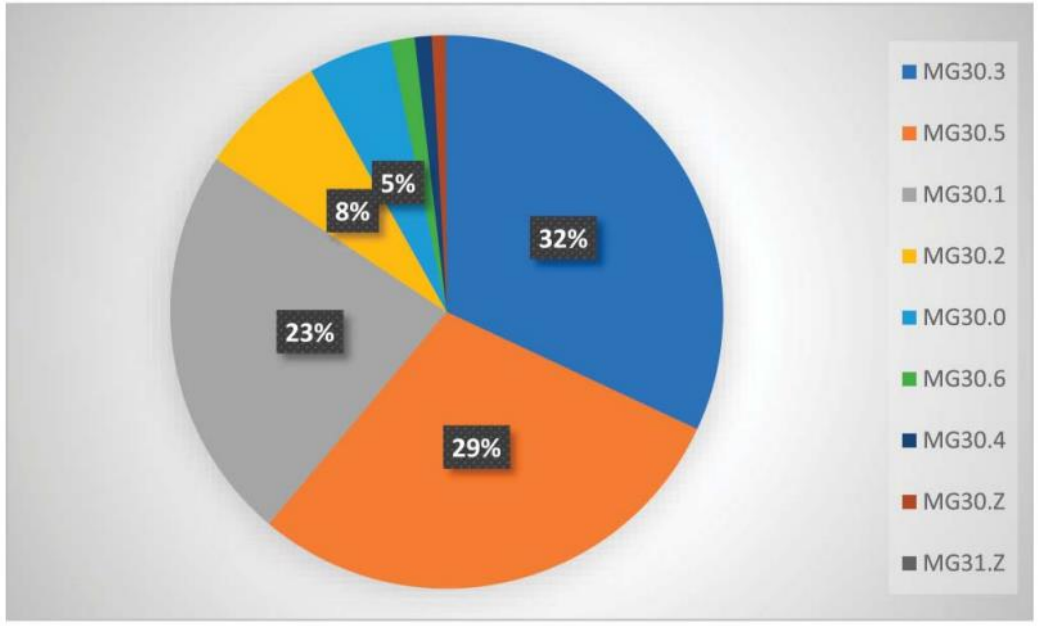


Figure 2. The percentage of top 10 codes for the ICD-11 group at the level of detail recommended for primary care. This pie chart shows percentages of each coding group of ICD-11 at the level of detail recommended for primary care.[19] MG30.3: chronic secondary musculoskeletal pain, MG30.5: chronic neuropathic pain, MG30.1: chronic cancer-related pain, MG30.2: chronic postsurgical or posttraumatic pain, MG30.0: chronic primary pain, MG30.6: chronic secondary headache or orofacial pain, MG30.4: chronic secondary visceral pain, MG30.Z: chronic pain, unspecified, and MG31.Z: acute pain, unspecified.

MG30 Chronic pain

International Classification of Diseases for Mortality and Morbidity Statistics, 11th Revision, v2020-09

Pain is an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage. Chronic pain is pain that persists or recurs for longer than 3 months. Chronic pain is multifactorial: biological, psychological and social factors contribute to the pain syndrome.

exclusions

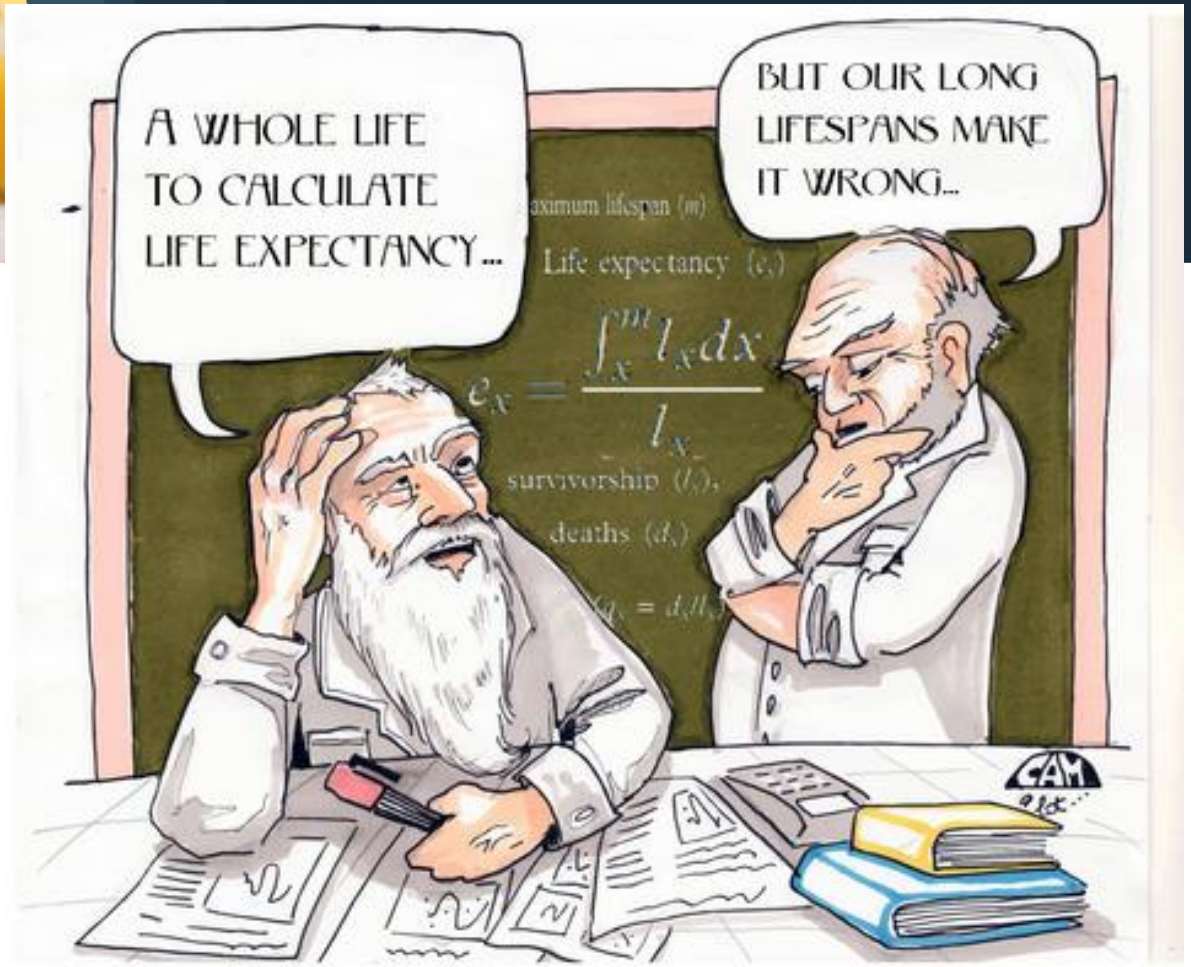
- Acute pain (MG31)

sections/codes in this section (MG30-MG30)

- Chronic primary pain (MG30.0)
- Chronic cancer related pain (MG30.1)
- Chronic postsurgical or post traumatic pain (MG30.2)
- Chronic secondary musculoskeletal pain (MG30.3)
- Chronic secondary visceral pain (MG30.4)
- Chronic neuropathic pain (MG30.5)
- Chronic secondary headache or orofacial pain (MG30.6)
- Other specified chronic pain (MG30.Y)
- Chronic pain, unspecified (MG30.Z)

coding note

This code should be used if a pain condition persists or recurs for longer than 3 months.


A WHOLE LIFE TO CALCULATE LIFE EXPECTANCY...
BUT OUR LONG LIFESPANS MAKE IT WRONG...
aximum lifespan (m)
Life expectancy (e_x)
$e_x = \frac{\int_x^m l_x dx}{l_x}$
survivorship (l_x)
deaths (d_x)
$q_x = d_x/l_x$
CAM