CENTERS FOR DISEASE™
CONTROL AND PREVENTION

ICD-10 Coordination and Maintenance Committee Meeting
March 17-18, 2026
ICD-10-CM Diagnosis Agenda

This is a virtual meeting. Registration is required at: https://cms.zoomgov.com/webinar/register/WN_LdBl5sC-T0mxdg_Lm6O6jg

After registering, you will receive a confirmation email containing information about joining the webinar.

Welcome and announcements
Capt. Monica Leonard
Co-Chair, ICD-10 Coordination and Maintenance Committee

Diagnosis Topics:

Contents

March 17, 2026, ICD-10 C & M Diagnosis

Order of presentation Day 1

*Please dial in 45 mins before the topic of interest in the event that the meeting is ahead of schedule, presentation times are subject to change

Topic*	Presenter
Opening Remarks, CMS	Mady Hue
Welcome, CDC, Diagnosis Day 1 **Time: 9:05-910amET**	Capt. Monica Leonard
Topic(s): Organizing Principles for Classification of Ultra-rare and Genetic Conditions **Time: 9:10 – 9:25amET**	Mary Stanfill
Topic: Cardiogenic Shock Staging **Time: 9:25-9:40amET**	David Berglund Sarah Adie
Topic: Carotid Web **Time: 9:40-9:55amET**	David Berglund Diogo Haussen
Topic: Carrier of Candidiasis **Time: 9:55-10:10amET**	Traci Ramirez
Topic: WPW Syndrome **Time: 10:10-10:25amET**	David Berglund Sandra O'Loughlin
Topic: Progressive Myopia **Time: 10:25-10:40amET**	Shannon McConnell-Lamptey Michael Repka
Topic: Macular Telangiectasia **Time: 10:40-10:55amET**	Shannon McConnell-Lamptey Michael Repka
Topic: Metabolic Dysfunction-and Alcohol Associated Liver Disease (MetALD) **Time: 10:55-11:10amET**	Shannon McConnell-Lamptey
Topic: Congenital Hyperinsulinism **Time: 11:10-11:25amET**	Cheryl Bullock Diva D. De Leon-Crutchlow
Topic: Facial Angiofibroma **Time: 11:25-11:40amET**	Cheryl Bullock Michael Pham

March 17, 2026, ICD-10 C & M Diagnosis Timed Agenda (Day 1)

Topic*	Presenter
Topic: Spontaneous Coronary Artery Dissection (SCAD) **Time: 11:40-11:55amET**	David Berglund
Topic: Sepsis **Time: 11:55-12:10pmET**	David Berglund
Topic: Gender Identity Disorder **Time: 12:10-12:25pmET**	Cheryl Bullock Kurt Miceli
LUNCH BREAK	12:30-1:30
Topic: Biomarkers for Alzheimer's **Time: 1:30-1:45pmET**	Cheryl Bullock
Topic: Dysphotopsia **Time: 1:45-2:00pmET**	Shannon McConnell-Lamptey Tawnya Pastuck
Topic: Postprocedural Open Deep Wound Without Disruption **Time: 2:00-2:15pmET**	Shannon McConnell-Lamptey
Topic: Corneal Pseudomicrocysts **Time: 2:15-2:30pmET**	Desiree Abrams Neel Pasricha
Topic: Anatomical Specificity for eyelid Disorders **Time: 2:30-2:45pmET**	Desiree Abrams
Topic: Encounter for Observation for Suspected Conditions:EEG monitoring **Time: 2:45-3:00pmET**	Cheryl Bullock
Topic: Neonatal Supraventricular Tachycardia **Time: 3:00-3:15pmET**	Cheryl Bullock
Topic: Medetomidine Withdrawal Syndrome **Time: 3:15-3:30pmET**	Shannon McConnell-Lamptey

March 17, 2026, ICD-10 C & M Diagnosis Timed Agenda (Day 1)

Topic*	Presenter
Topic: Toxic Effect of Hexane **Time: 3:30-3:45pmET**	Desiree Abrams Yvonne Martinez
Topic: Toxic Effect of Iron Oxide **Time: 3:45-4:00pmET**	Desiree Abrams Laree LaPierre
Topic: Toxic Effect of N-Butyl Acetate **Time: 4:00-4:15pmET**	Desiree Abrams Laree LaPierre
Topic: Titanium Dioxide Exposure **Time: 4:15-4:30pmET**	Desiree Abrams Yvonne Martinez
Topic: Pneumothorax that occurs after CPR **Time: 4:30-4:45pmET**	Cheryl Bullock
Topic: Addenda **Time**: **4:45-5:00pmET**	Traci Ramirez

March 18, 2026, ICD-10 C & M Diagnosis

Order of presentation Day 2

*Please dial in 45 mins before the topic of interest in the event that the meeting is ahead of schedule, presentation times are subject to change

Topic*	Presenter
Welcome, CDC, Diagnosis Day 2 **Time: 9:00-9:10amET**	Capt. Monica Leonard
Topic: Adverse Effects of COVID-19 Vaccines **Time: 9:10-9:25amET**	Mary Stanfill
Topic(s): Controlled Obesity **Time: 9:25-9:40amET**	David Berglund Angela Fitch
Topic: Chronic Hand Eczema **Time: 9:40-9:55amET**	Shannon McConnell-Lamptey
Topic: Lipedema Lipedema and Lipolymphedema **Time: 9:55-10:10amET**	Shannon McConnell-Lamptey
Topic: Hypertriglyceridemia **Time: 10:10-10:25amET**	Shannon McConnell-Lamptey
Topic: Floppy Eyelid Syndrome **Time: 10:25-10:40amET**	Desiree Abrams Anne Barmettler
Topic: Oral Epithelial Dysphasia **Time: 10:40-10:55amET**	Desiree Abrams Hussam Batal
Topic: Post Intensive Care Syndrome (PICS) **Time: 10:55-11:10amET**	Desiree Abrams Brian Peach

ICD-10 TIMELINE

A timeline of important dates in the ICD-10 process is described below:

March 17-18, 2026	The diagnosis code portion of the March 2026 ICD-10 Coordination and Maintenance Committee Meeting will be fully virtual by zoom and dial-in. Those who wish to attend must participate via Zoom Webinar or by dialing in. The procedure code topics will be open for public comment.
March 2026	Recordings and slide presentations of the diagnosis code topics and procedure code topic materials of the March 17-18, 2026 ICD-10 Coordination and Maintenance Committee Meeting will be posted on the following web pages: **Diagnosis code portion of the recording and related materials–** https://www.cdc.gov/nchs/icd/icd-10-maintenance/meetings.html **Procedure code portion of the materials–** https://www.cms.gov/medicare/coding-billing/icd-10-codes/icd-10-coordination-maintenance-committee-materials
April 17, 2026	**Deadline for receipt of public comments on proposed new codes and revisions presented at the March 17-18, 2026 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on October 1, 2026**.
April 2026	Notice of Proposed Rulemaking to be published in the Federal Register as mandated by the Omnibus Budget Reconciliation Act of 1986, Public Law 99-509 (Pub. L. 99-509). This notice will include references to the FY 2027 ICD-10-CM diagnosis and ICD-10-PCS procedure codes finalized to date. It will also include proposed revisions to the MS-DRG system based on ICD-10-CM/PCS codes on which the public may comment. The proposed rule can be accessed at: https://www.cms.gov/medicare/payment/prospective-payment-systems/acute-inpatient-pps

May 15, 2026	**Deadline for receipt of public comments on proposed new codes and revisions presented at the March 17-18, 2026 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on April 1, 2027.** **Deadline for receipt of public comments on proposed new diagnosis codes and revisions presented at the March 17-18, 2026 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on October 1, 2027**.
May/June 2026	Final addenda posted on web pages as follows: **Diagnosis addendum -** https://www.cdc.gov/nchs/icd/icd-10-cm/files.html **Procedure addendum -** https://www.cms.gov/medicare/coding-billing/icd-10-codes
June 5, 2026	**Deadline for requestors: Those members of the public requesting that topics be considered for the September 15-16, 2026 ICD-10 Coordination and Maintenance Committee Meeting must have their requests submitted to CMS for procedures and NCHS for diagnoses.** **Note: NCHS is actively working to develop organizing principles to guide classification of ultra-rare and genetic conditions in ICD-10-CM. During this time, NCHS is not accepting new proposals for ultra-rare or genetic conditions. We anticipate resuming new proposal submissions on these topics, in conformance with newly established principles, for the March 2027 C&M Meeting.** **Procedure code requests should be directed to CMS at:** https://mearis.cms.gov **Diagnosis code requests should be directed to NCHS at:** nchsicd10cm@cdc.gov Requestors should indicate if they are submitting their code request for consideration for an April 1, 2027 implementation date or an October 1, 2027 implementation date. **The ICD-10 Coordination and Maintenance Committee will make efforts to accommodate the requested implementation date for each request submitted, however, the Committee will determine which requests will be presented for consideration for an April 1, 2027 implementation date or an October 1, 2027 implementation date.**

July 2026	Federal Register notice for the September 15-16, 2026 ICD-10 Coordination and Maintenance Committee Meeting will be published. This will include the tentative agenda.
August 1, 2026	Hospital Inpatient Prospective Payment System final rule expected to be published in the Federal Register as mandated by Pub. L. 99-509. This rule will also include links to all the final codes to be implemented on October 1, 2026. This rule can be accessed at: https://www.cms.gov/medicare/payment/prospective-payment-systems/acute-inpatient-pps
August 2026	Tentative agenda for the procedure portion of the September 15, 2026 ICD-10 Coordination and Maintenance Committee Meeting will be posted on the CMS webpage at – https://www.cms.gov/medicare/coding-billing/icd-10-codes/icd-10-coordination-maintenance-committee-materials Tentative agenda for the diagnosis portion of the September 16, 2026 ICD-10 Coordination and Maintenance Committee Meeting will be posted on the NCHS webpage at – https://www.cdc.gov/nchs/icd/icd-10-maintenance/meetings.html
September 15-16, 2026	The September 2026 ICD-10 Coordination and Maintenance Committee Meeting is anticipated to be fully virtual by Teams and dial-in. Those who wish to attend must participate via Teams Webinar or by dialing in.
September 2026	Recordings and slide presentations of the September 15-16, 2026 ICD-10 Coordination and Maintenance Committee Meeting will be posted on the following web pages: **Diagnosis code portion of the recording and related materials–** https://www.cdc.gov/nchs/icd/icd-10-maintenance/meetings.html **Procedure code portion of the recording and related materials–** https://www.cms.gov/medicare/coding-billing/icd-10-codes/icd-10-coordination-maintenance-committee-materials
October 1, 2026	New and revised ICD-10-CM and ICD-10-PCS codes go into effect along with MS-DRG changes. Final addendum available on web pages as follows: **Diagnosis addendum –** https://www.cdc.gov/nchs/icd/icd-10-cm/files.html

	Procedure addendum – https://www.cms.gov/medicare/coding-billing/icd-10-codes
October 16, 2026	**Deadline for receipt of public comments on proposed new codes discussed at the September 15-16, 2026 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on April 1, 2027.**
November 2026	Any new ICD-10 codes that will be implemented on the following April 1 will be announced. Information on any new codes to be implemented April 1, 2027 will be posted on the following websites: https://www.cdc.gov/nchs/icd/icd-10-cm/files.html https://www.cms.gov/medicare/coding-billing/icd-10-codes
November 13, 2026	**Deadline for receipt of public comments on proposed new codes and revisions presented at the September 15-16, 2026 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on October 1, 2027.**
December 4, 2026	**Deadline for requestors: Those members of the public requesting that topics be considered for the March 16-17, 2027 ICD-10 Coordination and Maintenance Committee Meeting must have their requests submitted to CMS for procedures and NCHS for diagnoses.** **Procedure code requests should be directed to CMS at:** https://mearis.cms.gov **Diagnosis code requests should be directed to NCHS at:** nchsicd10cm@cdc.gov Requestors should indicate if they are submitting their code request for consideration for an October 1, 2027 implementation date, an April 1, 2028 implementation date, or an October 1, 2028 implementation date. **The ICD-10 Coordination and Maintenance Committee may not be able to consider all requests received for the next Committee code update and will determine if it would be appropriate to postpone consideration of any code requests to a future time. The Committee will make efforts to accommodate**

the requested implementation date for each request submitted, however, the Committee will determine which requests will be presented for consideration at the March 16-17, 2027 meeting for an October 1, 2027 implementation date, an April 1, 2028 implementation date, or an October 1, 2028 implementation date.

Contact Information
Mailing address:

National Center for Health Statistics
ICD-9-CM Coordination and Maintenance Committee
3311 Toledo Road
Hyattsville, Maryland 20782

Comments on the ICD-10-CM diagnosis proposals presented at the ICD Coordination and Maintenance Committee meeting should be sent to the following email address: nchsicd10CM@cdc.gov

Captain Monica Leonard	(404) 718-6443
Desiree Abrams	(301) 458-4384
David Berglund, MD	(301) 458-4095
Cheryl Bullock	(301) 458-4297
Shannon McConnell-Lamptey	(301) 458-4612
Traci Ramirez	(301) 458-4454

Continuing Education Credits

Continuing education credits may be awarded by the American Academy of Professional Coders (AAPC) or the American Health Information Management Association (AHIMA) for participation in CMS/NCHS ICD-10 Coordination and Maintenance (C&M) Committee Meeting.

Continuing Education Information for American Academy of Professional Coders (AAPC)

If you plan to attend or participate via telephone the ICD-10 Coordination and Maintenance (C&M) Committee Meeting, you should be aware that CMS /NCHS do not provide certificates of attendance for these calls. Instead, the AAPC will accept your printed topic packet as proof of participation. Please retain your topic packet copy as the AAPC may request them for any conference call you entered into your CEU Tracker if you are chosen for CEU verification. Members are awarded one (1) CEU per hour of participation.

Continuing Education Information for American Health Information Management Association (AHIMA)

AHIMA credential-holders may claim 1 CEU per 60 minutes of attendance at an educational program. Maintain documentation about the program for verification purposes in the event of an audit. A program does not need to be pre-approved by AHIMA, nor does a CEU certificate need to be provided, in order to claim AHIMA CEU credit. For detailed information about AHIMA's CEU requirements, see the Recertification Guide on AHIMA's web site.

Please note: The statements above are standard language provided to NCHS by the AAPC and the AHIMA. If you have any questions concerning either statement, please contact the respective organization, not NCHS.

Adverse Effect of COVID-19 Vaccines

While ICD-10-CM currently includes codes for post-COVID-19 conditions, there are no specific codes to reflect adverse health conditions due to COVID-19 vaccinations. COVID-19 vaccines and their side effects, are a focus of ongoing research and therapeutic studies.[1-6]

The World Health Organization (WHO) ICD-10 includes code U12.9, COVID-19 vaccines causing adverse effects in therapeutic use, unspecified. This WHO ICD-10 code is an external cause code, as it is considered a subcategory under external cause category Y59, Other and unspecified vaccines and biological substances. This code was not adopted in the ICD-10-CM because ICD-10-CM does not include the WHO ICD-10 external cause Y59 code category. In ICD-10-CM, vaccines causing adverse effects are classified in the injury chapter. Presently, an adverse effect of a COVID-19 vaccine would be assigned code T50.B95-, Adverse effect of other viral vaccines (with the appropriate 7th character for the encounter), following the appropriate code(s) for the nature of the adverse effect, as specified in the official coding guidelines for adverse effects.

React19 requests a code to uniquely identify adverse effects of COVID vaccines from other viral vaccines. In response, NCHS presents proposed modifications to the ICD-10-CM code set. The proposed unique code for adverse effects of COVID-19 vaccines will enable clinicians, researchers, and public health professionals to identify, track, and study these cases.

References

1. Lesgards JF, Cerdan D, Perronne C. Do Long COVID and COVID Vaccine Side Effects Share Pathophysiological Picture and Biochemical Pathways? Int J Mol Sci. 2025 Aug 15;26(16):7879. doi: 10.3390/ijms26167879. Erratum in: Int J Mol Sci. 2025 Sep 29;26(19):9513. doi: 10.3390/ijms26199513. PMID: 40869200; PMCID: PMC12386580
2. Patterson BK, Yogendra R, Francisco EB, Guevara-Coto J, Long E, Pise A, Osgood E, Bream J, Kreimer M, Jeffers D, Beaty C, Vander Heide R, Mora-Rodríguez RA. Detection of S1 spike protein in CD16+ monocytes up to 245 days in SARS-CoV-2-negative post-COVID-19 vaccine syndrome (PCVS) individuals. Hum Vaccin Immunother. 2025 Dec;21(1):2494934. doi: 10.1080/21645515.2025.2494934. Epub 2025 May 13. PMID: 40358138; PMCID: PMC12077440.
3. Scholkmann F, May CA. COVID-19, post-acute COVID-19 syndrome (PACS, "long COVID") and post-COVID-19 vaccination syndrome (PCVS, "post-COVIDvac-syndrome"): Similarities and differences. Pathol Res Pract. 2023 Jun;246:154497. doi: 10.1016/j.prp.2023.154497. Epub 2023 May 3. PMID: 37192595; PMCID: PMC10154064.
4. Yonker LM, Swank Z, Bartsch YC, Burns MD, Kane A, Boribong BP, Davis JP, Loiselle M, Novak T, Senussi Y, Cheng CA, Burgess E, Edlow AG, Chou J, Dionne A, Balaguru D, Lahoud-Rahme M, Arditi M, Julg B, Randolph AG, Alter G, Fasano A, Walt DR. Circulating Spike Protein Detected in Post-COVID-19 mRNA Vaccine Myocarditis. Circulation. 2023 Mar 14;147(11):867-876. doi: 10.1161/CIRCULATIONAHA.122.061025. Epub 2023 Jan 4. PMID: 36597886; PMCID: PMC10010667.
5. Chen Y, Xu Z, Wang P, Li XM, Shuai ZW, Ye DQ, Pan HF. New-onset autoimmune phenomena post-COVID-19 vaccination. Immunology. 2022 Apr;165(4):386-401. doi: 10.1111/imm.13443. Epub 2022 Jan 7. PMID: 34957554.
6. Cancarevic I, Nassar M, Medina L, Sanchez A, Parikh A, Hosna A, Devanabanda B, Vest M, Ayotunde F, Ghallab M, Omran I. Nephrotic Syndrome in Adult Patients With COVID-19 Infection or Post COVID-19 Vaccine: A Systematic Review. Cureus. 2022 Sep 26;14(9):e29613. doi: 10.7759/cureus.29613. PMID: 36312654; PMCID: PMC9595350

TABULAR MODIFICATIONS

T50 Poisoning by, adverse effect of and underdosing of diuretics and other and unspecified drugs, medicaments and biological substances

The appropriate 7th character is to be added to each code from category T50.

A initial encounter
D subsequent encounter
S sequela

T50.B Poisoning by, adverse effect of and underdosing of viral vaccines

New sub-subcategory

T50.B2 Poisoning by, adverse effect of and underdosing of COVID-19 vaccines

New code

T50.B25 Adverse effect of COVID-19 vaccines

Anatomical Specificity for Eyelid Disorders

The American Optometric Association (AOA) requests additional ICD-10-CM codes to more clearly and efficiently specify when a condition is present in the bilateral upper and/or lower lid, simplifying and enhancing the precision of coding for eye health and vision conditions.

The AOA is requesting additional ICD-10-CM codes for the following conditions: Hordeolum externum and Hordeolum internum, Chalazion, Allergic dermatitis, Discoid lupus erythematosus, Eczematous dermatitis, , Entropion and trichiasis of eyelid, Cicatricial entropion of eyelid, Mechanical entropion of eyelid, Senile entropion of eyelid, Spastic entropion of eyelid, Trichiasis without entropion, Ectropion of eyelid (Unspecified ectropion of eyelid), Cicatricial ectropion of eyelid, Mechanical ectropion of eyelid, Senile ectropion of eyelid, Spastic ectropion of eyelid, Paralytic ectropion of eyelid, Dermatochalasis of eyelid, Edema of eyelid, Elephantiasis of eyelid and Meibomian gland dysfunction of eyelid.

The proposal has been reviewed and supported by the American Academy of Ophthalmology.

TABULAR MODIFACTIONS

H00 Hordeolum and chalazion
H00.0 Hordeolum (externum) (internum) of eyelid
H00.01 Hordeolum externum
H00.011 Hordeolum externum right upper eyelid
H00.012 Hordeolum externum right lower eyelid
H00.013 Hordeolum externum right eye, unspecified eyelid
H00.014 Hordeolum externum left upper eyelid
H00.015 Hordeolum externum left lower eyelid
H00.016 Hordeolum externum left eye, unspecified eyelid
H00.019 Hordeolum externum unspecified eye, unspecified eyelid
New Code H00.01A Hordeolum externum right eye, upper and lower eyelids
New Code H00.01B Hordeolum externum left eye, upper and lower eyelids

H00.02 Hordeolum internum
Infection of meibomian gland
H00.021 Hordeolum internum right upper eyelid
H00.022 Hordeolum internum right lower eyelid
H00.023 Hordeolum internum right eye, unspecified eyelid
H00.024 Hordeolum internum left upper eyelid
H00.025 Hordeolum internum left lower eyelid
H00.026 Hordeolum internum left eye, unspecified eyelid

H00.029 Hordeolum internum unspecified eye, unspecified eyelid
New code H00.02A Hordeolum internum right upper and lower eyelids
New code H00.02B Hordeolum internum left upper and lower eyelids

H00.1 Chalazion
H00.11 Chalazion right upper eyelid
H00.12 Chalazion right lower eyelid
H00.13 Chalazion right eye, unspecified eyelid
H00.14 Chalazion left upper eyelid
H00.15 Chalazion left lower eyelid
H00.16 Chalazion left eye, unspecified eyelid
H00.19 Chalazion unspecified eye, unspecified eyelid
New code H00.1A Chalazion right upper and lower eyelids
New code H00.1B Chalazion left upper and lower eyelids

H01.1 Noninfectious dermatoses of eyelid
H01.11 Allergic dermatitis of eyelid
H01.111 Allergic dermatitis of right upper eyelid
H01.112 Allergic dermatitis of right lower eyelid
H01.113 Allergic dermatitis of right eye, unspecified eyelid
H01.114 Allergic dermatitis of left upper eyelid
H01.115 Allergic dermatitis of left lower eyelid
H01.116 Allergic dermatitis of left eye, unspecified eyelid
H01.119 Allergic dermatitis of unspecified eye, unspecified eyelid
New code H01.11A Allergic dermatitis of right eye, upper and lower eyelids
New code H01.11B Allergic dermatitis of left eye, upper and lower eyelids

H01.12 Discoid lupus erythematosus of eyelid
H01.121 Discoid lupus erythematosus of right upper eyelid
H01.122 Discoid lupus erythematosus of right lower eyelid
H01.123 Discoid lupus erythematosus of right eye, unspecified eyelid
H01.124 Discoid lupus erythematosus of left upper eyelid
H01.125 Discoid lupus erythematosus of left lower eyelid
H01.126 Discoid lupus erythematosus of left eye, unspecified eyelid
H01.129 Discoid lupus erythematosus of unspecified eye, unspecified eyelid

New code H01.12A Discoid lupus erythematosus of right eye, upper and lower eyelids
New code H01.12B Discoid lupus erythematosus of left eye, upper and lower eyelids

H01.13 Eczematous dermatitis of eyelid
H01.131 Eczematous dermatitis of right upper eyelid
H01.132 Eczematous dermatitis of right lower eyelid
H01.133 Eczematous dermatitis of right eye, unspecified eyelid
H01.134 Eczematous dermatitis of left upper eyelid
H01.135 Eczematous dermatitis of left lower eyelid
H01.136 Eczematous dermatitis of left eye, unspecified eyelid
H01.139 Eczematous dermatitis of unspecified eye, unspecified eyelid
New code H01.13A Eczematous dermatitis of right eye, upper and lower eyelids
New code H01.13B Eczematous dermatitis of left eye, upper and lower eyelids

H02 Other disorders of eyelid
H02.0 Entropion and trichiasis of eyelid
H02.00 Unspecified entropion of eyelid
H02.001 Unspecified entropion of right upper eyelid
H02.002 Unspecified entropion of right lower eyelid
H02.003 Unspecified entropion of right eye, unspecified eyelid
H02.004 Unspecified entropion of left upper eyelid
H02.005 Unspecified entropion of left lower eyelid
H02.006 Unspecified entropion of left eye, unspecified eyelid
H02.009 Unspecified entropion of unspecified eye, unspecified eyelid
New code H02.00A Unspecified entropion of right eye, upper and lower eyelids
New code H02.00B Unspecified entropion of left eye, upper and lower eyelids

H02.01 Cicatricial entropion of eyelid
H02.011 Cicatricial entropion of right upper eyelid
H02.012 Cicatricial entropion of right lower eyelid
H02.013 Cicatricial entropion of right eye, unspecified eyelid
H02.014 Cicatricial entropion of left upper eyelid
H02.015 Cicatricial entropion of left lower eyelid
H02.016 Cicatricial entropion of left eye, unspecified eyelid
H02.019 Cicatricial entropion of unspecified eye, unspecified eyelid

New code H02.01A Cicatricial entropion of right eye, upper and lower eyelids
New code H02.01B Cicatricial entropion of left eye, upper and lower eyelids

H02.02 Mechanical entropion of eyelid
H02.021 Mechanical entropion of right upper eyelid
H02.022 Mechanical entropion of right lower eyelid
H02.023 Mechanical entropion of right eye, unspecified eyelid
H02.024 Mechanical entropion of left upper eyelid
H02.025 Mechanical entropion of left lower eyelid
H02.026 Mechanical entropion of left eye, unspecified eyelid
H02.029 Mechanical entropion of unspecified eye, unspecified eyelid
New code H02.02A Mechanical entropion of right eye, upper and lower eyelids
New code H02.02B Mechanical entropion of left eye, upper and lower eyelids

H02.03 Senile entropion of eyelid
H02.031 Senile entropion of right upper eyelid
H02.032 Senile entropion of right lower eyelid
H02.033 Senile entropion of right eye, unspecified eyelid
H02.034 Senile entropion of left upper eyelid
H02.035 Senile entropion of left lower eyelid
H02.036 Senile entropion of left eye, unspecified eyelid
H02.039 Senile entropion of unspecified eye, unspecified eyelid
New code H02.03A Senile entropion of right eye, upper and lower eyelids
New code H02.03B Senile entropion of left eye, upper and lower eyelids

H02.04 Spastic entropion of eyelid
H02.041 Spastic entropion of right upper eyelid
H02.042 Spastic entropion of right lower eyelid
H02.043 Spastic entropion of right eye, unspecified eyelid
H02.044 Spastic entropion of left upper eyelid
H02.045 Spastic entropion of left lower eyelid
H02.046 Spastic entropion of left eye, unspecified eyelid
H02.049 Spastic entropion of unspecified eye, unspecified eyelid
New code H02.04A Spastic entropion of right eye, upper and lower eyelids
New code H02.04B Spastic entropion of left eye, upper and lower eyelids

H02.05 Trichiasis without entropion
H02.051 Trichiasis without entropion right upper eyelid
H02.052 Trichiasis without entropion right lower eyelid

H02.053 Trichiasis without entropion right eye, unspecified eyelid
H02.054 Trichiasis without entropion left upper eyelid
H02.055 Trichiasis without entropion left lower eyelid
H02.056 Trichiasis without entropion left eye, unspecified eyelid
H02.059 Trichiasis without entropion unspecified eye, unspecified eyelid
New code H02.05A Trichiasis without entropion right eye, upper and lower eyelids
New code H02.05B Trichiasis without entropion left eye, upper and lower eyelids

H02.1 Ectropion of eyelid
H02.10 Unspecified ectropion of eyelid
H02.101 Unspecified ectropion of right upper eyelid
H02.102 Unspecified ectropion of right lower eyelid
H02.103 Unspecified ectropion of right eye, unspecified eyelid
H02.104 Unspecified ectropion of left upper eyelid
H02.105 Unspecified ectropion of left lower eyelid
H02.106 Unspecified ectropion of left eye, unspecified eyelid
H02.109 Unspecified ectropion of unspecified eye, unspecified eyelid
New code H02.1A Unspecified ectropion of right eye, upper and lower eyelids
New code H02.1B Unspecified ectropion of left eye, upper and lower eyelids

H02.11 Cicatricial ectropion of eyelid
H02.111 Cicatricial ectropion of right upper eyelid
H02.112 Cicatricial ectropion of right lower eyelid
H02.113 Cicatricial ectropion of right eye, unspecified eyelid
H02.114 Cicatricial ectropion of left upper eyelid
H02.115 Cicatricial ectropion of left lower eyelid
H02.116 Cicatricial ectropion of left eye, unspecified eyelid
H02.119 Cicatricial ectropion of unspecified eye, unspecified eyelid
New code H02.11A Cicatricial ectropion of right eye, upper and lower eyelids
New code H02.11B Cicatricial ectropion of left eye, upper and lower eyelids

H02.12 Mechanical ectropion of eyelid
H02.121 Mechanical ectropion of right upper eyelid
H02.122 Mechanical ectropion of right lower eyelid
H02.123 Mechanical ectropion of right eye, unspecified eyelid
H02.124 Mechanical ectropion of left upper eyelid
H02.125 Mechanical ectropion of left lower eyelid
H02.126 Mechanical ectropion of left eye, unspecified eyelid
H02.129 Mechanical ectropion of unspecified eye, unspecified eyelid

New code H02.12A Mechanical ectropion of right eye, upper and lower eyelids
New code H02.12B Mechanical ectropion of left eye, upper and lower eyelids

H02.13 Senile ectropion of eyelid
H02.131 Senile ectropion of right upper eyelid
H02.132 Senile ectropion of right lower eyelid
H02.133 Senile ectropion of right eye, unspecified eyelid
H02.134 Senile ectropion of left upper eyelid
H02.135 Senile ectropion of left lower eyelid
H02.136 Senile ectropion of left eye, unspecified eyelid
H02.139 Senile ectropion of unspecified eye, unspecified eyelid
New code H02.13A Senile ectropion of right eye, upper and lower eyelids
New code H02.13B Senile ectropion of left eye, upper and lower eyelids

H02.14 Spastic ectropion of eyelid
H02.141 Spastic ectropion of right upper eyelid
H02.142 Spastic ectropion of right lower eyelid
H02.143 Spastic ectropion of right eye, unspecified eyelid
H02.144 Spastic ectropion of left upper eyelid
H02.145 Spastic ectropion of left lower eyelid
H02.146 Spastic ectropion of left eye, unspecified eyelid
H02.149 Spastic ectropion of unspecified eye, unspecified eyelid
New code H02.14A Spastic ectropion right eye, upper and lower eyelids
New code H02.14B Spastic ectropion left eye, upper and lower eyelids

H02.15 Paralytic ectropion of eyelid
H02.151 Paralytic ectropion of right upper eyelid
H02.152 Paralytic ectropion of right lower eyelid
H02.153 Paralytic ectropion of right eye, unspecified eyelid
H02.154 Paralytic ectropion of left upper eyelid
H02.155 Paralytic ectropion of left lower eyelid
H02.156 Paralytic ectropion of left eye, unspecified eyelid
H02.159 Paralytic ectropion of unspecified eye, unspecified eyelid
New code H02.15A Paralytic ectropion of right eye, upper and lower eyelids
New code H02.15B Paralytic ectropion of left eye, upper and lower eyelids

H02.8 Other specified disorders of eyelid
H02.83 Dermatochalasis of eyelid
H02.831 Dermatochalasis of right upper eyelid
H02.832 Dermatochalasis of right lower eyelid
H02.833 Dermatochalasis of right eye, unspecified eyelid
H02.834 Dermatochalasis of left upper eyelid
H02.835 Dermatochalasis of left lower eyelid
H02.836 Dermatochalasis of left eye, unspecified eyelid
H02.839 Dermatochalasis of unspecified eye, unspecified eyelid
New code H02.83A Dermatochalasis of right eye, upper and lower eyelids
New code H02.83B Dermatochalasis of left eye, upper and lower eyelids

H02.84 Edema of eyelid
Hyperemia of eyelid
H02.841 Edema of right upper eyelid
H02.842 Edema of right lower eyelid
H02.843 Edema of right eye, unspecified eyelid
H02.844 Edema of left upper eyelid
H02.845 Edema of left lower eyelid
H02.846 Edema of left eye, unspecified eyelid
H02.849 Edema of unspecified eye, unspecified eyelid
New code H02.84A Edema of right eye, upper and lower eyelids
New code H02.84B Edema of left eye, upper and lower eyelids

H02.85 Elephantiasis of eyelid
H02.851 Elephantiasis of right upper eyelid
H02.852 Elephantiasis of right lower eyelid
H02.853 Elephantiasis of right eye, unspecified eyelid
H02.854 Elephantiasis of left upper eyelid
H02.855 Elephantiasis of left lower eyelid
H02.856 Elephantiasis of left eye, unspecified eyelid
H02.859 Elephantiasis of unspecified eye, unspecified eyelid
New code H02.85A Elephantiasis of right eye, upper and lower eyelids
New code H02.85B Elephantiasis of left eye, upper and lower eyelids

Biomarkers for Alzheimer's Disease

Biomarkers are biological indicators that can be identified and measured in blood, other body fluids, and tissues. They can be linked to increased risk for certain diagnoses or used to make or confirm a specific diagnosis. Once a diagnosis is established, biomarkers can also be used to assess disease progression and to inform clinical decision-making.

This proposal was presented at the September 2025 ICD-10 Coordination and Maintenance Meeting. In response to public comments, changes have been made and noted in **bold.**

Biomarkers play a major role in detecting underlying pathology of Alzheimer's disease. Amyloid plaques and tau tangles in the brain are key pathological features of Alzheimer's disease that disrupt normal function of neurons. Amyloid plaques are abnormally folded beta-amyloid Aβ peptides that clump together in the brain. Aβ peptides are derived from amyloid precursor protein, which performs various neuronal development, signaling and stability functions [1].
Phosphorylated tau (p-tau) occurs when phosphate molecules bind to tau, a structural protein, and can lead to misfolded hyperphosphorylated tau tangles in the brain. Alzheimer's disease pathology may be detected up to two decades before the emergence of clinical symptoms, such as cognitive decline [2].

In the past, amyloid plaques and tau tangles could be identified only through post-mortem brain autopsy. Later, it was found that they could be reliably detected and measured using PET scans with specific radiotracers. Abnormal levels of Aβ peptides and tau in cerebrospinal fluid and blood correlate with amyloid plaques and tau tangles in the brain [3]. These can be detected through cerebrospinal fluid obtained via lumbar puncture or, more recently, with blood- based biomarker tests. In May 2025, the FDA cleared the first blood-based biomarker test for the detection of Alzheimer's pathology in individuals with cognitive decline [4].

While there is robust clinical discussion over whether biomarkers represent an early stage of Alzheimer's disease or a risk factor for the disease, there is consensus that they are present - and potentially clinically significant - even before onset of symptoms [3, 5-6]. As individuals move either toward ruling out or establishing a diagnosis, the presence of biomarkers provides a way to clearly describe the rationale for continued evaluation.

In the absence of clinical symptoms, the presence of biomarkers also impacts management of individuals, including addressing modifiable risk factors and determining appropriate follow-up. Lifestyle modifications may reduce or delay cognitive decline from Alzheimer's disease [7].
Clinical studies are also underway to identify drugs and other interventions in cognitively intact but biomarker-positive individuals to slow or prevent progression to symptomatic Alzheimer's disease [8-9].

After reviewing laboratory or imaging findings of Alzheimer's pathology, physicians may document the presence of biomarkers using phases such as "biomarker evidence of amyloid pathology in the brain" or "elevated p-tau in CSF concerning for Alzheimer's disease." At present, there are no specific codes for Alzheimer's biomarkers.

Given the clinical significance of biomarkers, they should be distinctly identified in the data to reflect accurate rationale for continuing clinical evaluations and work-up, identify possible participants in future clinical trials for asymptomatic patients with abnormal biomarkers, avoid misuse of diagnosis codes for Alzheimer's disease, and track evolving management.

This proposal has been submitted by The Global CEO Initiative on Alzheimer's Disease (CEOi), which is a convened consortium of the non-profit patient advocacy organization UsAgainstAlzheimer's. Founded in 2013, CEO brings together private-sector leaders and other collaborators to provide leadership in the fight against Alzheimer's.

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TABULAR MODIFICATIONS

R77 Other abnormalities of plasma proteins
Excludes1:disorders of plasma-protein metabolism (E88.0-)

R77.0 Abnormality of albumin
R77.1 Abnormality of globulin
Hyperglobulinemia NOS
R77.2 Abnormality of alphafetoprotein
R77.8 Other specified abnormalities of plasma proteins

New code R77.81 Abnormality in beta-amyloid and tau **plasma protein**
Add **Abnormality in** beta-amyloid **biomarkers** in plasma **protein**
Add **Abnormality** in **tau biomarkers** in plasma **protein**
Add Excludes1: Alzheimer's disease (G30.-)

New code R77.89 Other specified abnormalities of plasma proteins

R77.9 Abnormality of plasma protein, unspecified

R83 Abnormal findings in cerebrospinal fluid

R83.8 Other abnormal findings in cerebrospinal fluid
Delete ~~Abnormal chromosomal findings in cerebrospinal fluid~~

New code R83.81 **Abnormal** beta-amyloid and tau **findings** in cerebrospinal fluid
Add **Abnormality in** beta-amyloid **biomarkers** in cerebrospinal fluid
Add **Abnormality in** tau **biomarkers** in cerebrospinal fluid
Add Excludes1: Alzheimer's disease (G30.-)

New code R83.89 Other abnormal findings in cerebrospinal fluid
Add Abnormal chromosomal findings in cerebrospinal fluid

R90 Abnormal findings on diagnostic imaging of central nervous system

R90.8 Other abnormal findings on diagnostic imaging of central nervous system

New code **R90.83 Presence of beta-amyloid plaques and tau tangles on diagnostic imaging of the brain**
Add **Presence of beta-amyloid plaques of brain on Positron Emission Tomography [PET]**
Add **Presence of tau tangles of brain on Positron Emission Tomography [PET]**
Add Excludes1: Alzheimer's disease (G30.-)

Cardiogenic Shock Staging

Cardiogenic shock (CS) is a complex, high-acuity, and hemodynamically heterogeneous state associated with significant morbidity and mortality. Traditional definitions of CS in trials and registries relied primarily on hypotension, reduced cardiac output, and signs of end-organ hypoperfusion.[1] However, these definitions oversimplify a dynamic clinical syndrome that exists on a continuum of severity and fail to recognize the well-documented poor outcomes associated with normotensive cardiogenic shock.[2]

Current expert consensus statements from the Society for Cardiovascular Angiography and Interventions (SCAI) recommend classifying CS using a five-stage schema (A–E) that more accurately characterizes severity and prognostic risk.[3–4] SCAI stages B–E have been strongly correlated with in-hospital mortality and are now widely used to guide clinical decision-making, triage, resource allocation, and advanced therapies.

Currently ICD-10-CM has a single code R57.0, Cardiogenic shock, which is used for all levels of CS severity. Since this ranges from early or normotensive CS (SCAI B) to refractory shock (SCAI E), it combines a wide range of severity. Adding specific ICD-10-CM codes for the stages of cardiogenic shock will reflect current clinical practice and consensus-based definitions endorsed by major professional societies, improve diagnostic accuracy and consistency across institutions, and enable researchers and health systems to track outcomes and resource utilization across clinically meaningful CS subgroups. This will also enable better tracking for patients requiring high-complexity care, particularly for those needing escalation to mechanical circulatory support. These codes align with the validated SCAI classification framework and will allow clinicians and coders to accurately represent patient severity in administrative and claims data. Given the widespread clinical adoption and prognostic importance of SCAI cardiogenic shock staging, the addition of stage-specific ICD-10-CM codes is essential to improve diagnostic accuracy and enhance the reliability of national research and quality improvement efforts.

The proposal to create codes for stages of cardiogenic shock was received from the Society for Cardiovascular Angiography and Interventions (SCAI), and development of this proposal also included input and support from the American College of Cardiology.

References

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TABULAR MODIFICATIONS

R57 Shock, not elsewhere classified

R57.0 Cardiogenic shock

Add — Excludes1: Cardiogenic shock, SCAI Stage A (at risk for cardiogenic shock) (Z91.86)

New code — R57.00 Cardiogenic shock, unspecified

New code — R57.01 Cardiogenic shock, SCAI Stage B
Add — Beginning shock
Add — Society for Cardiovascular Angiography & Interventions cardiogenic shock stage B

New code — R57.02 Cardiogenic shock, SCAI Stage C
Add — Classic shock
Add — Society for Cardiovascular Angiography & Interventions cardiogenic shock stage C

New code — R57.03 Cardiogenic shock, SCAI Stage D
Add — Deteriorating shock
Add — Society for Cardiovascular Angiography & Interventions cardiogenic shock stage D

New code — R57.04 Cardiogenic shock, SCAI Stage E
Add — Extremis shock
Add — Society for Cardiovascular Angiography & Interventions cardiogenic shock stage E

Z91 Personal risk factors, not elsewhere classified

Z91.8 Other specified personal risk factors, not elsewhere classified

New code — Z91.86 Cardiogenic shock, SCAI Stage A
Add — At risk for cardiogenic shock
Add — Society for Cardiovascular Angiography & Interventions cardiogenic shock stage A

Carotid Web

Carotid web is an intimal variant of fibromuscular dysplasia characterized by a shelf-like projection into the lumen of the proximal internal carotid artery bulb [1]. It is increasingly recognized as an important cause of ischemic stroke, being recently incorporated into the American Heart Association guidelines for secondary stroke prevention as a discrete cause of stroke [2]. Carotid web related strokes occur particularly in younger patients without traditional atherosclerotic risk factors. These lesions generate flow disruption, with a large area of flow stasis and recirculation and thus subsequent thrombus formation. This can lead to artery-to-artery embolism. Stroke recurrence rates are high, reaching nearly 20% in 2 years [3, 4]. The diagnosis is made by vascular imaging, with CT angiography, MR angiography, or catheter angiography demonstrating the classic shelf-like intraluminal bulb protrusion [5].

Carotid web is underrecognized in clinical practice but is being reported with greater frequency in recent years [6]. In some series, it accounts for up to 20% of recurrent cryptogenic ischemic strokes in young and middle-aged adults [7, 8].

At this time carotid web should be coded to I77.3, Arterial fibromuscular dysplasia. However, this code is used for all types of fibromuscular dysplasia, including that involving the renal artery, vertebral artery, and mesenteric artery, as well as the carotid artery, among others. Carotid web has been referred to as an atypical variant of fibromuscular dysplasia. In contrast, typical fibromuscular dysplasia has a classic imaging appearance described as a “string of beads,” and it does not have a direct association with ischemic stroke [7]. However, renal artery fibromuscular dysplasia has often been related to hypertension, and hypertension can be associated with stroke risk. Differentiating carotid web from other types of fibromuscular dysplasia would be of clinical value and epidemiological utility.

A unique ICD-10-CM code for carotid web would improve clinical recognition and enable documentation and tracking, allow for accurate epidemiologic surveillance, facilitate ongoing and future research, and support the collection of standardized data for clinical trials and registries. It would also improve the precision of documentation in support of interventions such as stenting or endarterectomy in patients with this condition [9, 10]. This would also promote improved communication among healthcare providers, researchers, and others, which is critical for improving patient care. This proposal is based on a request to add specific codes for carotid web received from Diogo C. Haussen, MD, Grady Memorial Hospital, Emory University.

References

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TABULAR MODIFICATIONS

I77 Other disorders of arteries and arterioles

	I77.3	Arterial fibromuscular dysplasia	
Delete		~~Fibromuscular hyperplasia (of) carotid artery~~	
Delete		~~Fibromuscular hyperplasia (of) renal artery~~	
New code	I77.30	Arterial fibromuscular dysplasia, unspecified	
New sub-subcategory	I77.31	Carotid web	
New code		I77.310	Carotid web, right carotid artery
New code		I77.311	Carotid web, left carotid artery
New code		I77.319	Carotid web, unspecified
New code	I77.39	Other arterial fibromuscular dysplasia	
Add		Fibromuscular hyperplasia (of) carotid artery	
Add		Fibromuscular hyperplasia (of) renal artery	

INDEX MODIFICATIONS

	Dysplasia – see also Anomaly
Revise	- arterial, fibromuscular I77.30 – see also Hyperplasia, fibromuscular of artery
	Hyperplasia, hyperplastic
Revise	- artery, fibromuscular I77.30 – see also Hyperplasia, fibromuscular of artery
Revise	- fibromuscular of artery (carotid) (renal) I77.30
Add	- - carotid I77.39
Add	- - - web I77.319
Add	- - - - left I77.311
Add	- - - - right I77.310
Add	- - renal I77.39
Add	- - specified NEC I77.39
	Web, webbed (congenital)
Add	- carotid (artery) I77.319
Add	- - left I77.311
Add	- - right I77.310

Carrier of Candidiasis

Candida auris (C. auris) is a fungus infection that can cause serious illness. Some strains can become resistant to multiple drugs, making them hard to treat. It most commonly spreads in hospitals or long-term care facilities. A C. auris infection can come from contact with someone who has it (even if they have no symptoms) or from a contaminated surface. In healthcare settings, infections can pass from person to person or through contaminated medical equipment. It can also be transmitted through a medical device in your body (like a central venous line or breathing tube).

C. auris can cause infection in different parts of the body like the blood, wounds, and ears. Symptoms of a C. auris infection depend on the location and severity of infection. Symptoms may be similar to symptoms of infections caused by bacteria like fever or chills. There is not a common set of symptoms specific for C. auris infections.

C. auris patients may have an infection on their skin and other body area without having symptoms. Healthcare providers refer to this as 'colonization.' People who are colonized can spread C. auris onto surfaces and objects around them and to other patients.

Patients who are infected and who are colonized with C. auris often spread it onto surfaces and objects in healthcare settings like bedrails, doorknobs, and blood pressure cuffs. C. auris can survive on surfaces and objects for a long time and spread to other patients who can then become sick.

Many types of Candida live on the skin and in parts of the body and normally do not cause any health effects. Candida species are closely related to each other but not the same. The most common species that can overgrow and cause candidiasis are: Candida albicans, Candida glabrata, Candida krusei, Candida parapsilosis and Candida tropicalis.

The proposal was submitted by Angela Ellerbroek, RN, CWON from Bartrels Lutheran Retirement Community to create a new code for carrier of Candidiasis to include C. auris.

References

https://my.clevelandclinic.org/health/diseases/25152-candida-auris

https://www.cdc.gov/candida-auris/about/index.html

https://www.cdc.gov/candidiasis/about/index.html

TABULAR MODIFICATIONS

	B37	Candidiasis
Add		Includes: Candida albicans
Add		Candida auris
	Z22	Carrier of infectious disease
New code		Z22.A Carrier of Candidiasis
Add		Carrier of Candida albicans
Add		Carrier of Candida auris

Chronic hand eczema

This topic was originally presented at the September 2025 ICD-10 Coordination and Maintenance meeting. Based on comments received during the public comments period, a revised proposal is being presented for consideration.

Chronic hand eczema, also called chronic hand dermatitis, is a common inflammatory skin disease that lasts for more than three months or relapses twice or more within a year. It is the most common occupational disorder of the skin and creates a significant burden, greatly impacting quality of life for affected individuals due to its location on the hands.

The disorder is characterized by erythema, edema, and vesicles, leading to scaling, thickening, hyperkeratosis, lichenification, and fissures of the hands. Nail changes may also occur, including loss of the cuticle and thickening of the nail plate.

Chronic hand eczema is a very common skin disorder, with a general prevalence of about 4-5% at any given time and a lifetime prevalence of about 14%. It is a heterogeneous disease that can have many underlying and overlapping etiologies, including atopic dermatitis, irritant contact dermatitis and allergic contact dermatitis. Moreover, there are many morphologic presentations of this disease including chronic vesicular hand eczema (dyshidrosis/pompholyx), hyperkeratotic hand eczema, and nummular eczema. These etiologies can all present with the same signs, symptoms, and sites of involvement on the hand.

Due to the anatomic site and the symptoms of this condition, it presents with a significant burden to the patient's physical, psychological and social health and can result in additional challenges, for example at work. To this end, it is also more prevalent in people with certain occupations that involve frequent exposure of the hands to water, irritants, chemicals, and allergens, such as nurses, hairdressers, butchers, construction workers, metalworkers, and florists.

Chronic hand eczema is widely used terminology by the FDA, dermatologists, nurse practitioners, and physician assistants. The terminology indicates the anatomic site of the disorder but beyond that, it indicates that there are a number of etiologies of the disease that (1) cannot typically be distinguished based on clinical history, examination or testing, including allergy testing or biopsy; and (2) are often present in the same patient at the same time. For these reasons, providers use the overarching diagnosis of chronic hand eczema to avoid misclassifying patients. However, this terminology is not currently available within ICD-10-CM, resulting in misclassification of patients when coded, as the provider is currently forced to select one of the potential etiologies without knowing if this is accurate.

Feedback from dermatologists is that having an ICD-10-CM code for chronic hand eczema is important to enable appropriate patient classification and coding that aligns with the clinical or research scenarios.

Management of chronic hand eczema involves basic skincare steps including avoiding the allergens and irritants identified, using specific handwashing and drying techniques, and taking

skin protection measures such as gloves, barrier creams, and emollients. Treatment frequently involves topical anti-inflammatories, such as corticosteroids. In moderate to severe disease, systemic therapies may be needed, including oral corticosteroids. Immunosuppressants, such as cyclosporine and methotrexate, and retinoids are also used. However, these have significant side effects, including teratogenicity in retinoids, which may preclude use as a long-term treatment. Phototherapy may also be used, largely on the basis of its effectiveness in other skin disorders. Notably, none of these therapies are FDA-approved specifically for chronic hand eczema in the US. Recently, a pan-JAK inhibitor, delgocitinib cream, was approved by the FDA to specifically treat chronic hand eczema. It is the first and only FDA-approved medication to treat chronic hand eczema.

LEO Pharma, Incorporated, a pharmaceutical company, is requesting a new code to allow clinicians and researchers to accurately classify individuals with chronic hand eczema for research and treatment.

References:

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TABULAR MODIFICATION

New subcategory	L30.8	Other specified dermatitis
New code	L30.81	Idiopathic chronic dermatitis of hand
Add		Chronic hand eczema
Add		Code also, if applicable:
Add		allergic contact dermatitis (L23.-)
Add		atopic dermatitis (L20.-)
Add		dyshidrosis (L30.1)
Add		hyperkeratosis (L85.9)
Add		irritant contact dermatitis (L24.-)
Add		nummular dermatitis (L30.0)
New code	L30.89	Other specified dermatitis

Congenital Hyperinsulinism

Congenital hyperinsulinism (HI) is a distinct disease entity that was first described in 1954.[1] It is a group of heterogeneous β-cell disorders, which are characterized by recurrent episodes of hyperinsulinemic hypoglycemia due to dysregulated insulin secretion.[2-6] Congenital HI is the most common cause of persistent hypoglycemia in infancy and childhood,[3,4,6,7] and is associated with increased risk of seizures, motor and learning disabilities, permanent brain damage, and death if treatment is delayed.[3,4,8,9]

The incidence of congenital HI is estimated to be approximately 1:28,000 in most countries,[10,11] and can be up to 1:2,500 in limited gene pool populations.[2,3,12] Clinical presentation of congenital HI is heterogenous. Early signs and symptoms can be non-specific and may include jitteriness, poor feeding, palpitations, and sweating. More serious signs and symptoms reflect neuroglycopenia and include apnea, seizures, severe irritability, unconsciousness, lethargy, coma, status epilepticus, and even death.[2,6,13]

There are three histological forms of congenital HI: diffuse, focal, and mosaic (atypical). 60-70% of diffuse form cases are caused by the inactivating KATP channel mutations, responsible for the most common and severe types of disease.[6,14] This form may be inherited in an autosomal recessive or dominant manner. 30-40% of congenital HI cases are focal and are mainly caused by a monoallelic mutation in ABCC8 or KCNJ11 genes with paternal inheritance and a somatic loss of the maternal 11p15 in a small region of the pancreas. The atypical forms consist of a mosaic pattern of diffuse or focal forms.[6,14] Although over 30 genetic forms of congenital HI causing isolated or syndromic forms of the condition have been described to date,[15] approximately 40-50% of patients remain genetically unidentified.[16,17] International Guidelines recommend genetic testing for all children, and pancreatic imaging studies (e.g., 18F-DOPA PET scan) are recommended for patients with suspected focal forms.[14]

There are limited treatment options for infants and children with congenital HI. The International Guidelines for the Diagnosis and Management of Hyperinsulinism, published in 2024, provide recommendations for the treatment of infants and children with congenital HI.[14] The authors recommend to promptly restore and maintain plasma glucose levels within the normal range with intravenous (IV) glucose (dextrose) infusion. When high-dose IV dextrose is required, clinicians should add continuous glucagon IV infusion to help control hypoglycemia in infants who are at risk of fluid overload because of high glucose requirements. Diazoxide, which has been approved by the US Food and Drug Administration since 1976, is recommended as the first line therapy for patients with established congenital HI diagnosis. However, certain genetic forms (e.g., those with KATP-channel defects) are unresponsive to this treatment. Somatostatin analogs are recommended as the second line of therapy in patients who are diazoxide-unresponsive, have unacceptable diazoxide side effects, or are unable to obtain diazoxide. Surgical treatment should be considered in children with resectable focal lesion or if diffuse forms are not adequately controlled despite maximal medical therapy.[14] Patients with diffuse congenital HI lack curative treatment options, and currently available standard of care treatments are associated with significant side effects.

There is a substantial unmet need for this patient population, regardless of the disease form.

Congenital hyperinsulinism is a distinct disease entity with its unique etiology, clinical presentation, and diagnosis and treatment patterns. ICD-10-CM code E16.1, Other hypoglycemia currently encompasses a broader patient population with similar symptoms but different etiologies, including functional hyperinsulinism, functional nonhyperinsulinemic hypoglycemia, hyperinsulinism NOS, and hyperplasia of pancreatic beta cells NOS. E16.1 also describes ketotic hypoglycemia, a condition typically associated with normal physiological fasting and the single most common cause of hypoglycemia in childhood. It fails to accurately capture the appropriate congenital HI patient population and does not reflect current clinical practices in the US.

Congenital hyperinsulinism is a widely accepted term in clinical practice, which is used to characterize a group of heterogeneous β-cell disorders. Creating a new code will help align US practice with global standards, enable more accurate disease surveillance, ensure more precise coding and alignment with current clinical practice, research, and peer reviewed literature. The new code will empower researchers and clinicians to study this condition further, its current and future treatment options, and patient outcomes at a more granular level. A specific code for congenital HI will ensure consistency in coding practices between different treatment centers (e.g., centers of excellence, community clinics, etc.) and will support alignment between treating physicians' diagnosis.

This proposal is submitted by the Congenital Hyperinsulinism International organization and has been reviewed and supported by the American Academy of Pediatrics.

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TABULAR MODIFICATIONS

E16 Other disorders of pancreatic internal secretion

New code
Add — E16.B Congenital Hyperinsulinism
- Congenital ***β***-cell dysregulation
- Diffuse congenital hyperinsulinism
- Familial or persistent hyperinsulinemic hypoglycemia
- Focal congenital hyperinsulinism
- Genetic hyperinsulinism
- Persistent hyperinsulinemic hypoglycemia of infancy (PHHI)
- Persistent neonatal hypoglycemia due to hyperinsulinism

Add — Excludes1: Benign neoplasm of endocrine pancreas (D13.7)
- Iatrogenic neonatal hypoglycemia (P70.3)
- Other hypoglycemia (E16.1)
- Other transitory disorders of carbohydrate metabolism of newborn (P70.8)
- Transitory disorder of carbohydrate metabolism of newborn, unspecified (P70.9)

Add — Excludes 2: Syndrome of infant of mother with gestational diabetes (P70.0)
- Neonatal diabetes mellitus (P70.2)
- Other neonatal hypoglycemia (P70.4)
- Syndrome of infant of a diabetic mother (P70.1)

Controlled Obesity

Obesity is a chronic, relapsing, multifactorial disease that affects more than 2 in 5 adults in the United States and is associated with increased risk of related comorbidities, hospitalizations, and premature and avoidable mortality. Obesity persists biologically even when a patient's Body Mass Index (BMI), anthropometric measurements, and body composition parameters improve to levels considered healthy. Clinical control of obesity is defined not only by weight reduction but by improvement across multiple validated biomarkers, including BMI within or approaching a healthy range, waist circumference and waist-to-height ratio reflecting reduced central adiposity, body composition metrics such as decreased fat mass, visceral adiposity, and improved lean mass retention and improvements in metabolic markers such as glucose regulation, lipids and blood pressure.

These parameters often improve with effective treatment, but improvement does not equate to disease cure. Decades of research demonstrate that neurohormonal changes and metabolic adaptations driving weight regain persist beyond the restoration of healthy anthropometric or body composition measures. Adipose tissue alterations and inflammatory signaling pathways remain partially active after weight reduction as do genetic, environmental and behavioral contributors to obesity susceptibility. Thus, even when the disease appears normalized, the underlying pathophysiology of obesity remains, necessitating requiring sustained, long-term and structured intervention and comprehensive care.

Despite this impact, currently ICD-10-CM does not provide a way to identify individuals with Controlled Obesity – patients with a documented history of obesity who achieved medically significant improvements in weight, BMI, and anthropometric measurements with medical and/or surgical, nutritional, and behavioral management and continue to require the long-term, structured monitoring and treatment to maintain a healthy BMI, metabolic health, and body composition.

Creation of a specific ICD-10-CM code for controlled obesity would have multiple important clinical and public health impacts. It would allow clinicians to document, track and appropriately risk stratify the patients whose anthropometric markers will still have an elevated lifetime cardiometabolic risk. This will help differentiate individuals who never had obesity from those at higher probability of recurrence of visceral adiposity and ectopic fat deposition and a greater risk of relapse in the absence of continued treatment. Additionally, it will improve transparency into the true burden of the disease of obesity. Current ICD-10-CM coding does not differentiate individuals who have a normalized BMI or improved anthropometrics due to treatment but persistent metabolic vulnerability. These individuals become invisible in surveillance data, leading to the underestimation of the population requiring chronic obesity care.

Finally, this will empower clinicians to document and track the need for ongoing medical intervention and supportive therapies when BMI alone is normal, especially with obesity related cardiometabolic comorbidities. This will provide accurate documentation for health care stakeholders regarding the necessity of continued clinical management, preventing fragmented or episodic care. Integrating these multidimensional clinical measures into the definition aligns ICD-

10-CM coding with current scientific understanding, enhances risk stratification, and reflects the true burden of the chronic and relapsing disease of obesity.

The proposal to create a specific code for controlled obesity was received from Knownwell, a provider group focused on delivering weight-inclusive, comprehensive Primary and Metabolic Care and is endorsed by The Obesity Medicine Association, Stop Obesity Alliance, The Obesity Society and Obesity Action Coalition.

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TABULAR MODIFICATIONS

E66 Overweight and obesity

E66.8 Other obesity

New code E66.82 Controlled Obesity

Corneal Pseudomicrocysts

Corneal pseudomicrocysts, also known as microcyst-like epithelial changes (MECs), are a recognized adverse effect of antibody-drug conjugates (ADCs) that are used to treat various malignancies.

ADCs are a relatively recent development in cancer treatment. As the name implies, ADCs are composed of a monoclonal antibody linked to a cytotoxin. The monoclonal antibody targets cancer cells via the antigens they express and the cytotoxin then destroys the cancer cells. ADCs are administered as intravenous infusions. Among other malignancies, ADCs have been used in the treatment of multiple myeloma, leukemia and lymphoma, breast cancer, endometrial and cervical cancer, glioblastoma, and lung cancer. The first ADC was FDA-approved in 2000 and, as of 2025, there are 15 FDA-approved ADCs. Their effectiveness has been such that over 150 additional ADCs are currently in clinical trials worldwide, with utilization expected to increase substantially in the coming years.

Corneal pseudomicrocysts have been identified as an adverse effect of ADCs for over a decade.[1] These are small, round, reflective structures that arise in the corneal epithelium, the outermost layer of the cornea which makes up the surface of the eye. Histopathologically, they represent drug-related epithelial vacuolization and disruption rather than pigment deposition or corneal edema. As part of the ocular surface, the cornea plays a key role in focusing light onto the retina. Corneal pseudomicrocysts typically begin in the corneal periphery and migrate to the central cornea over time. They can be seen by slit-lamp examination and are the hallmark finding in ADC-induced ocular surface adverse effects. Dry eyes, photophobia, eye pain, and blurry vision are also common concurrent findings. However, these findings are themselves due to the breakdown of the corneal epithelium caused by the pseudomicrocysts. These ocular adverse events can lead to significant functional impairments and impact patient quality of life, for example by affecting the ability to read and to drive.[2, 3]

In two recent global trials of an ADC directed at relapsed/refractory multiple myeloma, with one submitting results in 2024 and the other in 2025, the data showed strong efficacy against multiple myeloma. However, the data also showed a high level of ocular adverse events, 79% in one trial and 89% in the other.[4] The FDA Oncologic Drugs Advisory Committee (ODAC) cited the high rates of ocular adverse events in voting against approval for this ADC drug. In other studies of patients being treated with various ADCs, the incidence of corneal pseudomicrocysts ranged from 41% to 100%.[2] Across multiple trials of different ADCs, delay in dosing (up to 50%), reduction in doses (up to 35%), and discontinuation (up to 25%) due to corneal findings demonstrate significant clinical, societal, and economic impact, including interrupted cancer therapy, increased ophthalmic visits, and additional diagnostic testing.[5]

Current strategies to mitigate corneal pseudomicrocysts include the use of artificial tears, corticosteroid eye drops, and ocular surface vasoconstrictors. Unfortunately, none of these have shown consistent, clinically meaningful benefit in preventing or treating corneal pseudomicrocysts. To date, the only effective management strategy for corneal pseudomicrocysts

has been interrupting, delaying, or reducing ADC therapy.[2] This creates an issue for treating oncologists in balancing whether to reduce, pause, or discontinue use of ADCs to address the corneal adverse effects against the need to continue treating the malignancy.

The need to assess and monitor ocular adverse events in patients receiving ADCs has brought ophthalmologists into the oncology treatment team. In July 2025, the American Academy of Ophthalmology (AAO) released recommendations for eye examinations in patients being treated with ADCs. These include ophthalmologists performing a baseline examination prior to the start of ADCs, monitoring the patient regularly for early identification of ocular adverse events, and providing assessments and guidance to the treating oncologists to enable them to determine if alterations in ADC dosing are needed.[3, 6] These guidelines explicitly call for standardized terminology and consistent reporting of ADC-related ocular toxicities.

Collaboration and coordination of care between ophthalmology and oncology can help to ensure that patients continue to receive potentially life-saving ADC treatment for malignancy while protecting their vision. Particularly as ADCs expand into more indications and earlier lines of therapy, being able to identify ADC-related corneal pseudomicrocysts in the data will be essential.

Current reporting of corneal pseudomicrocysts is recognized as limited and inconsistent.[2] The lack of standardization in coding impairs accurate tracking of ADC-associated corneal toxicity, obscures its true incidence and severity, and limits the ability of researchers and other stakeholders to evaluate its impact on care delivery and outcomes. Creation of a specific code will enable surveillance, quality improvement, and health services research.

This proposal is submitted by Neel Pasricha, MD Assistant Professor of Ophthalmology and Associate Member in Breast Oncology at University of California, on behalf of the University of California (San Francisco). The proposal has been reviewed and supported by the American Academy of Ophthalmology.

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TABULAR MODIFICATIONS

H18 Other disorders of cornea

H18.0 Corneal pigmentations and deposits

Add — Excludes2: Corneal pseudomicrocysts (H18.84-)

H18.1 Bullous keratopathy

H18.2 Other and unspecified corneal edema

H18.8 Other specified disorders of cornea

H18.83 Recurrent erosion of cornea

New sub-subcategory — H18.84 Corneal pseudomicrocysts

Add — Antibody-drug conjugate (ADC) keratopathy
Add — Corneal pseudomicrocysts due to antibody-drug conjugate (ADC)
Add — Microcyst-like epithelial changes (MECs)

Add — Code also, if applicable, underlying malignant neoplasm

Add — Use additional code for adverse effect, if applicable, to identify drug (T45.1X5)

New code — H18.841 Corneal pseudomicrocysts, right eye

New code — H18.842 Corneal pseudomicrocysts, left eye

New code — H18.843 Corneal pseudomicrocysts, bilateral

New code — H18.849 Corneal pseudomicrocysts, unspecified eye

H18.89 Other specified disorders of cornea

Add — Excludes1: Corneal pseudomicrocysts (H18.84-)
Add — Corneal cysts

Dysphotopsia

Dysphotopsias are unwanted visual phenomena. They can occur naturally, after many ocular surgeries, and following otherwise-successful cataract surgery. Positive Dysphotopsia (PD) includes phenomena such as light streaks, arcs, flashes, rings and halos, or glare caused by external light sources. Negative Dysphotopsia is a dark arc-shaped shadow or line in the peripheral vision. [1,2] PD is associated with light entering the eye, often caused by changes such as IOL implantation, where ND may stem from other intrinsic factors independent of the cataract surgery. [2]

The most common factors affecting occurrence of PD are characteristics of the intraocular lens (IOL), such as the IOL shape, edge design, and size. [1,2] Causes of ND are less well-understood and believed to be multi-factorial. ND can occur regardless of the type of IOL [3] and can be affected by placement and orientation of the IOL as well as the patient's anatomy. [4,5,6] Both PD and ND are transient symptoms in most patients but may persist for at least one year in 2-3% of patients.[2]

Knowing the type of dysphotopsia is important when evaluating a patient and leads to a different treatment pathway both pre and intra operatively. [2] Surgical interventions may be appropriate as a second line treatment for patients with persistent dysphotopsia. For PD, such intervention would be an IOL exchange. For ND, possible interventions include reverse optic capture technique, secondary (piggyback) IOL implantation, or IOL exchange.

Currently, both Positive Dysphotopsia (PD) and Negative Dysphotopsia (ND) are captured under ICD-10-CM code H53.19, Other subjective visual disturbances.

Johnson and Johnson Vision is requesting the following tabular modifications to support effective disease tracking of the condition and to facilitate research.

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TABULAR MODIFICATIONS

H53 Visual disturbances

H53.1 Subjective visual disturbances

New sub-subcategory	H53.17	Dysphotopsia
New sub-sub-subcategory	H53.170	Positive dysphotopsia
New code	H53.1701	Positive dysphotopsia, right eye
New code	H53.1702	Positive dysphotopsia, left eye
New code	H53.1703	Positive dysphotopsia, bilateral
New code	H53.1709	Positive dysphotopsia, unspecified eye
New sub-sub-subcategory	H53.171	Negative dysphotopsia
New code	H53.1711	Negative dysphotopsia, right eye
New code	H53.1712	Negative dysphotopsia, left eye
New code	H53.1713	Negative dysphotopsia, bilateral
New code	H53.1719	Negative dysphotopsia, unspecified eye
New sub-sub-subcategory	H53.179	Unspecified dysphotopsia
Add		Dysphotopsia NOS
New code	H53.1791	Unspecified dysphotopsia, right eye
New code	H53.1792	Unspecified dysphotopsia, left eye
New code	H53.1793	Unspecified dysphotopsia, bilateral
New code	H53.1799	Unspecified dysphotopsia, unspecified eye

Encounter for observation for suspected condition: Delirium - EEG Monitoring

Delirium is a manifestation of acute brain dysfunction defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-5)[1] as a disturbance in attention and awareness that develops over a short period of time, fluctuates, and is accompanied by a change in cognition. Delirium is common in critically ill adults and is the most common psychiatric disorder found in the general hospital setting, causing widespread adverse outcomes in medically ill subjects. Rates of delirium range from 20–40% among critically ill subjects, with the higher rates of 60–80% observed in mechanically ventilated medical or surgical subjects.[2-4] Delirium has multiple underlying causes, ranging from metabolic disorders, infection and sepsis, medication and sedative related causes, to pain, discomfort and sleep disturbance[17].

Studies have demonstrated that the occurrence of delirium is associated with a number of short- and long-term complications. In the short term, subjects suffering from delirium have an increased risk of self-injury (e.g., self-extubation, acute respiratory distress syndrome, pneumonia), prolonged hospital stay, and mortality[5]. Long-term, delirium has been associated with a slower physical recovery and an increased rate of placement in specialized intermediate and long-term care facilities. Furthermore, delirium is associated with poor functional and cognitive recovery, mental health impairments, decreased quality of life, and increased health care costs[6].

Despite a high prevalence, delirium remains unrecognized by most ICU clinicians in as many as 66%–84% of subjects, likely because of difficulty at making an accurate diagnosis in subjects who have hypoactive delirium[7-9].

The Diagnostic and Statistical Manual for Mental Disorders (DSM-5)[11] and the International Statistical Classification of Diseases and Related Health Problems (ICD-11)[12] are considered the diagnostic ‘gold standards for delirium diagnosis. To date, numerous surveillance and diagnostic tools have been developed to assist clinicians in the identification of delirium (i.e., Intensive Care Delirium Screen Checklist, Confusion Assessment Method for the Intensive Care ICU). These instruments have been designed to help non psychiatrists screen for and diagnose delirium and have demonstrated high sensitivity and specificity in validation studies[5], but have demonstrated low sensitivity when tested in real-life conditions[12-14]. Low sensitivity is a significant issue, likely due to heterogeneous subject populations, leading to the inconsistent diagnosis of subjects with hypoactive and subsyndromal delirium. Current assessment tools also require significant nursing burden, and as such, common practice is to only assess every 12 hours, which could be

insufficient given delirium's potential to develop over a short period of time and to fluctuate[15-16]. There is a need for the development of new tools that provide sufficient sensitivity balanced with high specificity to allow for proper delirium screening, monitoring, and detection.

The Society of Critical Care Medicine (SCCM) has included delirium in the Clinical Practices Guidelines as a common diagnosis in critically ill patients that should be regularly assessed using a valid tool and that early detection is a potential benefit of delirium monitoring[18]. Therefore, delirium is accepted as a distinct neurological condition associated with multiple underlying causes.

Ceribell, a medical technology company, requests a modification to the ICD-10-CM code set in order to align clinical documentation with the practice of continuous EEG monitoring to evaluate patients for delirium. The proposed codes align with proposed indications in an application for new technology add-on payments. Diagnosis and treatment must be based on patient's neurological condition. Clinical studies have been rapidly evolving, and the implications on patient clinical outcomes and long-term neurological impairment are now being fully recognized. Further specificity of the codes will also support patient and provider education and epidemiology research.

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Encounter for observation for suspected condition: Status Epilepticus- EEG Monitoring

Status epilepticus is a neurological emergency defined as a prolonged seizure or a series of seizures. Frequent seizures and status epilepticus carry substantial risk, contributing to significant morbidity and mortality[1].

In critical care environments, seizures are particularly common among patients with altered mental status, with approximately 30% affected. Seizures and status epilepticus affect patients across diverse clinical settings, including those with known epilepsy, acute neurologic injury (e.g., stroke, traumatic brain injury, CNS infection), metabolic derangements, toxic exposures, and anoxic brain injury.

Clinically, diagnosis of status epilepticus relies on the integration of bedside assessment with neurophysiological monitoring, particularly continuous electroencephalography (EEG). While convulsive status epilepticus is usually identifiable through overt motor activity, nonconvulsive status epilepticus —which may present solely as altered mental status or subtle motor signs—can only be detected through EEG monitoring[2-5]. EEG is considered the diagnostic gold standard for detecting ongoing epileptiform activity, determining seizure burden, and guiding response to therapy. Early EEG monitoring also helps distinguish status epilepticus from mimics such as metabolic encephalopathy, psychogenic events, or postictal states, each of which may appear clinically similar but require fundamentally different management.

A high seizure burden, defined as the proportion of time a patient spends in seizure activity, has been consistently linked to greater risk of neurological deterioration, poorer functional outcomes, and the later development of epilepsy [6-10]. These findings underscore the importance of rapid, reliable seizure detection and quantification in order to prevent ongoing neuronal injury. Timeliness becomes especially crucial when seizure burden exceeds 20% of the EEG record [6,11], a threshold associated with electrographic status epilepticus[12].

Status epilepticus is commonly accepted as a distinct clinical entity with specific diagnostic criteria. The 2012 Neurocritical Care Society guidelines[13] defines convulsive, non-convulsive and refractory status epilepticus, based on its semiology, duration and underlying etiology.

Ceribell, a medical technology company, requests a modification to the ICD-10-CM code set in order to align clinical documentation with clinical practice and optimize the diagnosis and treatment of status epilepticus using EEG-based solution. The proposed codes align with proposed indications in an application for new technology add-on payments

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TABULAR MODIFICATIONS

NOTE: The proposed codes, associated with a new technology add-on payment application, are presented in consideration for implementation April 1, 2027, following the usual 60-day public comment period.

Z03 Encounter for medical observation for suspected diseases and conditions ruled out

Z03.8 Encounter for observation for other suspected diseases and conditions ruled out

New sub- subcategory Z03.84 Encounter for observation for suspected conditions related to continuous EEG monitoring device, ruled out

New code Z03.841 Encounter for observation for suspected delirium related to continuous EEG monitoring device, ruled out

New code Z03.842 Encounter for observation for suspected status epilepticus related to continuous EEG monitoring device, ruled out

New code Z03.848 Encounter for observation for other suspected conditions related to continuous EEG monitoring device, ruled out

Facial Angiofibroma

Facial angiofibroma is a benign dermal neoplasm composed of dermal fibrous tissue and blood vessels that present clinically as multiple small, dome-shaped papules on the central face, including the malar regions, nasal dorsum, nasolabial folds, forehead, and chin. Lesions typically arise in early childhood (often between 2 and 5 years of age) and may enlarge and increase in number over time. Depending on the relative proportions of fibrous and vascular components, lesions may appear skin-colored, erythematous, or hyperpigmented. Facial angiofibromas can substantially affect quality of life by causing disfigurement, spontaneous bleeding, secondary infection, pain, and functional impairment involving vision, breathing, or perioral mobility.[6-9] Management options include topical and systemic mTOR inhibitors, laser or abrasive therapies, and surgical procedures, with treatment goals focused on reducing lesion size, bleeding, and facial appearance burden.

Facial angiofibroma is one of the most common cutaneous manifestations of tuberous sclerosis complex (TSC), occurring in approximately 75%–90% of affected individuals. [1, 2] TSC is an autosomal dominant genetic disorder caused by pathogenic variants in the TSC1 or TSC2 gene and is characterized by hamartomatous lesions involving multiple organs, including the skin, brain, kidneys, heart, lungs, and eyes. The condition is also associated with neurologic and neuropsychiatric manifestations, such as epilepsy, intellectual disability, autism spectrum disorder, anxiety, and depression. [1-4] TSC has an estimated incidence of one in 6,000–10,000 live births and affects nearly 2 million people worldwide, including approximately 50,000 individuals in the United States.[1]

Although classically associated with TSC, facial angiofibroma is not specific to this condition. Multiple facial angiofibromas are seen in patients with multiple endocrine neoplasia type 1 (MEN1), and recognition of this finding can prompt appropriate endocrine evaluation and surveillance.[12] Facial angiofibromas have also been reported as a cutaneous manifestation of Birt–Hogg–Dubé (BHD) syndrome, sometimes as a predominant initial presentation.[13] In addition, facial angiofibromas may occur as isolated lesions or as multiple lesions in patients without clinical or genetic evidence of TSC, MEN1, BHD, or other systemic disease.[14]

At present, there is no specific ICD-10-CM code for Facial Angiofibroma; therefore, it is not possible to capture precise epidemiological factors for this rare disorder. It is often coded under Q85.1, Tuberous sclerosis, which is inclusive of many other manifestations. Therefore, it does not distinguish patients whose primary or only clinically significant manifestation is facial angiofibroma. This limitation prevents accurate capture of facial angiofibroma in patients without TSC, facial angiofibroma associated with other genetic syndromes such as MEN1 and BHD, and the epidemiology, natural history, and treatment patterns specific to facial angiofibroma across different patient populations.

A distinct diagnostic code would: 1) improve diagnostic specificity and surveillance by distinguishing facial angiofibroma from TSC overall, facilitating identification of isolated, TSC-associated, or syndromic cases (e.g., MEN1, BHD); 2) support epidemiologic and clinical research by enabling accurate assessments of disease burden, age of onset, progression, and comorbidities such as seizures and other TSC features; 3) enhance early detection and management, aligning with the *Updated International Tuberous Sclerosis Complex Diagnostic Criteria and Surveillance Recommendations*[11], which list facial angiofibroma as a major diagnostic feature and emphasize early treatment; 4) strengthen healthcare planning and quality measurement by enabling data-driven resource allocation, treatment tracking (including mTOR-based and procedural therapies), and support for affected patients and families.

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TABULAR MODIFICATIONS

Q85 Phakomatoses, not elsewhere classified

Q85.8 Other phakomatoses, not elsewhere classified

New code	Q85.84 Facial angiofibroma
Add	Adenoma sebaceum

Floppy Eyelid Syndrome

Floppy eyelid syndrome (FES) is a frequently underdiagnosed cause of eye irritation and discharge, often seen in the presence of obstructive sleep apnea. FES is defined by hyperlaxity of the upper and lower eyelids, lack of rigidity in the underlying tarsus, easy or spontaneous lid eversion, and associated chronic reactive papillary conjunctivitis.[1] Estimated prevalence of FES is 3.8-8.0% of the adult population, with the majority of cases going undiagnosed.[2,3]

Floppy eyelid syndrome is strongly correlated with obstructive sleep apnea, with studies showing 75-95% of patients with FES have obstructive sleep apnea.[1,4,5] There is a positive correlation between FES and obstructive sleep apnea severity, and treatment of sleep apnea with CPAP has even been linked to improvement in FES symptoms.[2,6] Proper documentation of FES is therefore crucial for sleep apnea surveillance and screening, enabling timely treatment. Treatment of obstructive sleep apnea has been shown to decrease cardiovascular events, and motor vehicle accidents, resulting in a 40% relative reduction in all-cause mortality.[7–10] FES is also associated with other conditions, such as ectropion, entropion, keratoconus, and obesity, heightening the importance of proper FES documentation.[1,5,11]

The hyperlaxity in FES is caused by a weakening of the tarsal plate due to elastin breakdown.[12] Symptoms include tearing, redness, photosensitivity, foreign body sensation, dryness, and decreased vision.[11,13,14] The mechanism of conjunctival and corneal irritation in FES is thought to be secondary to a combination of three factors: 1) lid eversion during sleep with direct mechanical irritation of the ocular surface, 2) inadequate tear film contact, 3) and/or underlying ischemia-reperfusion damages.[14] FES can lead to ocular surface damage and in severe cases corneal ulcers and scarring.[4,13,15] Treatments for FES include eye shields during sleep to prevent mechanical irritation, lubricating ointments, and surgery to tighten the eyelids.[13,14]

Current ICD-10-CM coding does not capture FES as a distinct syndrome. An ICD-10-CM code designation for FES would enable more consistent diagnosis of this underdiagnosed condition. Furthermore, a FES ICD-10-CM code would facilitate obstructive sleep apnea surveillance and screening for patients with FES, resulting in earlier diagnosis and treatment of this life-threatening disease. A FES ICD-10-CM code would also allow for improved research into FES pathophysiology, treatment, and its association with other disorders. Due to the degeneration of the tarsal plate and decreased elastin found in FES, the new codes should be placed as a subcategory under H02.7 - “Other and unspecified degenerative disorders of eyelid and periocular area”.[12]

This proposal is submitted by oculoplastic surgeons Anne Barmettler, MD, and Lalita Gupta, MD.

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TABULAR MODIFICATIONS

H02 Other disorders of eyelid

H02.7 Other and unspecified degenerative disorders of eyelid and periocular area

New Subcategory	H02.74	Floppy eyelid syndrome
New Code	H02.741	Floppy eyelid syndrome right upper eyelid
New Code	H02.742	Floppy eyelid syndrome right lower eyelid

New Code	H02.743	Floppy eyelid syndrome right eye, unspecified eyelid
New code	H02.744	Floppy eyelid syndrome left upper eyelid
New code	H02.745	Floppy eyelid syndrome left lower eyelid
New code	H02.746	Floppy eyelid syndrome left eye, unspecified eyelid
New Code	H02.749	Floppy eyelid syndrome, unspecified eyelid

Gender Identity Disorder, in remission: Transition and Detransition Codes

This topic was presented at the September 2025 ICD-10 Coordination and Maintenance meeting. Based on comments received during the public comments period and additional clinical consultations, Do No Harm is presenting a revised proposal for consideration. Changes are indicated in **bold.**

The literature on gender dysphoria encompasses cases of desistance, referring to a resolution of clinical symptoms associated with this mental health condition whereby one no longer meets diagnostic criteria for gender dysphoria. Additionally, some individuals with gender dysphoria who underwent medical and surgical interventions to transition from their natal sex have later pursued detransition.[1-5] Many of these individuals, who once experienced incongruence between their experienced/expressed gender and natal sex, have come to later accept their biological sex. Whereas they once met the diagnostic criteria for gender dysphoria, they have now entered a period of remission (desistance) from this condition. Their cognitive experience of incongruence has remitted whereby experienced/expressed gender and natal sex are now aligned. This reality, not accounted for in ICD-10-CM, is reflected in the new code in category F64 Gender identity disorder. The new code F64.A, Gender identity disorder, in remission, presented at the September 2025 ICD-10 Coordination and Maintenance meeting, has been accepted for implementation on October 1, 2026.

The clinical history of gender transition or gender detransition, are similarly not included in the ICD-10-CM classification system. Thus, a set of new codes is proposed at subcategory Z87.89, personal history of other specified conditions, to reflect this clinical circumstance which may have life-long health implications.[1,6,7]

Gender transition is defined by the American Psychological Association (APA) as "the process of shifting toward a gender role different from that assigned at birth, which can include social transition, such as new names, pronouns and clothing, and medical transition, such as hormone therapy or surgery."[8] The proposed personal history of gender transition codes reflect the circumstance of an individual who has begun social gender transition (e.g. change in name, pronouns, presentation, breast binding, genital tucking), medical gender transition (e.g. pharmacological with puberty blockers, cross-sex hormones), and/or surgical gender transition (e.g. surgical alteration or removal of sexual organs).

Gender detransition is defined as the act of stopping or reversing gender transition.[1,2,4] It entails reverting back to living as one's natal sex after having medically and/or surgically transitioned.[1] The proposed personal history of gender detransition code reflects the clinical circumstance of an

individual with a history of gender transition who has begun the process to return to their gender assigned at birth.

The new code for gender identity disorder, in remission, as well as proposed personal history of gender transition and detransition codes, in combination with existing codes to reflect complications or related factors influencing health and well-being, will allow providers to more accurately document an individual's clinical state to support the appropriate delivery of care. In addition, the resultant clinical data will support and improve the quality of care for patients with gender dysphoria who transition with or without subsequent desistance and/or detransition.

In summary, the ICD-10-CM diagnosis codes proposed by Do No Harm will provide a means to collect valuable health information to do research, improve patient care and safety, and inform the public health needs in this evolving area of medicine.

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TABULAR MODIFICATIONS

NOTE: The proposed codes are presented in consideration for implementation October 1, 2026, following a 30-day public comment period. Thus, public comments on this proposal are due April 17, 2026.

F64. Gender identity disorder

New code F64.A Gender identity disorder, in remission
Add Gender dysphoria, in remission (desistance)

Z87 Personal history of other diseases and conditions

Z87.8 Personal history of other specified conditions

Z87.89 Personal history of other specified conditions

Z87.890 Personal history of sex reassignment

Add Personal history of gender transition

New code Z87.8901 Personal history of social gender transition

New code Z87.8902 Personal history of medical gender transition

Add Personal history of pharmacological (hormonal) gender transition

New code Z87.8903 Personal history of surgical gender transition

New code Z87.8904 Personal history of intersex surgery

New code Z87.8909 Personal history of unspecified gender transition

New code Z87.893 Personal history of gender detransition

Add Code also personal history of sex reassignment (Z87.890-)

Hypertriglyceridemia

This topic was presented at the September 2025 ICD-10 Coordination and Maintenance meeting. Based on public comments, revisions to the proposal have been made for reconsideration.

National Lipid Association and the American Society for Preventive Cardiology issued a joint expert clinical consensus statement in March of 2025 titled, "Recognition and management of persistent chylomicronemia: A joint expert clinical consensus by the National Lipid Association and the American Society for Preventive Cardiology." The consensus statement uses the terminology normal, mild to moderate, severe, and extreme for defining the spectrum of hypertriglyceridemia (HTG) and identification of chylomicronemia.

HTG is diagnosed based on a fasting serum (or plasma) triglyceride panel. Individuals are diagnosed with HTG when fasting plasma concentrations of triglycerides exceed a threshold value of 150 mg/dL. Performance of and results from a lipid panel are critical to assessing diagnosis, disease risks, and treatment of HTG in clinical practice guidelines. Optimal triglyceride levels are below 150 mg/dL. HTG occurs when triglyceride levels exceed 150 mg/dL, and severe HTG (sHTG) occurs when triglyceride levels exceed 500 mg/dL.

Both sHTG [TGs ≥ 500 mg/dL] and extreme HTG (eHTG) [TGs ≥ 880 mg/dL] are characterized by very high levels of triglycerides. People with sHTG and eHTG experience an increased risk of developing other medical conditions, including acute pancreatitis and cardiovascular diseases. In the United States, 1.5% of adults, equal to approximately 3.9 million people, suffer from sHTG, and 0.29% of adults, equal to approximately 750,000 people, are diagnosed with eHTG.

eHTG, also referred to as chylomicronemia, involves excessive accumulation of chylomicrons in the blood, resulting in extremely high triglyceride levels. eHTG includes multifactorial chylomicronemia syndrome (MCS), characterized by genetic variances that on top of environmental factors lead to eHTG, and familial chylomicronemia syndrome (FCS), a distinct genetic disorder characterized by a limited ability to breakdown triglycerides in chylomicrons and, therefore, accumulation of those chylomicrons in the blood. MCS is more common and presents in adulthood, while FCS is rare and patients may present with eHTG even in childhood. Individuals with either condition are at high lifetime risk of acute pancreatitis.

Ionis pharmaceuticals is requesting the following new codes to facilitate research and characterization of the patient population, disease management, and treatment of patients with HTG.

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TABULAR MODIFICATIONS

E78 Disorders of lipoprotein metabolism and other lipidemias

E78.1 Pure hyperglyceridemia

Elevated fasting triglycerides
Endogenous hyperglyceridemia
Fredrickson's hyperlipoproteinemia, type IV
Hyperlipidemia, group B
Hyperprebetalipoproteinemia

Add — Pure hyperglyceridemia NOS
Very-low-density-lipoprotein-type [VLDL] hyperlipoproteinemia

Add — Use additional code to identify hypertriglyceridemia level, if applicable (E78.A-)

New sub-subcategory — E78.A Hypertriglyceridemia level

New code — E78.A0 Hypertriglyceridemia, mild to moderate

New code — E78.A1 Hypertriglyceridemia, severe

Add — Hypertriglyceridemia, severe [sHTG]

New code — E78.A9 Other Hypertriglyceridemia

Lipedema and lipolymphedema

This topic was presented at the September 2020, September 2024, and September 2025 ICD-10 Coordination and Maintenance meetings. Based on comments received during the public comments period and additional clinical consultations, a revised proposal is being presented for consideration.

Lipedema, initially described at the Mayo clinic in 1940,[1 2] is a loose, connective-tissue (fat) disease (lipomatosis) with a pathological deposition of fibrotic fatty tissue on the limbs of women sparing the trunk, hands and feet,[3 4] resulting in a disproportionate body habitus. Lipedema is thought to affect 11% of the female population.[5] There is no specific ICD-10-CM code for lipedema. Deposition of lipedema fat increases with stage and body mass index (BMI) and likely involves sex hormones during times when weight is gained (puberty, pregnancy and menopause). Lipedema is inherited in 60% of women likely through genes affecting microvessels resulting in excess fluid bound to glycosaminoglycans in the interstitial space.[6]

Unique to lipedema is fat that is highly resistant to loss by diet, exercise, or bariatric surgery.[7-9] Lipedema is often confused with secondary obesity or lymphedema. Because of the many signs and symptoms associated with lipedema, lipedema is also known as a syndrome.[2 10-12]

There are four stages of lipedema:[13]

Stage 1: Smooth skin over an enlarged hypodermis often with palpable pearl-sized nodules
Stage 2: Indentations of the skin often with a mattress pattern appearance due to the presence of fibrosis in the fibers and interstitial space[5] overlying a hypertrophic hypodermis with pearl-size and larger masses the size of walnuts or larger
Stage 3: Indentations of the skin accompanied by lobules of skin and hypodermal tissue that often form over the elbow, at the waist, at the hips, on the medial inner thighs and around the knees accompanied by small to very large masses
Stage 4: Extensive lobular and pendulous adipose tissue folds affecting multiple regions of the body, including the arms, abdomen, hips, thighs, buttocks, and legs

Stage 4 lipedema should be diagnosed by the presence of extensive lobular and pendulous adipose tissue folds affecting multiple regions of the body, including the arms, abdomen, hips, thighs, buttocks, and legs. This presentation has been described in Földi and Földi's *Textbook of Lymphology* as "elephantiastic lobar lipedema," due to its clinical similarity to advanced elephantiasis observed in severe lymphedema.[14] The term "lipolymphedema" is sometimes used to describe Stage 4 lipedema; however, lymphedema can arise at any stage of lipedema.

We also propose a code for *other lipedema stage* (E88.838) to be used when providers are unable to accurately assign a specific stage—for example, when disease severity is described as intermediate between stages (e.g., between stages 1 and 2 or 2 and 3),[15] or when suspected truncal involvement complicates staging. In contrast, E88.839 (*unspecified stage*) should be used by coders as a default when lipedema is documented but the provider does not specify a stage

Lymphedema is a chronic and progressive swelling caused by a low output failure of the lymphatic system, resulting in the development of a high-protein edema in the tissues.

Lymphedema is a lifelong condition for which no cure exists. Lymphedema can be either primary (hereditary) or secondary. Secondary lymphedema is the most common cause of the disease and affects approximately 1 in 1000 Americans. Complications of lymphedema include recurrent bouts of cellulitis and/or lymphangitis, bacterial and fungal infections, lymphangio-adenitis, deep venous thrombosis, poor wound healing, leg ulcers, severe functional impairment, disability, and necessary amputation. Patients with chronic lymphedema for 10 years have a 10% risk of developing lymphangiosarcoma.

Idiopathic lymphedema is a term used by non-specialists to document lymphedema in everyday medical practice. This term is usually based on a doctor's clinical judgment. Right now, medical coders see this diagnosis written in charts but have no way to record it with a code. Having a code would allow tracking and development of educational materials for healthcare providers in the future.

Dr. Karen Herbst, with support from the American Vein & Lymphatic Society (AVLS), is submitting the following modifications to identify and track lipedema with and without lymphedema patients.

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TABULAR MODIFICATIONS

E88 Other and unspecified metabolic disorders

New subcategory E88.2 Lipomatosis, not elsewhere classified
Delete ~~Lipomatosis NOS~~
Delete ~~Lipomatosis (Cheek) dolorosa [Dercum]~~

New code E88.21 Dercum disease
Add Lipomatosis dolorosa [Dercum]

New code E88.29 Lipomatosis, not elsewhere classified
Add Lipomatosis NOS

E88.8 Other specified metabolic disorders

New sub-subcategory E88.83 Lipedema
Add Lipedema syndrome

Add Code also, if applicable, lymphedema (I89.-)

New code E88.831 Lipedema, Stage 1
New code E88.832 Lipedema, Stage 2
New code E88.833 Lipedema, Stage 3
New code E88.834 Lipedema, Stage 4
Add Lipolymphedema

New code E88.838 Other lipedema stage
New code E88.839 Lipedema, unspecified stage
Add Lipedema NOS

E88.89 Other specified metabolic disorders
Delete ~~Launois-Bensaude adenolipomatosis~~

New code E88.891 Multiple symmetric lipomatosis
Add Launois Bensaude adenolipomatosis
Add Madelung's disease

New Code E88.898 Other specified metabolic disorders

I89 Other noninfective disorders of lymphatic vessels and lymph nodes

	I89.0	Lymphedema, not elsewhere classified Elphantiasis (nonfilarial) NOS Lymphangiectasis Obliteration, lymphatic vessel
Delete		~~Praecox lymphedema~~
		Secondary lymphedema
New code	I89.A	Idiopathic lymphedema

Q82 Other congenital malformations of skin

	Q82.0	Hereditary lymphedema
Add		Praecox lymphedema

Macular Telangiectasia

Macular telangiectasia is an uncommon disease affecting the macula. Macular telangiectasia causes vessel abnormalities, neovascularization, and microaneurysms around the fovea resulting in leakage and degeneration of the macula with loss of central vision. There are currently three types of macular telangiectasia identified.

Type 1 is congenital, typically unilateral, and uncommon. Blood vessels dilate, creating aneurysms that leak fluid damaging macular cells.[1]

Type 2 is bilateral and acquired. [1,2] Blood vessels around the fovea become abnormal and may dilate and leak, but the finding is also associated with neurodegeneration of the retina. Type 2 affects approximately 0.1% of the global population.[3] There is a gene therapy treatment of Type 2 with an implant producing ciliary neurotrophic factor to prevent retinal degeneration.

Type 3 is very rare, poorly understood, and primarily occlusive.

The American Academy of Ophthalmology is requesting the following tabular modifications to facilitate accurate data collection and proper tracking of this condition. The condition is frequently misdiagnosed[4] and development of an ICD-10 CM code could improve awareness by physicians and public health advocates, and such awareness could possibly improve access to timely intervention.

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TABULAR MODIFICATIONS

H35 Other retinal disorders

H35.0 Background retinopathy and retinal vascular changes

New sub-subcategory	H35.08	Macular telangiectasia	
New sub-sub-subcategory	H35.080	Macular telangiectasia, Type 1	
Add		Congenital macular telangiectasia	
New code		H35.0801	Macular telangiectasia, Type 1, right eye
New code		H35.0802	Macular telangiectasia, Type 1, left eye
New code		H35.0803	Macular telangiectasia, Type 1, bilateral
New code		H35.0809	Macular telangiectasia, Type 1, unspecified eye
New sub-sub-subcategory	H35.081	Macular telangiectasia, Type 2	
New code		H35.0811	Macular telangiectasia, Type 2, right eye
New code		H35.0812	Macular telangiectasia, Type 2, left eye
New code		H35.0813	Macular telangiectasia, Type 2, bilateral
New code		H35.0819	Macular telangiectasia, Type 2, unspecified eye
New sub-sub-subcategory	H35.088	Other macular telangiectasia	
Add		Macular telangiectasia, Type 3	
New code		H35.0881	Other macular telangiectasia, right eye
New code		H35.0882	Other macular telangiectasia, left eye
New code		H35.0883	Other macular telangiectasia, bilateral
New code		H35.0889	Other macular telangiectasia, specified eye

New sub-sub-subcategory	H35.089	Unspecified macular telangiectasia
Add		Macular telangiectasia NOS
New code	H35.0891	Unspecified macular telangiectasia, right eye
New code	H35.0892	Unspecified macular telangiectasia, left eye
New code	H35.0893	Unspecified macular telangiectasia, bilateral
New code	H35.0899	Unspecified macular telangiectasia, unspecified eye

Medetomidine Withdrawal Syndrome

This topic was presented at the September 2025 ICD-10 Coordination and Maintenance meeting. Based on public comments, revisions have been made for reconsideration.

Medetomidine is an alpha-2-agonist veterinary sedative medication that has been detected alongside illicitly manufactured fentanyl (IMF) with serious clinical consequences for people who use drugs (PWUD).[1–3] In May 2025, the Centers for Disease Control and Prevention (CDC) published two Morbidity and Mortality Weekly Reports (MMWR) characterizing the emerging syndrome of medetomidine withdrawal.[1,2] This syndrome is characterized by severe autonomic dysfunction, such as severe hypertension and tachycardia, as well as nausea, vomiting, tremor, and altered mental status.[1,2] Almost all patients suspected of having Medetomidine Withdrawal Syndrome required admission to an intensive care unit and nearly a quarter were intubated.[1,2] Accurate diagnosis and documentation of Medetomidine Withdrawal Syndrome is fundamental to improving treatment and health outcomes for PWUD, as well as monitoring the clinical implications of the introduction of medetomidine to IMF, but is not yet available. This proposal addresses the need for a diagnostic code for Medetomidine Withdrawal Syndrome.

Medetomidine is a sedative medication, but characterization of Medetomidine Withdrawal Syndrome does not align with diagnostic criteria for sedative withdrawal. This may be because sedative withdrawal is currently characterized by GABA-agonists, and medetomidine is an alpha-2-agonist. Withdrawal from GABA-agonists, such as benzodiazepines and alcohol, typically presents as soon as 6 hours after last use, but Medetomidine Withdrawal Syndrome can present as soon as 1-2 hours after last use.[4–7] In addition, Medetomidine Withdrawal Syndrome has been characterized as having a rapid progression that is distinct from other withdrawal syndromes.[8,9] Diagnostic criteria for sedative withdrawal includes developing two or more of the following cessation or reduction in use: autonomic hyperactivity, hand tremor, insomnia, nausea or vomiting, transient hallucinations or illusions, psychomotor agitation, anxiety, or grand mal seizures.[10] The coding note in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, uses the ICD 10-CM code F13.239 for sedative withdrawal without perceptual disturbances, which is the closest diagnostic code for Medetomidine Withdrawal Syndrome but is not appropriate due to the ways this differs from other sedative withdrawal.[10] First, Medetomidine Withdrawal Syndrome has not been associated with seizure activity.[1–3,11] Second, Medetomidine Withdrawal Syndrome has been associated with altered mental status that may be secondary to Posterior Reversible Encephalopathy Syndrome (PRES), but has not been associated with hallucinations or illusions.[1,3] Third, Medetomidine Withdrawal Syndrome has been associated with end organ damage, such as PRES and Non-ST Elevation Myocardial Infarction, in the setting of severe hypertension which is not a characteristic of other sedative withdrawal syndromes. Lastly, the tremor associated with Medetomidine Withdrawal Syndrome has not been

described as "hand tremor".[1–3] Withdrawal is a criterion for diagnosing substance use disorders.[10] Inaccurate coding of Medetomidine Withdrawal Syndrome may lead to inaccurate diagnosis of substance use disorders and limit the ability of clinicians to provide appropriate treatment.

Medetomidine was first detected in Philadelphia's illicit fentanyl supply in May, 2025, and recognized by the Philadelphia Department of Public Health (PDPH) to be associated with a severe withdrawal syndrome in December, 2025.[3,12] Since the introduction of medetomidine to Philadelphia's IMF supply the number of people presenting to the emergency department for withdrawal has more than doubled.[13] The efficacy of withdrawal management in Philadelphia's health systems has been shown to decrease with the introduction of medetomidine, which has required updates to withdrawal protocols to effectively treat Medetomidine Withdrawal Syndrome.[14] For example, new protocols use therapies not previously used in withdrawal treatment and novel strategies such as simultaneous initiation of 3 alpha-agonist pathways, increasing the ceiling dose of dexmedetomidine infusion, and using dopamine antagonists for treating intractable nausea and vomiting not responsive to typical therapies.[11] Using approaches that have been historically used for sedative withdrawal from GABA-agonists have been shown to have a limited role in treating Medetomidine Withdrawal Syndrome.[1,2,11,14] Accurate diagnostic coding for Medetomidine Withdrawal Syndrome is critically needed to continue to monitor changes in withdrawal presentation and improve treatment protocols.

Prior to medetomidine being detected, xylazine was the most prevalent alpha-2-agonist veterinary sedative medication in Philadelphia's illicit fentanyl supply. In 2023, nearly 100% of illicit fentanyl in Philadelphia contained xylazine.[15] At the end of 2024, xylazine was present in 30-40% of illicit fentanyl and medetomidine was present in over 70% of illicit fentanyl.[3,16] Medetomidine is rapidly proliferating across the United States, and is present in the illicit fentanyl supply in several states.[17] Accurate diagnostic coding for Medetomidine Withdrawal Syndrome is fundamental to provide appropriate clinical care for PWUD, and provides a critical public health tool for surveillance of clinical implications of a changing IMF supply in the United States.

Daniel Teixeira da Silva, MD, MSHP of the Philadelphia Department of Public Health's Division of Substance Use Prevention and Harm Reduction Division is requesting the following tabular modifications for monitoring and tracking Medetomidine Withdrawal Syndrome.

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TABULAR MODIFICATIONS

F13 Sedative, hypnotic, or anxiolytic related disorders
F13.2 Sedative, hypnotic, or anxiolytic dependence with withdrawal
F13.23 Sedative, hypnotic, or anxiolytic dependence with withdrawal

New code — F13.238 Sedative, hypnotic, or anxiolytic dependence with withdrawal with other disturbances

(The following new codes are approved and scheduled for October 1, 2026, implementation)

T65 Toxic effect of other and unspecified substances
The appropriate 7th character is to be added to each code from category T65
A-initial encounter
D-subsequent encounter
S-sequela

T65.8 Toxic effect of other specified substances

New sub-subcategory — T65.85 Toxic effect of Medetomidine

Add — Use additional code to identify associated manifestations, such as:
bradycardia NOS (R00.1)
somnolence, stupor and coma (R40)

New code — T65.851 Toxic effect of medetomidine, accidental (unintentional)

Add — Toxic effect of medetomidine NOS

New code — T65.852 Toxic effect of medetomidine, intentional self-harm

New code — T65.853 Toxic effect of medetomidine, assault

New code — T65.854 Toxic effect of medetomidine, undetermined

Metabolic dysfunction-and alcohol associated liver disease

This topic was presented at the September 2025 ICD-10 Coordination and Maintenance meeting. Based on public comments, revisions to the proposal have been made for reconsideration.

An addendum was proposed at the March 2024 Coordination and Maintenance meeting to add two new inclusion terms for existing nonalcoholic liver disease classifications, creating inclusion terms and indexing to support both MASH (metabolic dysfunction-associated steatohepatitis) and MASLD (metabolic dysfunction-associated steatotic liver disease).

One of the newly adopted terms from the global NAFLD nomenclature steering committee was not included: Metabolic dysfunction- and alcohol-associated liver disease (MetALD). MetALD applies to patients with liver steatosis, at least one metabolic risk factor, and a history of moderate alcohol intake. This category recognizes that liver steatosis can result from both metabolic factors and alcohol consumption. Defined as MASLD with moderate alcohol consumption (30-60 g/day for men and 20-50 g/day for women), MetALD currently lacks an ICD-10-CM code due to its recent identification. The consensus suggests coding MetALD using the ICD-10-CM for either MASLD or alcoholic liver disease (ALD), depending on the predominant factor in each case.

The Permanente Federation is requesting the following tabular modification to differentiate specific types of liver diseases based on the new nomenclature.

TABULAR MODIFICATIONS

K70 Alcoholic liver disease

New code
Add
K70.8 Other alcoholic liver disease
Metabolic dysfunction- and alcohol associated liver disease (MetALD)

K76 Other diseases of liver

K76.0 Fatty (change of) liver, not elsewhere classified
Nonalcoholic fatty liver disease (NAFLD)
Metabolic dysfunction-associated steatotic liver disease (MASLD)

Neonatal Supraventricular Tachycardia

NCHS received a request from a HIM professional from Mercy Medical Center, for a unique code for Neonatal Supraventricular Tachycardia (NSVT).

Neonatal supraventricular tachycardia is a serious and most common arrhythmia encountered in the newborn that is characterized by an abnormally rapid heart rate originating above the ventricles. Neonatal supraventricular tachycardia typically presents within the first few days or weeks of life, can cause symptoms ranging from subtle to severe, making prompt diagnosis and management critical to prevent complications.

Neonatal supraventricular tachycardia is more severe than just mild tachycardia. This condition will often be evaluated with ECG/ECHO, may require escalated interventions (more than mild tachycardia) and has the potential for future healthcare work up or needs.

The heart rate in neonatal supraventricular tachycardia generally exceeds 220 beats per minute, often with a narrow QRS complex and a regular rhythm. Infants may present with signs such as irritability, poor feeding, lethargy, or even respiratory distress. In some cases, the tachycardia may be asymptomatic and discovered incidentally during routine examinations. The rapid heart rate can compromise cardiac function and diminish cardiac output, potentially leading to congestive heart failure if left untreated.

NSVT is frequently hard to manage, requiring pharmacologic and occasionally interventional ablation. Furthermore, even though it begins in the neonatal period it frequently persists well beyond 28 days. Also, in can occur *in utero,* leading to fetal cardiac failure and hydrops fetalis.

Existing ICD-10-CM code P29.11, Neonatal tachycardia, does not fully represent the clinical severity of the neonate when supraventricular tachycardia is present, which may represent a congenital issue and have additional future healthcare needs

The request has been reviewed and supported by the American Academy of Pediatrics.

TABULAR MODIFICATIONS

P29 Cardiovascular disorders originating in the perinatal period
Excludes2: congenital malformations of the circulatory system (Q20-Q28)

Add — Code also, specified type of tachycardia, if known (I47.1- , I47.2-., I47.9)

P29.1 Neonatal cardiac dysrhythmia
P29.11 Neonatal tachycardia
P29.12 Neonatal bradycardia
New code — P29.13 Neonatal supraventricular tachycardia

Oral Epithelial Dysplasia

Oral epithelial dysplasia is a histopathologic diagnosis indicating disordered epithelial maturation and cellular atypia within the oral mucosa. It is considered a *precancerous lesion* with the potential to progress to oral squamous cell carcinoma if left untreated. Dysplasia is typically graded as mild, moderate, or severe based on architectural and cytologic changes.

Oral dysplasia warrants its own diagnosis because its risk of malignant transformation is real, measurable, and supported by decades of clinical evidence. Mild dysplasia carries an approximate 5–10% risk of progressing to oral squamous cell carcinoma, moderate dysplasia 10–15%, and severe dysplasia or carcinoma in situ up to 30–40%. Erythroplakic lesions containing dysplasia have even higher transformation potential. Without appropriate diagnostic recognition, these high-risk patients may not receive the level of monitoring or intervention required to prevent cancer. Treatment depends on severity and may include surgical excision, laser ablation, or close surveillance with regular follow-up.

Currently, oral epithelial dysplasia is often inaccurately coded under nonspecific categories such as K13.79 (Other lesions of oral mucosa) or K13.29 (Other disturbances of oral epithelium), which fail to distinguish true dysplastic, precancerous changes from benign or reactive lesions.

This lack of specificity leads to a perception of these findings as low risk, resulting in treatment delays. Establishing a distinct diagnosis for oral epithelial dysplasia is essential, as it enables clinicians to identify and treat a verified precancerous condition early, ensuring patients receive appropriate care, including medically necessary biopsies, excisions, and close surveillance.

Early recognition prevents progression of invasive oral cancer, avoiding far more complex interventions such as composite resections, free flap reconstruction, neck dissection, radiation therapy, tracheostomy, and long-term rehabilitation. A formal diagnostic category for dysplasia is therefore both medically necessary and supports timely treatment, and improved patient outcomes.

Historically, oral epithelial dysplasia may have been grouped with broader mucosal lesions due to variability in diagnostic criteria and lack of standardized terminology. However, recent advances in histopathologic grading and consensus guidelines now support consistent identification of dysplasia as a distinct entity.

A unique ICD-10-CM also supports standardized surveillance, coding accuracy, and quality tracking. It allows consistent documentation and data collection on recurrence and malignant transformation. This level of specificity aligns with major national and international standards, National Comprehensive Cancer Network guidelines, and the recommendations of the American Academy of Oral & Maxillofacial Pathology. Recognizing oral dysplasia as its own diagnosis is therefore aligned with established cancer-prevention frameworks.

There is growing consensus among oral pathology and maxillofacial surgery communities that a unique ICD-10-CM code would adequately capture the clinical significance of oral epithelial

dysplasia. The condition's role as a precursor to malignancy warrants distinct recognition. The American Association of Oral and Maxillofacial Surgeons (AAOMS)Committee on Healthcare Policy, Coding and Reimbursement has acknowledged this gap and supports the development of a standalone code to improve diagnostic clarity and coding accuracy.

Establishing a unique ICD-10-CM code for oral epithelial dysplasia, particularly hyperkeratosis associated with benign dysplasia, would enhance diagnostic specificity and support clinical research and epidemiologic tracking while aligning coding with current clinical practice and pathology standards. The ICD-10-CM already sets a precedent for distinct, severity-based codes for dysplastic conditions, as seen with vaginal dysplasia classified under N89.0 (mild), N89.1 (moderate), and N89.2 (severe). Therefore, creating a dedicated category for oral epithelial dysplasia with similar severity-stratified codes is essential for diagnostic clarity and cancer prevention data tracking. This change would also improve communication among providers and public health entities regarding the management of precancerous oral conditions.

This proposal has been reviewed and submitted by Kelly Zak on behalf of the American Association of Oral and Maxillofacial Surgeons (AAOMS) This proposal has also been reviewed and is supported by the American Dental Association Council.

Resources

- Springer Nature Link | Oral Epithelial Dysplasia: Review – Current Surgery Reports
- National Library of Medicine | Oral Epithelial Dysplasia

TABULAR MODIFICATIONS

	K13 Other diseases of lip and oral mucosa
	K13.2 Leukoplakia and other disturbances of oral epithelium, including tongue
	K13.21 Leukoplakia of oral mucosa, including tongue
	K13.22 Minimal keratinized residual ridge mucosa
	K13.23 Excessive keratinized residual ridge mucosa
	K13.24 Leukokeratosis nicotina palati
New sub-subcategory	K13.25 Oral epithelial dysplasia
New code	K13.251 Oral epithelial dysplasia, mild Low grade
New code	K13.252 Oral epithelial dysplasia, moderate
New code	K13.253 Oral epithelial dysplasia severe High grade
New code	K13.259 Oral epithelial dysplasia, unspecified
	K13.29 Other disturbances of oral epithelium, including tongue

Organizing Principles for Classification of Ultra-rare and Genetic Conditions

The CDC's National Center for Health Statistics (NCHS) is seeking input on organizing principles for classification of ultra-rare conditions and conditions with gene-level specificity.

The clinical content in ICD-10-CM is not exhaustive as it was designed to represent broad characterizations to organize and aggregate clinical morbidity data in a consistent way. Therefore, ICD-10-CM does not include unique codes for every ultra-rare or rare disease. ICD-10-CM constituents have requested guidance on what is generally too rare for unique classification.

In addition, ICD-10-CM does not currently include a standardized approach to classify genetic conditions with gene-level specificity, many of which are ultra-rare or rare. In response to proposals for new codes for genetic conditions, with and without unique codes to identify specific gene names, commenters have repeatedly requested that we develop an approach to address genetic specificity in a scalable and sustainable manner.

For these reasons, **current proposals related to ultra-rare conditions and conditions with gene-level specificity are on hold and the CDC/NCHS is committed to establishing principles to ensure these proposals, and future related submissions, can move forward efficiently and effectively.**

The Current State
The ICD-10-CM morbidity classification format and structure is based on organizing principles that address certain classification features. For example, it includes an organizing schema to classify poisonings or adverse effects of drugs and chemicals in the injury chapter based on what the substance is, what it was used for, and the presenting circumstance (whether it was an adverse event or poisoning and the intent). These factors guide maintenance; as new medications are developed for example, they can be placed in the classification based on these same factors.

Presently, there are no organizing factors defined for genetic conditions that would determine how genetic susceptibility to a disease, genetic disorders, or specificity of pathogenic gene variants are consistently represented and reported throughout ICD-10-CM. Currently, genetic conditions and related genetic features may be found in ICD-10-CM in multiple ways, including within chapter-specific codes, in genetic disorder codes in Chapter 17, or in category Z15, Genetic susceptibility to disease.

The Problem
The growing number of genetic-related codes in ICD-10-CM and increasing requests for gene-level specificity in proposals for unique ICD-10-CM codes raises several concerns.

Complexity and rate of change: A pathogenic gene variant may be associated with multiple rare diseases, thus gene-disease relationships require clinical expertise to evaluate the clinical validity of the available evidence at a point in time.[1] Furthermore, due to the pace at which such evidence

is evolving, it may be misleading to tie a specific gene to a condition in a pre-coordinated (combination) ICD-10-CM code.[2]

Lack of standardization: There are multiple systems available to organize and identify genotypes and associated phenotypes. As a result, there is the potential for conflicting, overlapping, or redundant data on disease-gene relationships.[3]

Delay and ambiguity: Without organizing principles, ICD-10-CM genetic code requests must be investigated in a lengthier case-by-case approach which can cause delays and could lead to inadvertent inconsistencies or ambiguity[4] among the codes in the ICD-10-CM code set.

Data inconsistencies: An organizational strategy is necessary for consistent reporting. It is important to define how genetic conditions, and related manifestations, will be represented. Without such, confusion and misinterpretation lead to inaccurate coding. As a result, coded data are inconsistent and difficult to interpret with confidence.

Structural constraints: There are several thousand single gene disorders, gene mutations may cause more than one disorder, and there are many gene-disease relationships yet to be discovered.[5] ICD-10-CM codes are constrained to a maximum of seven characters, thus continuing to add pre-coordinated (combination) codes with specific gene names without a defined approach is not sustainable.

Commenter Input
The CDC/NCHS is requesting input to guide development of principles that will provide clarification on the ICD-10-CM scope and process for incorporating, applying, and maintaining codes for genetic conditions. One approach to classifying genetic conditions might be to represent known disorders in ICD-10-CM with additional gene-specific attributes represented in a separate data standard that is updated on an ongoing basis and curated with direct input from geneticists (such as OMIM, HUGO, Mondo, Orphanet). In contrast, an entirely different approach is to strategically expand ICD-10-CM to include conditions with known pathogenic gene variants specified, perhaps via an organizing genetic schema analogous to the table of drugs and chemicals. There are no doubt other potential approaches, each with differing advantages and disadvantages, to achieve the necessary ICD-10-CM data for statistical and administrative purposes. Thus, the CDC/NCHS is seeking input.

Next Steps
The CDC/NCHS invites commenters to provide input on this topic in the 60-day comment period following this March 2026 ICD-10 Coordination and Maintenance Committee meeting. Please submit comments to this inbox: nchsicd10cm@cdc.gov. We will use this input and conversations with subject matter experts to draft organizing principles for presentation at the September 2026 meeting. We hope to use such principles to advance the current ultra-rare and gene-specific proposals that are on hold and to guide proposal submissions, starting next year.

Please submit your comments on what factors need to be defined in a standardized approach to classify rare or ultra-rare conditions and conditions with gene-level specificity.

In addition, we welcome input for example on the following questions and additional thoughts on how to strategically and consistently classify ultra-rare and gene-level specificity in ICD-10-CM.

- What criteria should be used to determine whether unique codes should be created for rare or ultra-rare diseases? What factors, in addition to prevalence or incidence, are relevant?
- What level of evidence or confidence in a gene-disease relationship is needed to develop a pre-coordinated ICD-10-CM code that supports concept permanence in the code set over time?
- How do genetic susceptibility codes fit with the genetic disease codes? In what circumstances should these codes be reported, alone or together?
- How should chromosomal disorders be classified distinct from or consistent with genetic disorders?
- What is the best way to align ICD-10-CM development with existing gene-disease curation efforts in the industry?

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Pneumothorax that occurs after CPR

Cardiopulmonary resuscitation (CPR) involves artificially circulating the blood through repeated sternal compression to rescue the patient from cardiac arrest. This can exert excessive physical force on the chest of the patient which can result in chest injuries. Studies have reported that CPR-related chest injuries range from non-complicated skeletal injuries to life-threatening injuries, such as mediastinal hemorrhage or large pneumothorax. [1-4]

This proposal was presented at the September 2025 ICD-10 Coordination and Maintenance Meeting. In response to public comments, changes have been made and noted in **bold.**

Wijck, et al. (2024), performed a systematic review and meta-analysis on the prevalence of rib fractures and other injuries resulting from CPR. They performed database searches to identify studies reporting on CPR-related injuries in patients who underwent chest compression for a non-traumatic cardiopulmonary arrest and included 74 studies encompassing a total of 16,629 patients. They found CPR-related injury documented in 60% of patients, with fractures identified as the most common injury.[2]

Pneumothorax resulting from CPR-related rib fractures is a known risk that may result from this life-saving procedure. In one hospital study of 237 cases, pneumothorax occurred in about 11% of patients after CPR. In the same study, the condition was more common - about 23% - in patients with history of obstructive lung disease.[3] In a similar study by another hospital, the most common thoracic injuries found in patients after CPR were those associated with rib and sternal fractures, including pneumothorax which was found in 10% of the patients.[4]

A unique code for fractures due to CPR (M96.A, Fracture of ribs, sternum and thorax associated with compression of the chest and cardiopulmonary resuscitation) was implemented in ICD-10-CM in October 2022. However, there is currently no unique code to represent pneumothorax due to CPR. The ICD-10-CM code currently being used for this, J95.811 (postprocedural pneumothorax), is not specific to CPR-related incidence of pneumothorax.

The requesting facility recently implemented a dedicated sudden cardiac arrest (SCA) - whole-body computed tomography (WBCT) protocol to evaluate SCA patients with return of spontaneous circulation (ROSC) following cardiopulmonary resuscitation (CPR). They seek a new code to allow for accurate reporting of CPR-related chest injuries, including not only fractures but also pneumothorax.

A unique code to identify CPR-related pneumothorax, as distinct from other causes, is important for clinical care and quality reporting. The proposed new code for Pneumothorax due to rib fracture associated with chest compression and cardiopulmonary resuscitation will help accurately report the clinical picture and promote better data mining.

This proposal was submitted by Mercy Health Systems.

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TABULAR MODIFICATIONS

J93 Pneumothorax and air leak

J93.8 Other pneumothorax and air leak

New code **J93.84 Pneumothorax associated with chest compression and cardiopulmonary resuscitation**

Add **Excludes1: Postprocedural pneumothorax (J95.811)**

J95 Intraoperative and postprocedural complications and disorders of respiratory system, not elsewhere classified

J95.8 Other intraoperative and postprocedural complications and disorders of respiratory system, not elsewhere classified

J95.811 Postprocedural pneumothorax

Add **Excludes 1: Pneumothorax associated with chest compression and cardiopulmonary resuscitation (J93.84)**

Add **Code also any associated rib fracture with chest compression and cardiopulmonary resuscitation (M96.A-)**

Post-Intensive Care Syndrome (PICS)

Post-intensive care syndrome (PICS) refers to new or worsening cognitive, mental health, or physical impairments that arise after critical illness and persist for at least three months following acute care hospitalization[1-2] This condition can affect patients aged one year and older and must be differentiated from issues related to chronic diseases or the acute condition that necessitated intensive care.[2-4]

The term PICS was introduced in 2010 by clinicians and researchers at the Society of Critical Care Medicine (SCCM) stakeholders' conference to describe impairments such as executive dysfunction, anxiety, depression, post-traumatic stress disorder (PTSD), and intensive care unit(ICU)-acquired weakness following critical illness.[1] Risk factors for PICS include high disease severity (e.g., sepsis, acute respiratory distress syndrome), ICU delirium, benzodiazepine therapy, and ICU length of stays exceeding four days. [2,4-6]

Though ICD-10-CM codes exist for individual conditions like fatigue, anxiety, and PTSD, none reflect the ICU-related etiology. Without a unified code, the national and global PICS prevalence is difficult to determine. Most of the studies available have been conducted in single centers or health systems. A recent meta-analysis found a pooled prevalence of 54.35%.[7] Co-occurring PICS problems (i.e., two or more domains- cognitive, mental, and/or physical health) are present in 25% of critical illness survivors at three months and 21% at twelve months.[8] A 2019 meta-analysis of 52 studies reported just 36% of survivors returned to work at 1-3 months, 60% at 12 months, and 68% at 42-60 months.[9] Compared to matched non-ICU patients, ICU survivors had over 2.5x more new disease episodes within 1 year of discharge.[10] From years 2-5, they averaged 3.86 new episodes/year vs. 2.86 in the comparison group, suggesting a greater need for outpatient medical care years after their ICU admission that can impact survivors' ability to return to work.[10]

An ICD-10-CM code for PICS is needed to: enable epidemiological investigation of PICS and allow providers to screen, diagnose, and coordinate care. [11] In a 2020 SCCM consensus conference, clinicians and researchers agreed upon serial assessments that should be started 2-4 weeks post-discharge with survivors to establish baseline post-acute functionality after critical illness. However, providers are limited in conducting serial PICS assessments and treatment without a PICS code, thus hindering the adoption of these recommendations. [1, 12]

This proposal has been submitted by Brian C. Peach, PhD, RN, CCRN-K of University of Central Florida, Jennifer MacDonald, LMSW, of Vanderbilt University Medical Center's Critical Illness, Brain Dysfunction and Survivorship Center (VUMC CIBS), Paula Blonski, President of the ARDS Alliance on behalf of critical illness survivors, VUMC CIBS Center's PICS Advocacy Network and the ARDS Alliance.

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TABULAR MODIFICATIONS

T88 Other complications of surgical and medical care, not elsewhere classified

T88.8 Other specified complications of surgical and medical care, not elsewhere classified

New code T88.81 Post-intensive care syndrome

Add PICS

Add Code also any associated:

Add delirium due to known physiological condition (F05)
Add depressive episode (F32.-)
Add generalized anxiety disorder (F41.1)
Add intercranial injury (S06.-)
Add post-traumatic stress disorder (PTSD) (F43.1-)
Add sequelae of cerebrovascular disease (I69.-)

New code T88.89 Other specified complications of surgical and medical care

Postprocedural open deep wound without disruption

This topic was presented at the September 2024 and September 2025 ICD-10 Coordination and Maintenance meeting. Based on public comments, revisions to the proposal have been made for reconsideration. Changes are indicated in **BOLD**.

In certain circumstances, the surgical wound is temporarily left open at the end of a procedure. For some scenarios, an abdominal incision may intentionally be left open at the completion of surgery to allow an abdominal infection to resolve, and to facilitate a "second look" laparotomy at a later time. Other scenarios involve open chest, open fractures, and others where there is a high risk of postoperative compartment syndrome or deep surgical site infection. The initial operation may have occurred in the same hospital (i.e., not present on admission) or at a different hospital (i.e., present on admission). Having an open surgical wound after a prior operation is a high risk situation that strongly affects resource use and care in the hospital, at least until the wound is closed. This is an important, clinically significant concept, but there currently is no way to account for it in ICD-10-CM.

The Agency for Healthcare Research and Quality (AHRQ) is requesting the following tabular modifications to report intended postprocedural state wherein the surgical wound is deliberately left open, to be closed at a later time.

TABULAR MODIFICATIONS

T81 Complications of procedures, not elsewhere classified

T81.3 Disruption of wound, not elsewhere classified
Disruption of any suture materials or other closure methods

Add — Excludes1: postprocedural open surgical wound (Z98.88-)

Z98 Other postprocedural states

Z98.8 Other specified postprocedural states

New sub-subcategory — Z98.88 **Postprocedural intentionally open surgical wound**

New code — Z98.880 **Postprocedural intentionally open abdomen**
Add — Delayed abdominal closure
Add — Intentional delayed closure of abdominal surgical wound

Add		**Open abdomen**
Add		Postprocedural temporary open abdomen
New code	Z98.881	**Postprocedural intentionally open chest**
Add		Delayed chest closure
Add		Intentional delayed closure of chest surgical wound
Add		**Open chest**
Add		Postprocedural temporary open chest
New code	Z98.888	**Other postprocedural intentionally open surgical wound**
Add		Other delayed surgical wound closure
Add		Other intentional delayed closure of surgical wound
Add		Other postprocedural temporary open surgical wound
New code	Z98.889	**Postprocedural intentionally open surgical wound, unspecified**
Add		Intentional delayed surgical wound closure NOS

Progressive Myopia

Myopia is one of the most common ocular conditions worldwide and its prevalence has been increasing in both children and adults. Global projections suggest that nearly half of the world's population could be myopic by 2050, with a rise in high myopia and associated visual impairment from abnormal enlargement of the eye.[1] Population-based data from the United States demonstrate growing prevalence of myopic refractive error across age groups.[2] Higher degrees of myopia are associated with structural changes of the entire eye and a substantially increased risk of sight-threatening complications including myopic maculopathy, choroidal neovascularization, retinal detachment, glaucoma, and cataract. Pathologic myopia is estimated to affect 1–3% of the global population with its prevalence expected to rise as myopia prevalence increases.[3]

This request is submitted in response to the growing body of literature evaluating the use of optical and pharmacologic strategies to slow myopia progression in children (termed myopia control). These publications include randomized clinical trials demonstrating varied degrees of efficacy of dual-focus soft contact lenses,[4] specialized spectacle lens designs,[5] and low-concentration atropine[6] designed to slow myopia progression. There has been a large uptake of such treatment strategies in the US and around the world. There are now two FDA-authorized treatments in the US. For these reasons there is a public health need to understand how many eligible children there are in United States and how many are being treated.

Currently, myopia as a refractive error is coded in ICD-10-CM using H52.1 Myopia, in the "Disorders of refraction and accommodation" chapter. This code describes the refractive state of the eye but does not identify whether the myopia is progressing, and which children could benefit from myopia control treatment to slow progression. While myopia progression indicates that there are increases in the refractive error and the eyeglasses prescription, it is more serious related to abnormal eye growth including abnormal changes in the structure of globe and retina. Codes in ICD-10-CM reporting ocular changes associated with abnormal myopia progression are included in section H44.2A through H44.2E Degenerative Myopia with the most severe complications including retinal detachment, macular hole and neovascularization. There is no code in either section of ICD-10-CM, H44.2- or H52.1, noting whether there is ongoing progression of the myopia.

The American Academy of Ophthalmology is requesting the following tabular modifications to facilitate accurate data collection of patients with progressive myopia and progressive high myopia at risk for degenerative complications of the retina.

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TABULAR MODIFICATIONS

H44 Disorders of globe
H44.2 Degenerative myopia
Malignant myopia

New sub- subcategory	H44.2F	Degenerative myopia with progression
Add		Progressive degenerative myopia
Add		Progressive high myopia
Add		Progressive myopia
New code	H44.2F1	Degenerative myopia with progression, right eye
New code	H44.2F2	Degenerative myopia with progression, left eye
New code	H44.2F3	Degenerative myopia with progression, bilateral
New code	H44.2F9	Degenerative myopia with progression, unspecified eye

H52 Disorders of refraction and accommodation
H52.1 Myopia
Revise Excludes~~1~~2: degenerative myopia (H44.2-)

Sepsis

There was a proposal to make updates to ICD-10-CM presented September 2019 related to the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3) from 2016.[1] However, proposed changes did not go forward at that time based on negative comments received. Some concerns were related to a need for sepsis criteria for children, and that was addressed in the Phoenix criteria.[2] From the original Sepsis 3 definition, "Sepsis should be defined as life-threatening organ dysfunction caused by a dysregulated host response to infection."[1] The organ dysfunction can be represented by an increase in the Sequential [Sepsis-related] Organ Failure Assessment (SOFA) score, with components for respiration, coagulation, liver, cardiovascular, central nervous system, and renal.[1]

It is proposed to create an expansion of sepsis codes, to identify (by organism) sepsis and impending sepsis, with at this time a single example given. Also, it is proposed to create codes to identify the organ dysfunction present, while also deleting the severe sepsis code and terminology, based on the definitional changes in Sepsis 3.

Multiple sources have provided input related to sepsis. This proposal is a draft that shows potential examples of how modifications may be handled with public input requested prior to an expected more complete proposal presentation anticipated at a future Coordination and Maintenance meeting.

References
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TABULAR MODIFICATIONS

Note: this proposal shows expansion of a single code below in category A41, but it is anticipated that this same approach would need to be applied across about 40 codes for sepsis in total.

	A41	Other sepsis	
Revise	A41.9	Sepsis <u>and impending sepsis</u>, unspecified organism	
New code	A41.91	Sepsis, unspecified organism	
Add		Use additional code for organ dysfunction, such as:	
Add		acute kidney failure (N17.-)	
Add		altered mental status (R41.82)	
Add		other organ dysfunction associated with sepsis (R65.2-)	
Add		septic shock (R65.21)	
Add		tachypnea (R06.82)	

New code	A41.92	Impending sepsis, unspecified organism	
Add		Infection with systemic inflammatory response syndrome (SIRS)	
Add		Infection with positive sepsis diagnostic aid	
	R65	Symptoms and signs specifically associated with systemic inflammation and infection	
Revise	R65.2	Organ dysfunction associated with sepsis ~~Severe sepsis~~	
Delete		~~R65.20 Severe sepsis without septic shock~~	
Revise	R65.21	~~Severe sepsis with septic~~ Septic shock	
New sub-subcategory	R65.22	Other specific organ dysfunction associated with sepsis	
New code		R65.220	Respiratory dysfunction associated with sepsis
New code		R65.221	Coagulation dysfunction associated with sepsis
New code		R65.222	Liver dysfunction associated with sepsis
New code		R65.223	Cardiovascular dysfunction associated with sepsis
Add			Excludes1: septic shock (R65.21)
New code		R65.224	Central nervous system dysfunction associated with sepsis
New code		R65.225	Renal dysfunction associated with sepsis

Spontaneous Coronary Artery Dissection

Spontaneous coronary artery dissection (SCAD) involves a tear in one of the coronary arteries. The spectrum ranges from intramural hematoma to intimal rupture and false lumen formation.[1] SCAD is differentiated from coronary artery dissection from other causes such as trauma or coronary artery interventions (iatrogenic causes). [1] SCAD can be a cause of a type 2 myocardial infarction (MI), although it should be differentiated from type 1 MI related to ruptured atherosclerotic plaque as well as other coronary atherosclerosis.[2] Also, SCAD is the leading cause of acute coronary syndrome in young women, including peripartum women, without any cardiovascular risk factors. [1]

Spontaneous Coronary Artery Dissection is a well defined, non-atherosclerotic, non-traumatic cause of acute coronary syndrome (ACS) resulting from the formation of an intramural hematoma or an intimal tear in a coronary artery. It predominantly affects women aged 30–60, including a strong association with pregnancy and postpartum physiology. The creation of a new ICD-10-CM code for spontaneous coronary artery dissection (SCAD) has been proposed by SCAD Alliance, a 501c3 founded in 2013 which strives to empower SCAD patients with an accurate diagnosis, superior outcome and answers. Having a specific code for SCAD will enable it to specifically be differentiated from other causes of coronary artery dissection.

References

1. Nepal S, Chauhan S, Bishop MA. Spontaneous Coronary Artery Dissection. StatPearls. NLM, NIH. June 21, 2023. https://www.ncbi.nlm.nih.gov/sites/books/NBK582143/
2. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth Universal Definition of Myocardial Infarction (2018). Circulation 138(20):e618-e651. https://doi.org/10.1161/CIR.0000000000000617

TABULAR MODIFICATIONS

I25 Chronic ischemic heart disease

I25.4 Coronary artery aneurysm and dissection

I25.42 Coronary artery dissection

New code	I25.420	Spontaneous coronary artery dissection
Add		Use additional code, if applicable, for myocardial infarction type 2 (I21.A1)
New code	I25.428	Other coronary artery dissection
Add		Code first, if applicable, underlying cause of dissection
Add		Use additional code, if applicable, for myocardial infarction (I21.-)
New code	I25.429	Coronary artery dissection, unspecified
Add		Use additional code, if applicable, for myocardial infarction (I21.-)

Titanium Dioxide Exposure

Titanium dioxide exposure typically occurs through inhalation, ingestion, injection, skin contact, or absorption in the alimentary tract. Studies show that titanium dioxide can accumulate in the body, particularly in the lungs, alimentary tract, liver, heart, spleen, kidneys and cardiac muscle.

People may be exposed to titanium dioxide through various industrial applications, including building engineering, water and sewage treatment, gas combustion, and its use as an antibacterial material for decontamination. Additionally, it is utilized in the production of fertilizers and pesticides, biomedical applications such as pharmaceuticals and medical devices, and in the food industry for processing and packaging.

In recent years, concerns about titanium dioxide exposure have also extended to military environments. Service members deployed to Iraq and Afghanistan were frequently stationed near open-air burn pits used to dispose of waste materials, resulting in chronic exposure to airborne particulate matter and toxic metals. Titanium-containing particles have been identified in deployment-exposed veterans' lung tissue, raising concern that titanium compounds (including potentially titanium dioxide) may play a role in inhalation-related injury. Documented health effects of titanium dioxide nanoparticle exposure in humans include accumulation in the lungs, liver, heart, and kidneys, oxidative stress and apoptosis in intestinal epithelial cells, DNA damage, and disruption of cellular function in internal organs.

Currently, no ICD-10-CM code exists specifically for exposure to titanium dioxide. A new designated ICD-10-CM code will allow documentation of exposure to titanium dioxide in the electronic health records of active-duty Service Members (Department of Defense, DOD) and Veterans (Department of Veteran Affairs, VA). Documentation of toxic exposures during military service is a high priority for both Departments due to their potential long-term health consequences as well as directives in recent legislation [Promise to Address Comprehensive Toxics (PACT) Act].

Titanium dioxide was the 36th most common hazard (out of 767), based on data pulled January 2023) tracked within the Individual Longitudinal Exposure Record (ILER) which tracks toxic exposures for the DOD and VA.

This proposal, submitted by Laree LaPierre, MPH on behalf of the DOD, VA, and the Office of Federal Electronic Health Record Modernization (FEHRM).

References:

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United States Congress. (2022). Sergeant First Class Heath Robinson Honoring our Promise to Address Comprehensive Toxics Act of 2022. https://www.congress.gov/117/plaws/publ168/PLAW-117publ168.pdf

TABULAR MODIFICATIONS

T56 Toxic effect of metals

T56.8 Toxic effect of other metals

New sub-subcategory	T56.84 Toxic effect of titanium dioxide
New code	T56.841 Toxic effect of titanium dioxide, accidental (unintentional) Toxic effect of titanium dioxide NOS
New code	T56.842 Toxic effect of titanium dioxide, intentional self-harm
New code	T56.843 Toxic effect of titanium dioxide, assault
New code	T56.844 Toxic effect of titanium dioxide, undetermined

Toxic Effects of Hexane

Hexane is a hydrocarbon to which humans are commonly exposed in everyday life and in higher levels in many occupations. Low levels of hexane exposure can occur while fueling a car with gasoline. It is a byproduct of burning plastic water bottles and Styrofoam trays – a common exposure route for military service members who were in close proximity to burn pit smoke and other airborne hazards while serving in Iraq, Afghanistan, Djibouti, Saudia Arabia, UAE, Kuwait, and Qatar (Olsen et al., 2022). Among civilian workers, exposure to n-hexane is common in the manufacturing of leather, glues and adhesives, shoemaking, laboratories, and as an extractant while making cooking oils (Faroon et al, 2024).

Since hexane is highly volatile, quickly evaporating into air, the most common, and most studied, route of exposure is inhalation. Common symptoms include lung injury and airway dysfunction (Olsen et al., 2022). Those exposed through inhalation have also been known to experience peripheral neuropathy (Faroon et al., 2024). This is especially true among occupational exposures but in these cases nerve function can return after the toxic exposure ends (Kutlu et al, 2009.). Other poisoning effects include damage to organs such as the eyes, heart and liver. Long term exposure can cause motor and sensory dysfunction (Zhang et al., 2021).

N-hexane was the 35th most common hazard (out of 767, based on data pulled January 2023) tracked within the Individual Longitudinal Exposure Record (ILER), which tracks toxic exposures for the DOD and VA.

Currently no ICD-10-CM code exists that specifically records exposures to hexane. Documentation of toxic substances and exposures during or associated with military service is a high priority for both the Department of Defense (DOD) and the Department of Veteran Affairs (VA) due to their potential long term health consequences and directives for care of these exposures in recent legislation, namely in the Promise to Address Comprehensive Toxics (PACT) Act of 2022 (United States Congress, 2022).

This proposal, submitted by Laree LaPierre, MPH on behalf of the DOD, VA, and Federal Electronic Health Record Modernization (FEHRM) Office. This new concept will facilitate documentation of hexane exposure in the electronic health records of active-duty service members and Veterans within the DOD and VA, their beneficiaries (family members), as well as the general public who may come in contact with this substance.

References:

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TABULAR MODIFICATIONS

T52 Toxic effect of organic solvents

T52.B Toxic effect of other organic solvents

New sub-subcategory T52.BX Toxic effects of hexane

Use additional code(s) for all associated manifestations, such as:

contact with and (suspected) exposure to other war theater

New code T52.BX1 Toxic effects of hexane, accidental (unintentional)

New code T52.BX2 Toxic effects of hexane, intentional self-harm

New code T52.BX3 Toxic effects of hexane, assault

New code T52.BX4 Toxic effects of hexane, undetermined

Toxic Effect of Iron Oxide

The chemical compound iron oxide is used throughout a variety of occupations, including welding, foundry work, iron and steel manufacturing, mining, and even has biomedical applications for clinical use. US military service members typically face exposure to iron oxide through airborne hazards, including dust storms and burn pits. Exposure to iron oxide fumes can cause metal fume fever. This is a flu-like illness with symptoms of fever and chills, aches, chest tightness and cough. Prolonged or repeated contact can discolor the eyes causing permanent iron staining. Some studies suggest that exposure to iron oxide may cause neurodegeneration or stress to the body's organs.

Iron oxide was the 30th most common hazard (out of 767), based on data pulled January 2023, tracked within the Individual Longitudinal Exposure Record (ILER) which tracks toxic exposures for the Department of Defense (DOD) and Department of Veteran Affairs (VA).

Currently, no ICD-10-CM code exists specifically for exposure to iron oxide. This new concept will allow documentation of exposure to iron oxide in the electronic health records of active-duty service members within the DOD and Veterans within the VA and those in the general public who may become exposed. Documentation of toxic exposures during military service is a high priority for both Departments due to their potential long term health consequences and directives in recent legislation, namely the Promise to Address Comprehensive Toxics (PACT) Act.

A consortium of clinicians, researchers, and advocates propose the following tabular modifications to aid clinical care of affected veterans, advance epidemiological tracking of this exposure and improve health outcomes in all individuals experiencing adverse effects from toxic exposure to iron oxide.

This proposal, submitted by Laree LaPierre, MPH on behalf of the DOD, VA, and the Office of Federal Electronic Health Record Modernization (FEHRM).

References:

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United States Congress. (2022). Sergeant First Class Heath Robinson Honoring our Promise to Address Comprehensive Toxics Act of 2022. https://www.congress.gov/117/plaws/publ168/PLAW-117publ168.pdf

TABULAR MODIFICATIONS

T56 Toxic effect of metals

T56.8 Toxic effect of other metals

New sub-subcategory	T56.83 Toxic effect of iron oxide
New code	T56.831 Toxic effect of iron oxide, accidental (unintentional) Toxic effect of iron oxide NOS
New code	T56.832 Toxic effect of iron oxide, intentional self-harm
New code	T56.833 Toxic effect of iron oxide, assault
New code	T56.834 Toxic effect of iron oxide, undetermined

Toxic Effect of N-Butyl Acetate

N-butyl acetate is an important solvent in the chemical industry, mainly used in paint and coating manufacturing and the lacquer industry. The chemical is rapidly hydrolyzed to n-butanol and acetic acid following inhalation or dermal exposure, with n-butanol responsible for many of its systemic effects. Exposure typically occurs through inhalation of vapors or accidental dermal contact during use as a solvent in industrial or maintenance applications. U.S. military personnel may have been exposed to n-butyl acetate through maintenance of military vehicles, cleaning operations, or from use of paints, varnishes, paint removers, and degreasing products. Acute inhalation exposure in humans has been associated with eye, nose, and throat irritation at concentrations as low as 300 ppm.

N-butyl acetate was the 37th most frequently reported hazard (of 767) based on January 2023 data tracked within the Individual Longitudinal Exposure Record (ILER), which monitors toxic exposures for the Department of Defense (DoD) and the Department of Veterans Affairs (VA).

The addition of a new ICD-10-CM code for n-butyl acetate exposure would allow documentation of this solvent within the electronic health records of active-duty Service Members and Veterans. Tracking this exposure is critical because both Departments are directed under the Sergeant First Class Heath Robinson Honoring Our Promise to Address Comprehensive Toxics (PACT) Act to improve identification and documentation of service-related toxic exposures.

A consortium of clinicians, researchers, and advocates propose the following ICD-10 tabular modifications to improve recognition of health effects linked to n-butyl acetate, support clinical care of affected Veterans, and advance epidemiologic surveillance of exposure-related illness.

This proposal, submitted by Laree LaPierre, MPH on behalf of the DOD, VA, and the Office of Federal Electronic Health Record Modernization (FEHRM).

References:

Segal, D., Bale, A. S., Phillips, L. J., Sasso, A., Schlosser, P. M., Starkey, C., & Makris, S. L. (2020). Issues in assessing the health risks of n-butanol. *Journal of Applied Toxicology, 40*(1), 72–86. https://pmc.ncbi.nlm.nih.gov/articles/PMC9528569/

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United States Congress. (2022). *Sergeant First Class Heath Robinson Honoring our Promise to Address Comprehensive Toxics*

TABULAR MODIFICATIONS

T52 Toxic effect of organic solvents

New subcategory T52.A Toxic effect of n-butyl acetate

New sub-subcategory T52.AX Toxic effect of n-butyl acetate

New code	T52.AX1	Toxic effect of n-butyl acetate, accidental (unintentional) Toxic effect of n-butyl acetate, NOS
New code	T52.AX2	Toxic effect of n-butyl acetate, intentional self-harm
New code	T52.AX3	Toxic effect of n-butyl acetate, assault
New code	T52.AX4	Toxic effect of n-butyl acetate, undetermined

Wolff-Parkinson-White (WPW) syndrome

In Wolff-Parkinson-White (WPW) syndrome an accessory atrioventricular pathway allows conduction to bypass the atrioventricular (AV) node. This activates the ventricles early, which is called preexcitation.[1] It also predisposes patients to tachyarrhythmias and, in certain scenarios, life-threatening ventricular fibrillation. The presence of WPW has specific implications for pharmacologic therapy, particularly in the context of atrial fibrillation.[1-3]

For patients with WPW syndrome who have tachyarrhythmias, usual therapy for controlling the heart rate can instead worsen symptoms.[1] Thus, WPW syndrome requires distinct management strategies and unique treatment considerations, such as avoiding AV nodal blocking agents during atrial fibrillation and certain types of atrioventricular reentrant tachycardia (AVRT). These drugs can accelerate conduction through the accessory pathway, precipitating ventricular fibrillation or hemodynamic instability. Beta blockers can occasionally be used as chronic suppressive therapy of AVRT in low-risk accessory pathways, but this is a clinical decision to be made after consideration of the individual patient and the existing contraindication. Other pre-excitation syndromes do not universally share this contraindication.

Having a specific code for WPW syndrome will enable clinicians, pharmacists, and others to identify at-risk WPW patients when specific medications is being prescribed (such as AV nodal blockers) and assist with support for electronic screening systems that can screen for drug–disease interactions. A distinct code will also enable better clinical decision support tailored specifically to WPW to reduce adverse drug events and provide point-of-care guidance. This will enable differentiation of WPW syndrome from Lown-Ganong-Levine and multiple other accessory pathway disorders, which share the feature of pre-excitation, but differ significantly in clinical management and risk profiles.

A proposal to create a specific code for Wolff-Parkinson-White (WPW) syndrome was received from Sandra K. O'Loughlin, PA-C, MBA; with Pharmacy and Health Technology Solutions at Wolters Kluwer Health.

References

1. UpToDate. (n.d.). Treatment of arrhythmias associated with the Wolff-Parkinson-White syndrome. In *UpToDate*. Retrieved February 18, 2026. https://www.uptodate.com/contents/treatment-of-arrhythmias-associated-with-the-wolff-parkinson-white-syndrome
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TABULAR MODIFICATIONS

I45 Other conduction disorders

I45.6 Pre-excitation syndrome

Delete	~~Accelerated atrioventricular conduction~~	
Delete	~~Accessory atrioventricular conduction~~	
Delete	~~Anomalous atrioventricular excitation~~	
Delete	~~Lown-Ganong-Levine syndrome~~	
Delete	~~Pre-excitation atrioventricular conduction~~	
Delete	~~Wolff-Parkinson-White syndrome~~	

New code	I45.60	Pre-excitation syndrome, unspecified
Add		Accessory atrioventricular conduction
Add		Pre-excitation atrioventricular conduction
New code	I45.61	Wolff-Parkinson-White [WPW] syndrome
Add		Anomalous atrioventricular excitation
New code	I45.69	Other pre-excitation syndrome
Add		Accelerated atrioventricular conduction
Add		Lown-Ganong-Levine syndrome

INDEX MODIFICATIONS

Add	Conduction
Add	- atrioventricular
Add	- - accelerated I45.69
Add	- - accessory I45.60
Add	- - anomalous I45.61
Add	- - pre-excitation I45.60
Add	- enhanced atrioventricular nodal I45.69
	Tachycardia R00.0
Add	- atriofascicular (pathway) I45.69
Add	- Mahaim (fiber) (pathway) I45.69

TABULAR MODIFICATIONS ADDENDA

All approved modifications will be effective October 1, 2027

E28 Ovarian dysfunction
E28.3 Primary ovarian failure
Add Primary ovarian insufficiency

E88 Other and unspecified metabolic disorders
E88.1 Lipodystrophy, not elsewhere classified
E88.12 Generalized lipodystrophy
Revise Berardinelli-~~Siep~~ Seip syndrome

H40 Glaucoma
H40.8 Other glaucoma
H40.84 Neovascular secondary angle closure glaucoma
Code also the underlying condition such as:
Revise central retinal vein occlusion (H34.81-)

I67 Other cerebrovascular diseases
I67.4 Hypertensive encephalopathy
Code also, if applicable, associated hypertensive conditions such as:
Add hypertensive emergency (I16.1)

J96 Respiratory failure, not elsewhere classified
Delete Excludes1: ~~postprocedural respiratory failure (J95.82-)~~
Add Excludes2: postprocedural respiratory failure (J95.82-)

L51 Erythema multiforme
Use additional code to identify associated manifestations, such as:
Revise conjunctival edema (H11.42-)

P29 Cardiovascular disorders originating in the perinatal period
P29.1 Neonatal cardiac dysrhythmia
P29.11 Neonatal tachycardia
Add Code also, specified type of tachycardia, if known (I47.1- , I47.2-.,
I47.9)

R10 Abdominal and pelvic pain
Revise Excludes2: costovertebral (angle) tenderness (R39.85-)

INDEX MODIFICATION ADDENDA

All approved modifications will be effective October 1, 2027

Revise Arachnodactyly ~~- see Syndrome, Marfan~~ R29.898

Foreign body
- entering through
- - natural orifice W44.9
- - - other
Add - - - - sharp object unspecified W44.H0

Fracture, traumatic (abduction) (adduction) (separation) (see also Fracture, pathological) T14.8
- tibia (shaft) S82.20-
- - upper end S82.10-
Revise - - - plateau - see Fracture, tibia, upper end~~, bicondylar~~

Genetic
- susceptibility to disease NEC Z15.89
- - malignant neoplasm Z15.09
Revise - - - small bowel ~~Z15.08~~ Z15.068

Histiocystosis D76.3
Add - lipochrome D71.8

Hyperoxaluria R82.992
Add - secondary E75.54-

Injury
- intracranial (traumatic) (see also, if applicable, Compression, brain, traumatic) S06.9-
Revise - - cerebellar ~~- see also Injury, intracranial, focal brain injury~~ S06.37-
Add - - - contusion S06.37-
Add - - - hemorrhage S06.37-
Add - - - laceration S06.37-

Osteoporosis (female) (male) M81.0
- postmenopausal M81.0
- -with pathological fracture M80.00
Revise - - ilium ~~M80.0A~~ M80.0B
Revise - - ischium ~~M80.0A~~ M80.0B

Syndrome
Revise - Berardinelli-~~Siep~~ Seip E88.12
Revise - ~~u~~Usher Q99.819
Revise - - specified NEC ~~Q99.814~~ Q99.818
Revise - - type 4 ~~Q99.814~~ Q99.818

External Cause of Injuries Index
All approved modifications will be effective October 1, 2027

Revise Discharge ~~(accidental)~~ (undetermined)
Revise - firearm ~~(accidental)~~ (undetermined)Y24.9

TABLE of NEOPLASMS

All approved modifications will be effective October 1, 2027

Neoplasm	Malignant Primary	Malignant Secondary	Ca in situ	Benign	Uncertain Behavior	Unspecified Behavior
Neoplasm, neoplastic						
- abdomen, abdominal						
Revise - - wall -see also Neoplasm, abdomen, wall, skin		~~C79.2~~ C79.2				
bone						
Revise - patella	~~C40.2~~ C40.3-					
Revise patella	~~C40.20~~ C40.3-					
Revise pudenda, pudendum (~~femaie~~ female)						