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ICD-10 Coordination and Maintenance Committee Meeting
March 20, 2024
Diagnosis Agenda

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

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ICD-10 Coordination and Maintenance Committee Meeting
March 20, 2024

ICD-10 TIMELINE

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

March 19-20, 2024	The ICD-10 Coordination and Maintenance Committee Meeting.
March 2024	Recordings and slide presentations of the March 19-20, 2024 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/icd10cm_maintenance.htm **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
April 1, 2024	There will be no new ICD-10-CM codes implemented on April 1, 2024, but there will be addenda changes.
April 2024	Notice of Proposed Rulemaking to be published in the Federal Register as mandated by Public Law 99-509. This notice will include references to the FY 2025 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 17, 2024	**Deadline for receipt of public comments on proposed new diagnosis codes and revisions discussed at the March 19-20, 2024 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on October 1, 2025**.
May/June 2024	Final addendum posted on web pages as follows: **Diagnosis addendum -** https://www.cdc.gov/nchs/icd/Comprehensive-Listing-of-ICD-10-CM-Files.htm **Procedure addendum -** https://www.cms.gov/medicare/coding-billing/icd-10-codes
June 7, 2024	**Deadline for requestors: Those members of the public requesting that topics be discussed at the September 10-11, 2024 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 of new ICD-10-CM diagnosis codes should indicate if they are submitting their code request for consideration for an early April 1, 2025 implementation date or an October 1, 2025 implementation date.

The ICD-10 Coordination and Maintenance Committee will make efforts to accommodate requested early implementation dates if the following criteria are met: new diseases or disorders and / or public health importance or emergency.

July 2024	Federal Register notice for the September 10-11, 2024 ICD-10 Coordination and Maintenance Committee Meeting will be published. This will include the tentative agenda.
August 1, 2024	Hospital Inpatient Prospective Payment System final rule expected to be published in the Federal Register as mandated by Public Law 99-509. This rule will also include links to all the final codes to be implemented on October 1, 2024. This rule can be accessed at: https://www.cms.gov/medicare/payment/prospective-payment-systems/acute-inpatient-pps
August 2024	Tentative agenda for the Procedure portion of the September 10, 2024 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 11, 2024 ICD-10 Coordination and Maintenance Committee Meeting will be posted on the NCHS webpage at - https://www.cdc.gov/nchs/icd/icd10cm_maintenance.htm
September 10-11, 2024	The September 2024 ICD-10 Coordination and Maintenance Committee Meeting is anticipated to be fully virtual by Zoom and dial-in. Those who wish to attend must participate via Zoom Webinar or by dialing in.
September 2024	Recordings and slide presentations of the September 10-11, 2024 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/icd10cm_maintenance.htm
	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, 2024	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/Comprehensive-Listing-of-ICD-10-CM-Files.htm
	Procedure addendum – https://www.cms.gov/medicare/coding-billing/icd-10-codes
October 11, 2024	**Deadline for receipt of public comments on proposed new codes discussed at the September 10-11, 2024 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on April 1, 2025, if applicable.**
November 2024	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, 2025 will be posted on the following websites:
	https://www.cdc.gov/nchs/icd/Comprehensive-Listing-of-ICD-10-CM-Files.htm
	https://www.cms.gov/medicare/coding-billing/icd-10-codes/latest-news
November 15, 2024	**Deadline for receipt of public comments on proposed new codes and revisions discussed at the September 10-11, 2024 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on October 1, 2025.**
December 6, 2024	**Deadline for requestors: Those members of the public requesting that topics be discussed at the March 18-19, 2025 ICD-10 Coordination and Maintenance Committee Meeting must have their requests submitted to CMS for procedures and to NCHS for diagnoses by this date.**
	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 of new ICD-10-CM diagnosis codes should indicate if they are submitting their code request for consideration for an early October 1, 2025, or an early April 1, 2026, implementation date or an October 1, 2026 implementation date. The ICD-10 Coordination and Maintenance Committee will make efforts to accommodate requested early implementation dates if the following criteria are met; new diseases or disorders and / or public health importance or emergency.
January 2025	Federal Register notice for the March 18-19, 2025 ICD-10 Coordination and Maintenance Committee Meeting will be published. This will include the tentative agenda.
February 2025	Tentative agenda for the Procedure portion of the March 18, 2025 ICD-10 Coordination and Maintenance Committee Meeting posted on CMS webpage at: https://www.cms.gov/Medicare/Coding/ICD10/C-and-M-Meeting-Materials.html Tentative agenda for the Diagnosis portion of the March 19, 2025 ICD-10 Coordination and Maintenance Committee Meeting posted on NCHS homepage at: https://www.cdc.gov/nchs/icd/icd10cm_maintenance.htm
February 1, 2025	ICD-10 MS-DRG Grouper software and related materials posted on CMS webpage at: https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/AcuteInpatientPPS/MS-DRG-Classifications-and-Software
February 1, 2025	Any updates to the ICD-10-CM and ICD-10-PCS Coding Guidelines will be posted on the following websites: https://www.cdc.gov/nchs/icd/Comprehensive-Listing-of-ICD-10-CM-Files.htm https://www.cms.gov/Medicare/Coding/ICD10/
February 1, 2025	All ICD-10-CM and ICD-10-PCS code update files (includes April 1 update and full files from prior October 1) will be posted on the following websites: https://www.cdc.gov/nchs/icd/Comprehensive-Listing-of-ICD-10-CM-Files.htm https://www.cms.gov/Medicare/Coding/ICD10/
March 18-19, 2025	The ICD-10 Coordination and Maintenance Committee Meeting is anticipated to be fully virtual by Zoom and dial-in. Those who wish to attend must participate via Zoom Webinar or by dialing in.

March 2025	Recordings and slide presentations of the March 18-19, 2025 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/icd10cm_maintenance.htm **Procedure code portion of the recording and related materials–** https://www.cms.gov/Medicare/Coding/ICD10/C-and-M-Meeting-Materials.html
April 1, 2025	Any new or revised ICD-10 codes or addenda previously announced will be implemented on April 1, 2025.
April 18, 2025	**Deadline for receipt of public comments on proposed new codes and revisions discussed at the March 18-19, 2025 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on October 1, 2025.**
April 2025	Notice of Proposed Rulemaking to be published in the Federal Register as mandated by Public Law 99-509. This notice will include references to the FY 2026 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/medicare-fee-for-service-payment/acuteinpatientpps
May 16, 2025	**Deadline for receipt of public comments on proposed new codes and revisions discussed at the March 18-19, 2025 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on April 1, 2026, if applicable.** **Deadline for receipt of public comments on proposed new diagnosis codes and revisions discussed at the March 18-19, 2025 ICD-10 Coordination and Maintenance Committee Meeting being considered for implementation on October 1, 2026**.
May/June 2025	Final addendum posted on web pages as follows: **Diagnosis addendum -** https://www.cdc.gov/nchs/icd/Comprehensive-Listing-of-ICD-10-CM-Files.htm **Procedure addendum -** https://www.cms.gov/Medicare/Coding/ICD10/index.html

June 6, 2025	**Deadline for requestors: Those members of the public requesting that topics be discussed at the September 9-10, 2025 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 of new ICD-10-CM diagnosis codes should indicate if they are submitting their code request for consideration for an early April 1, 2026, implementation date or an October 1, 2026 implementation date. The ICD-10 Coordination and Maintenance Committee will make efforts to accommodate requested early implementation dates if the following criteria are met: new diseases or disorders and / or public health importance or emergency.
July 2025	Federal Register notice for the September 9-10, 2025 ICD-10 Coordination and Maintenance Committee Meeting will be published. This will include the tentative agenda.
August 1, 2025	Hospital Inpatient Prospective Payment System final rule expected to be published in the Federal Register as mandated by Public Law 99-509. This rule will also include links to all the final codes to be implemented on October 1, 2025. This rule can be accessed at: https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/AcuteInpatientPPS/index.html
August 2025	Tentative agenda for the Procedure portion of the September 9, 2025 ICD-10 Coordination and Maintenance Committee Meeting will be posted on the CMS webpage at – https://www.cms.gov/Medicare/Coding/ICD10/C-and-M-Meeting-Materials.html Tentative agenda for the Diagnosis portion of the September 10, 2025 ICD-10 Coordination and Maintenance Committee Meeting will be posted on the NCHS webpage at - https://www.cdc.gov/nchs/icd/icd10cm_maintenance.htm
September 9-10, 2025	The September 2025 ICD-10 Coordination and Maintenance Committee Meeting is anticipated to be fully virtual by Zoom and dial-in. Those who wish to attend must participate via Zoom Webinar or by dialing in.

September 2025	Recordings and slide presentations of the September 9-10, 2025 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/icd10cm_maintenance.htm **Procedure code portion of the recording and related materials–** https://www.cms.gov/Medicare/Coding/ICD10/C-and-M-Meeting-Materials.html
October 1, 2025	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/Comprehensive-Listing-of-ICD-10-CM-Files.htm **Procedure addendum –** https://www.cms.gov/Medicare/Coding/ICD10/

Contact Information

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

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ICD-10 Coordination and Maintenance Committee Meeting
March 20, 2024

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.

Abnormal rheumatoid factor and anti-citrullinated protein antibody without a diagnosis of rheumatoid arthritis

The proposal was previously presented at the September 2022 and September 2023 ICD10 Coordination and Maintenance committee meeting and is being represented with changes received from public comments.

Rheumatoid arthritis (RA) is a well-known autoimmune condition that is characterized by the presence of inflammatory arthritis (IA)[1]. Furthermore, in up to 80% of individuals with RA there are also abnormalities of circulating biomarkers including but not limited to the autoantibodies rheumatoid factor (RF) and anti-citrullinated proteins antibody (called 'ACPA') a subset of which that is commonly tested in clinical is anti-cyclic citrullinated protein (or peptide) antibodies (called 'anti-CCP')[2].

The current paradigm for the diagnosis and treatment of RA is for a clinician to identify joint findings that are determined to be IA, diagnose that as RA based on clinical, laboratory and radiographic features, and initiate treatment. Furthermore, this is the typical clinical situation when the existing ICD-10-CM codes for RA (e.g. M06/M05) are applied. Notably, RA may be formally classified according to established criteria[3, 4]; however, in clinical practice RA is a clinical diagnosis that may or may not meet these criteria.

However, it is now well-established that RA-related immunologic tests such as RF and ACPA/anti-CCP can be present in individuals in absence of and prior to the appearance of IA, and predictive of future onset of clinical RA[5]. Furthermore, individuals who have abnormal RA-related immunologic tests without IA are identified in growing numbers in clinical care. Notably, these individuals may have symptoms such as joint pain, stiffness or swelling, but no other objective evidence of IA[6-8]. There are current recommendations for medical follow-up and lifestyle changes (e.g., smoking cessation) that can be applied to these individuals. In addition, the predictive ability of RF and ACPA for future clinical RA has underpinned multiple clinical observational studies and prevention trials in RA[9-13]. In particular, two studies have recently demonstrated that abatacept significantly reduces rate of progression from an anti-CCP positive state to clinical RA[14, 15]. Based on this, there are efforts underway to obtain approval for pharmacologic therapy in this condition to prevent or delay the future onset of clinical RA.

There are ICD-10-CM codes that can be used to designate clinical RA. However, there is not currently a way in the existing ICD-10-CM system to have a single code to designate clearly in the main code title individuals who may have abnormal RA-related autoantibodies, but <u>not</u> have a diagnosis of clinical RA. As such, the introduction of a new code to accurately designate an

individual who has abnormal immunologic tests of RF and/or anti-CCP will facilitate clinical designation and care of these individuals, as well as facilitate clinical research.

This proposal was submitted by The University of Colorado, Division of Rheumatology and is supported by the American College of Rheumatology (ACR).

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TABULAR MODIFICATIONS

	M05	Rheumatoid arthritis with rheumatoid factor
New code	M05.A	Abnormal rheumatoid factor and anti-citrullinated peptide antibody without rheumatoid arthritis
Add		Abnormal anti-cyclic citrullinated peptide antibody and rheumatoid factor
Add		Abnormal CCP
Add		Excludes1: rheumatoid arthritis without rheumatoid factor (M06.0)

Adverse effect to Fluoroquinolones

Fluoroquinolones (FQs) are a family of broad spectrum antibacterial agents that have been used widely as therapy for respiratory and urinary tract infections, as well as for other bacterial diseases [1]. In the United States in 2022, there were 14.8 million total fluoroquinolone prescriptions dispensed and 44 prescriptions per 1,000 of the population [2].

While the therapeutic efficacy of fluoroquinolones is clearly recognized and they are valuable for severe life-threatening infections, it is evident that fluoroquinolones are accompanied by a variety of systemic side effects and adverse effects. As reported by the FDA: "disabling side effects involving tendons, muscles, joints, nerves and the central nervous system, including agitation tremor, hallucinations, psychosis and convulsions" [3]. "Other adverse effects include, peripheral neuropathy, photosensitivity, retinal detachment, QTc prolongation, cardiac arrhythmia, gastrointestinal effects, hyperglycemia, hypoglycemia, and more" [4]. These side effects can occur hours to weeks after exposure to fluoroquinolones and may potentially be permanent [3].

In 2015, the FDA reported statistics showing that the top 6 disabling antibiotics were all fluoroquinolones, ranging from 9.9% - 31.1% of the total disabled percentage [5]. In 2010, the National Institute of Health reported the following fluoroquinolone statistics: besides FQ-induced tendon injury (0.14–0.4%), the most common adverse effects associated with FQ use are gastrointestinal (1–7%), neurological (0.1–0.3%), cutaneous eruptions (0.5–2.5%), gait disturbances (<1%), elevation of serum transaminases (1.8–2.5%) [6]. Nausea, vomiting, diarrhea and taste disturbance have been reported to occur in up to 20% of patients [7]. Fluoroquinolones are associated with a two- to fourfold increased risk of acute tendinopathy and tendon rupture in all ages. The incidence of this adverse effect may be up to 2% in patients aged 65 years and above [4]. FQ treated patients followed from 1997 to 2012, experienced 37,338 (2.1%) tendon ruptures, 3,246 (0.2%) retinal detachments, and 18,391 (1.1%) aortic aneurysms [7]. Adverse events of this antibiotic class range from acute to chronic, as noted in numerous FDA Black Box Warnings [8,9,10], briefings [11], webinar [6], and in fluoroquinolone research studies.

The FDA notes that adverse fluoroquinolone effects are underreported [12] and the occurrence is high in absolute numbers because of the vast quantities of prescriptions. In 2019, Bayer's Medical Director stated that the total combined use of fluoroquinolones was 800 million worldwide, and that is just data from one manufacturer [13]; the percentage that experience adverse effects (approximately 1 percent) would equate to 8 million individuals.

Representatives of the Fluoroquinolone Toxicity Study have requested the following tabular modifications.

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TABULAR MODIFICATIONS

	T36	Poisoning by, adverse effect of and underdosing of systemic antibiotics
New subcategory	T36.A	Poisoning by, adverse effect of and underdosing of fluoroquinolone antibiotics
New sub-subcategory	T36.AX	Poisoning by, adverse effect of and underdosing of fluoroquinolone antibiotics
New code	T36.AX1	Poisoning by fluoroquinolone antibiotics, accidental (unintentional)
New code	T36.AX2	Poisoning by fluoroquinolone antibiotics, intentional self-harm
New code	T36.AX3	Poisoning by fluoroquinolone antibiotics, assault
New code	T36.AX4	Poisoning by fluoroquinolone antibiotics, undetermined
New code	T36.AX5	Adverse effect of fluoroquinolone antibiotics
New code	T36.AX6	Underdosing of fluoroquinolone antibiotics

APOL1-Mediated Kidney Disease

APOL1- Mediated Kidney Disease (AMKD) is a serious and progressive kidney condition associated with specific inheritable mutations of the APOL1 gene. AMKD clinically manifests as a spectrum of kidney pathologies and is associated with rapid progression to kidney failure. AMKD overwhelmingly affects people of Sub-Saharan African ancestry and is believed to be the most significant contributor to persistent health disparities in end-stage renal disease (ESRD) in the United States, which disproportionately affects (by roughly 3.5-fold) African Americans compared to European Americans.

New ICD-10-CM diagnosis codes ("N-codes") are being requested to specifically identify individuals with AMKD. In addition, status codes are being requested to track individuals with genetic risk-factor variants that have not yet manifested into clinically overt AMKD. These individuals are at much higher risk for severe and rapidly progressive kidney disease and should be both advised and monitored more closely.

AMKD is a kidney disorder associated with certain APOL1 genetic variants. The APOL1 gene is on chromosome 22 and encodes to the APOL 1 protein, a trypanolytic protein that plays a role in innate immunity against various *Trypanosoma* parasite species. The root cause of AMKD is two variants in the APOL1 gene (G1 and G2), which are predominantly found in people of Sub-Saharan African ancestry, including approximately 13% of African Americans (or about 5 million individuals). The G1 variant consists of "two amino acid substitutions (NM_003661.3 p.S342G and p.I384M) in near-perfect linkage, and the G2 [variant is] defined by a 2-aa deletion at the C terminus (NM_003661.3 p.delN388/Y389)." Two copies of either of these variants "cause a 7- to 10-fold increased risk of hypertension associated end-stage renal disease, a 10- to 17-fold increase in focal and segmental glomerulosclerosis, and a 29-fold increase in HIV nephropathy." APOL1 genetic variants have been associated with much of the excess risk of chronic kidney disease and ESRD, with some estimates showing that the lifetime risk of kidney disease in APOL1 dual-risk genetic variants individuals is at least 15% (versus 2% lifetime risk for European Americans).

Not all individuals with the G1/G2 variants develop AMKD. Rather, individuals with G1/G2 APOL1 risk variants develop AMKD when genetic factors interplay with epigenetic factors. Infectious or inflammatory epigenetic factors are known to be the "second hit" required to trigger AMKD in individuals with the two APOL1 risk variants.

AMKD is characterized by "kidney function decline, variable proteinuria levels, and hypertension." Precise expression of AMKD can have diverse clinical manifestations of different primarily kidney and kidney-associated pathologies. Generally, however, the presence of two APOL1 risk variants and chronic kidney disease is associated with rapid kidney disease progression to kidney failure relative to patients without two APOL1 risk variants, including higher rates of ESRD and greater progression of chronic kidney disease irrespective of diabetes status. For people with non-diabetic kidney disease and two APOL1 risk variants, dialysis is initiated on average 9-12 years earlier than people without the two risk variants.

There are limitations on the current data about the incidence, prevalence, and epidemiology of AMKD due to deficits in identification, which are compounded by the absence of a unique diagnosis code. Nonetheless, researchers have estimated that AMKD accounts for meaningful proportions of the clinical presentations of certain notable categories of kidney disease in people of Sub-Saharan African ancestry, including between 54% to 73% of presentation of focal segmental glomerulosclerosis (FSGS), 62% to 79% of HIV-associated neuropathy, 23% of hypertension-associated kidney disease, and 17% to 25% of lupus nephritis within this sub-population. A study also is underway to further estimate the prevalence of APOL1 genotypes and to identify individuals who may be eligible for clinical trials evaluating APOL1-targeted therapies. A rough estimate suggests that AMKD may affect as many as 800,000 people in the United States.

Current treatments for people with AMKD include sodium glucose transporter 2 inhibitor (SGLT2i) therapies, renin-angiotensin-aldosterone system inhibitor (RAASI) therapies and other antihypertensives, diuretics, immunosuppressants and other drugs such as mineralocorticoid receptor antagonists. These treatments are not unique to targeting AMKD but instead target the consequences of kidney disease and are used across a range of kidney diseases to delay progression to ESRD. At present, there is no Food and Drug Administration approved treatment specifically targeting the underlying cause of AMKD, although there are a number of potential treatments for AMKD in clinical trials now.

An ICD-10-CM code is of value to the continued development of epidemiological initiatives, ongoing research into treatments targeting AMKD and to improving clinical practice (including prognosis, informing kidney donation decisions, supporting family planning, improving understanding of treatment options and success, and supporting quality assurance efforts related to blood pressure control and kidney disease screening).

The Renal Physicians Association has submitted the proposal for consideration.

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TABULAR MODIFICATIONS

N07 Hereditary nephropathy, not elsewhere classified

New Code N07.B Hereditary nephropathy, not elsewhere classified with APOL1-mediated kidney disease [AMKD]

Add Note: Presence of two APOL1 alleles are genetically established.

Add AMKD with glomerulonephritis

Add AMKD with glomerulosclerosis

Z15 Genetic susceptibility to disease

New subcategory Z15.3 Genetic susceptibility to kidney disease

New code Z15.31 Genetic susceptibility to APOL1-mediated kidney disease

Add Genetic susceptibility to AMKD

Add Note: Presence of two APOL1 alleles are genetically established.

Add Use additional code to identify presence of:

hypertension (I10-I1A)

New code Z15.39 Other genetic susceptibility to kidney disease

Z84 Family history of other conditions

Z84.1 Family history of disorders of kidney and ureter
Conditions classifiable to N00-N29

New code Z84.11 Family history of APOL1-mediated kidney disease [AMKD]

New code Z84.12 Family history of disorders of kidney disease and ureter

New code Z84.19 Family history of other disorders of kidney and ureter

Baked Egg Tolerance in Egg Allergy

Egg allergy affects an estimated 0.5% to 2.5% of children younger than 5 years of age.[1] While 80% of children eventually outgrow egg allergy, and most were thought to develop tolerance by school age, studies indicate that some children are retaining egg allergy into adolescence.[2] It appears that the longer the egg allergy persists, the less likely tolerance will develop. Thus, it is important to understand individualized prognosis of egg allergy and develop clinical management that will improve the quality of life of egg-allergic children and, ideally, promote earlier tolerance development. This proposal was presented at the September 2023 C&M Meeting. In response to public comments, changes have been made and noted in **bold.**

It has become clear that different phenotypes of egg allergy exist, and these appear to be associated with different prognoses. There are egg-allergic patients who tolerate egg in baked products (baked egg-tolerant) but still react to stove-top cooked eggs (scrambled, fried, and boiled), and then there are patients who react to all forms of egg including well-baked products (baked egg-reactive). Baking modifies egg proteins and makes them less allergenic by destroying conformational epitopes and/or blocking epitopes through interactions with the food matrix (e.g., wheat flour).[3,4] This results in decreased IgE binding to egg proteins and increased tolerability. Clinical studies have indicated that a majority, 70-80%, of egg-allergic individuals can tolerate baked egg.[5,6]

Ovalbumin is the predominant protein in egg, making up 54% of egg white (EW), and is heat labile. Ovomucoid makes up only 11% of EW but is considered the more dominant allergen and is heat stable. In one study, higher specific IgE (sIgE) to ovomucoid was associated with reactivity to heated (well-cooked, but not baked) egg and 94% of subjects who reacted to heated egg subsequently tolerated ovomucoid-depleted heated egg.[7] Many studies have looked at using sIgE levels to total EW and components, such as ovomucoid, and/or skin prick testing to egg and components as a way to predict baked egg reactivity, however consistent cut-offs have not been found.[8,9] Additional studies are ongoing to find a biomarker for baked egg reactivity.[10]

In the meantime, baked egg tolerability is typically assessed clinically. Either the patient is already tolerating baked egg in their diet at the time of evaluation or an oral food challenge to a baked egg product (most often a muffin) is offered under supervision. Providers may use history, severity of past reactions, and testing as a guide for who to offer a baked egg challenge. Even if patients do not initially tolerate baked egg, tolerance may develop with time and regular reassessment is suggested.[11]

Egg-allergic children that tolerate baked egg appear to be more likely to outgrow their egg allergy.[12,13] Studies have shown that predominant IgE binding to ovomucoid, particularly sequential or linear epitopes, is associated with persistent egg allergy.[14,15] Those epitopes are thought to be less effected by heating or baking and matrix effects. It is possible that egg-allergic children without predominant ovomucoid IgE binding are more likely to tolerate baked egg and be naturally predisposed to outgrowing their allergy, representing a more transient egg allergy.

There is also evidence that regular ingestion of baked egg in the diet may help children outgrow their egg allergy.[11,13,16] In one study, subjects ingesting baked egg regularly were 14.6 times more likely

than subjects in the comparison group (P< .0001) to develop regular egg tolerance, and they developed tolerance earlier (median 50.0 vs 78.7 months; P< .0001). Baked egg ingestion was associated with immunologic changes, including decreasing skin prick testing to egg and egg-specific IgE levels, and increasing egg-specific IgG4 levels.[11] These immunologic changes parallel those seen in the natural resolution of egg allergy. The authors concluded that initiation of a baked egg diet accelerates the development of regular egg tolerance compared with strict avoidance. Therefore, differentiation of the different phenotypes of egg allergy can lead to different management, specifically earlier and sustained exposure to baked egg in tolerant patients as a form of possible treatment for egg allergy.

While tolerance to baked egg is associated with a better prognosis - i.e., more likely to outgrow their egg allergy, reactivity to baked egg is associated with poorer outcomes. Those that react to baked egg are less likely to outgrow their egg allergy and more likely to react to small amounts of egg. Thus, baked egg reactive patients may be considered for specific therapies due to this and a code to identify them would be beneficial.

Current ICD-10-CM codes include anaphylactic reaction due to eggs and a historical report of allergy to eggs (Z code). Neither specify tolerance of the baked form of egg, which a majority of egg-allergic patients tolerate, and which is associated with a better prognosis and increased likelihood of tolerance development. An additional ICD-10-CM code to signify baked egg tolerance would help to identify patients who may benefit from intervention and who are likely to outgrow their egg allergy, warranting close follow-up, repeat testing, and tolerance assessment.

This proposal is submitted jointly by physicians and coding professionals within the American Academy of Allery, Asthma & Immunology (AAAAI).

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TABULAR MODIFICATIONS

T78 Adverse effects, not elsewhere classified
Excludes2:complications of surgical and medical care NEC (T80-T88)

T78.0 Anaphylactic reaction due to food
Anaphylactic reaction due to adverse food reaction
Anaphylactic shock or reaction due to nonpoisonous foods
Anaphylactoid reaction due to food

T78.08 Anaphylactic reaction due to eggs

New code	**T78.080 Anaphylactic reaction due to egg with tolerance to baked egg**
Add	**Excludes1: Anaphylactic reaction due to egg with reactivity to baked egg (T78.081)**
New code	**T78.081 Anaphylactic reaction due to egg with reactivity to baked egg**
Add	**Excludes1: Anaphylactic reaction due to egg with tolerance to baked egg (T78.080)**
New code	**T78.089 Anaphylactic reaction due to eggs, unspecified**

T78.1 Other adverse food reactions, not elsewhere classified
Use additional code to identify the type of reaction, if applicable
Excludes1: anaphylactic reaction or shock due to adverse food reaction (T78.0-)
anaphylactic reaction due to food (T78.0-)
bacterial food borne intoxications (A05.-)

Excludes2: allergic and dietetic gastroenteritis and colitis (K52.29)
allergic rhinitis due to food (J30.5)
dermatitis due to food in contact with skin (L23.6, L24.6, L25.4)
dermatitis due to ingested food (L27.2)
food protein-induced enterocolitis syndrome (K52.21)
food protein-induced enteropathy (K52.22)

New subcategory **T78.12 Other adverse food reaction due to eggs**

New code **T78.120 Other adverse food reaction due to egg with tolerance to baked egg**
Add **Excludes1: Other adverse food reaction due to egg with reactivity to baked egg (T78.121)**

New code **T78.121 Other adverse food reaction due to egg with reactivity to baked egg**
Add **Excludes1: Other adverse food reaction due to egg with tolerance to baked egg (T78.120)**

New code T78.19 Other adverse food reactions, not elsewhere classified

Z91 Personal risk factors, not elsewhere classified
Z91.0 Allergy status, other than to drugs and biological substance
Z91.01 Food allergy status
Z91.012 Allergy to eggs

New code	**Z91.0120 Allergy to eggs, unspecified**
New code	**Z91.0121 Allergy to eggs with tolerance to baked egg**
Add	**Excludes1: Allergy to eggs with reactivity to baked egg (Z91.0122)**
New code	**Z91.0122 Allergy to eggs with reactivity to baked egg**
Add	**Excludes1: Allergy to egg with tolerance to baked egg (Z91.0121)**

Baked Milk Tolerance in Milk Allergy

Cow's milk (CM) allergy affects up to 2% of children younger than 5 years of age.[1] While 80% of children eventually outgrow milk allergy, and most were thought to develop tolerance by school age, studies indicate that some children are retaining milk allergy into adolescence.[2] It appears that the longer the milk allergy persists, the less likely tolerance will develop. Thus, it is important to understand individualized prognosis of milk allergy and develop clinical management that will improve the quality of life of milk-allergic children and, ideally, promote earlier tolerance development.

It has become clear that different phenotypes of milk allergy exist, and these appear to be associated with different prognoses. There are milk-allergic patients who tolerate milk in baked products (baked milk-tolerant) but still react to uncooked milk, and then there are patients who react to all forms of milk including well-baked products (baked milk-reactive). Baking modifies milk proteins and makes them less allergenic by destroying conformational epitopes and/or blocking epitopes through interactions with the food matrix (e.g., wheat flour).[3,4] This results in decreased IgE binding to milk proteins and increased tolerability. Clinical studies have indicated that a majority, 70-80%, of milk-allergic individuals can tolerate baked milk.[5,6]

The predominant protein in CM is casein, making up 80%. Casein is heat stable and is considered the more dominant allergen. Whey makes up 20% of CM protein and is heat labile. Studies have reported that higher specific IgE (sIgE) to casein is associated with reactivity to baked milk.[7,8] Many studies have looked at the using sIgE levels to total CM and components, such as casein, and/or skin prick testing to CM and components as a way to predict baked milk reactivity, however consistent cut-offs have not been found.[9]

In the meantime, baked milk tolerability is typically assessed clinically. Either the patient is already tolerating baked milk in their diet at the time of evaluation or an oral food challenge to a baked milk product (most often a muffin) is offered under supervision. Providers may use history, severity of past reactions, and testing as a guide for who to offer a baked milk challenge. Even if patients do not initially tolerate baked milk, tolerance may develop with time and regular reassessment is suggested.[10]

Milk-allergic children that tolerate baked milk appear to be more likely to outgrow their milk allergy.[10,11] Studies have shown that predominant IgE binding to casein, particularly sequential or linear epitopes, is associated with persistent milk allergy.[12,13] Those epitopes are thought to be less effected by heating or baking and matrix effects. It is possible that milk-allergic children without predominant casein IgE binding are more likely to tolerate baked milk and be naturally predisposed to outgrowing their allergy, representing a more transient milk allergy.

There is also evidence that regular ingestion of baked milk in the diet may help children outgrow their milk allergy. In one study, subjects ingesting baked milk regularly were 16 times more likely than subjects in the comparison group ($P < .0001$) to develop regular milk tolerance.[10] Baked milk ingestion was associated with immunologic changes, including decreasing skin prick testing to CM,

and increasing casein-specific IgG4 levels.[5,10] These immunologic changes parallel those seen in the natural resolution of milk allergy. The authors concluded that initiation of a baked milk diet accelerates the development of regular CM tolerance compared with strict avoidance. Therefore, differentiation of the different phenotypes of milk allergy can lead to different management, specifically earlier and sustained exposure to baked milk in tolerant patients as a form of possible treatment for milk allergy.

While tolerance to baked milk is associated with a better prognosis - i.e., more likely to outgrow their milk allergy, reactivity to baked milk is associated with poorer outcomes. Those that react to baked milk are less likely to outgrow their milk allergy and more likely to react to small amounts of milk. Thus, baked milk reactive patients may be considered for specific therapies due to this and a code to identify them would be beneficial.

Current ICD-10 codes include anaphylactic reaction due to milk and dairy products, and a historical report of allergy to milk products (Z code). Neither specify tolerance of the baked form of milk, which a majority of milk-allergic patients tolerate, and which is associated with a better prognosis and increased likelihood of tolerance development.

An additional ICD-10-CM code to signify baked milk tolerance would help to identify patients who may benefit from intervention and who are likely to outgrow their milk allergy, warranting close follow-up, repeat testing, and tolerance assessment.

This proposal is submitted jointly by physicians and coding professionals within the American Academy of Allergy, Asthma & Immunology (AAAAI).

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TABULAR MODIFICATIONS

T78 Adverse effects, not elsewhere classified
Excludes2:complications of surgical and medical care NEC (T80-T88)

T78.0 Anaphylactic reaction due to food

T78.07 Anaphylactic reaction due to milk and dairy products

New code **T78.070 Anaphylactic reaction due to milk and dairy products with tolerance to baked milk**
Add **Excludes1: Anaphylactic reaction due to milk and dairy products with reactivity to baked milk (T78.071)**

New code **T78.071 Anaphylactic reaction due to milk and dairy products with reactivity to baked milk**
Add **Excludes1: Anaphylactic reaction due to milk and dairy products with tolerance to baked milk (T78.070)**

New code **T78.079 Anaphylactic reaction due to milk and dairy products, unspecified**

T78.1 Other adverse food reactions, not elsewhere classified
Use additional code to identify the type of reaction, if applicable
Excludes1:anaphylactic reaction or shock due to adverse food reaction (T78.0-)
anaphylactic reaction due to food (T78.0-)
bacterial food borne intoxications (A05.-)

Excludes2:allergic and dietetic gastroenteritis and colitis (K52.29)
allergic rhinitis due to food (J30.5)
dermatitis due to food in contact with skin (L23.6, L24.6, L25.4)

dermatitis due to ingested food (L27.2)
food protein-induced enterocolitis syndrome (K52.21)
food protein-induced enteropathy (K52.22)

New subcategory **T78.11 Other adverse food reactions due to milk and dairy products**

New code **T78.110 Other adverse food reactions due to milk and dairy products with tolerance to baked milk**

Add **Excludes1: Other adverse food reaction due to milk and dairy products with reactivity to baked milk (T78.111)**

New code **T78.111 Other adverse food reaction due to milk and dairy products with reactivity to baked milk**

Add **Excludes1: Other adverse food reaction due to milk and dairy products with tolerance to baked milk (T78.110)**

New code T78.19 Other adverse food reactions to other food, not elsewhere classified

Z91 Personal risk factors, not elsewhere classified

Z91.0 Allergy status, other than to drugs and biological substance

Z91.01 Food allergy status

Z91.011 Allergy to milk products

New code **Z91.0110 Allergy to milk products, unspecified**

New code **Z91.0111 Allergy to milk products with tolerance to baked milk**

Add **Excludes1: Allergy to milk products with reactivity to baked milk (Z91.0112)**

New code	**Z91.0112**	**Allergy to milk products with reactivity to baked milk**
Add		**Excludes1: Allergy to milk products with tolerance to baked milk (Z91.0111)**

Cannabis Hyperemesis Syndrome

Cannabis Hyperemesis Syndrome (CHS), also called Cannabinoid Hyperemesis Syndrome, is an emerging syndrome among cannabis consumers characterized by symptoms of cyclical nausea, vomiting, and stomach pain and attributed to prolonged, frequent (i.e., weekly) cannabis use. Reports of suspected CHS have increased in recent years, correlating with widespread cannabis legalization in the United States and Canada.[1,2] Individuals experiencing CHS often seek medical care as symptoms can be severe and debilitating. Reported medical outcomes vary widely, from contributing to death[3] to full resolution of symptoms with cessation of cannabis use.[4]

Determining the prevalence of CHS in emergency department and hospitalization data is difficult as no single ICD-10-CM code describing this diagnosis exists. An ICD-10-CM code is critically needed to identify CHS cases and document trends over time.

CHS was first described in 2004 in a case series of 9 patients with cyclical vomiting from South Australia.[5] In all cases, chronic (in this case, daily and prolonged) cannabis use predated the onset of cyclical vomiting illness. Since then, additional case reports and case series have characterized the syndrome as including the following: a history of chronic cannabis use (long-term weekly, and usually daily, use) predating the onset of vomiting illness; hyperemesis following a cyclical pattern every few weeks or months, often for years, in combination with chronic cannabis use; resolution of cyclical vomiting following cessation of cannabis use; a return of hyperemesis with resumption of regular cannabis use; and severe abdominal pain that is often relieved by compulsive bathing (i.e., taking multiple hot showers or baths while experiencing illness).[6-14] Survey data suggest patients with CHS almost universally have cannabis use disorder.[15] Diagnosis of CHS is often made by exclusion.[4]

Apart from cannabis use disorder, CHS is the first clinical syndrome attributable to cannabis consumption, and therefore important to monitor with increasing legalization of cannabis in the United States. In 2021, 5.8% of adults aged 12 or older (16.3 million people) had a marijuana use disorder in the past year; prevalence is higher among those aged 18 to 25, at 14.4%.[16] Daily or near daily cannabis use has increased in recent years; among adults using cannabis during 2002-2017, daily/near daily use increased from 18.0% to 27.2%.[17] In addition, concentrations of tetrahydrocannabinol (THC), which is impairing or mind-altering, in cannabis products have increased over time.[18] Higher THC concentration (>10% THC) is associated with continued and more frequent use among adolescents and young adults.[19,20] While higher THC concentrations have not been associated with CHS directly, it is likely they lead to increased chronic use, thereby increasing risk of CHS.

CHS is likely underrecognized in both the adult and adolescent populations.[9] A retrospective chart review in a Canadian emergency department found that clinicians are either failing to question patients' cannabis use, or failing to document it, leading to underdiagnosis or misdiagnosis as cyclical vomiting syndrome (CVS), for which there is an

ICD-10-CM code.[21]

Lack of an ICD-10-CM code for CHS and associated awareness among providers may lead to unnecessary and costly investigation to identify a diagnosis, as well as potential increased risk of severe outcomes (e.g., seizures, kidney failure, shock) for patients if CHS is not accurately identified. The addition of an ICD-10-CM code for CHS would allow for improved identification of CHS in clinical settings, including increased understanding of its presenting features; interventions to prevent complications and effective treatment for CHS including increased availability of counseling on cannabis cessation for patients.[1,9] Importantly, an ICD- 10-CM code is also needed to develop a standardized surveillance definition for CHS. This would allow for a better understanding of the incidence, prevalence, and potential risk factors of CHS, as well as monitoring of trends and cross-jurisdictional comparisons of CHS as cannabis use patterns change.

This proposal, submitted by the Division of Overdose Prevention, National Center for Injury Prevention and Control in the Centers for Disease Control and Prevention and the Colorado Department of Public Health and Environment.

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TABULAR MODIFICATIONS

R11 Nausea and vomiting
Excludes1: cyclical vomiting associated with migraine (G43.A-)
excessive vomiting in pregnancy (O21.-)
hematemesis (K92.0)
neonatal hematemesis (P54.0)
newborn vomiting (P92.0-)
psychogenic vomiting (F50.89)
vomiting associated with bulimia nervosa (F50.2)
vomiting following gastrointestinal surgery (K91.0)

R11.0 Nausea

Nausea NOS
Nausea without vomiting

R11.1 Vomiting

New code R11.16 Cannabis hyperemesis syndrome
Add Cannabinoid hyperemesis syndrome

Add Code also: cannabis abuse (F12.1-)
cannabis dependence (F12.2-)
cannabis use, unspecified (F12.92-, F12.93, F12.95-, F12.98-, F12.99)

Add Code also manifestations

Coding of Firearm Injuries Default

The National Center for Health Statistics received a proposal to change the default in the Alphabetic Index for External Causes.

This is a representation of a proposal that was originally presented at the March 2023 and September 2023, ICD10 Coordination and Maintenance meetings. Two coding options are being presented for consideration.

Option 1: change the intent category to which firearm injuries would default to (assault) or another intent category (accidental, intentional self-harm, legal intervention, terrorism or undetermined) is indicated by the medical documentation.

Option 2: change the intent category to which firearm injuries would default to (undetermined) or another intent category (accidental, intentional self-harm, legal intervention, terrorism or assault) is indicated by the medical documentation.

Please note that coding generally requires direct documentation by the patient's provider. However, the ICD-10-CM Official Guidelines for Coding and Reporting will be updated to show as an exception: *firearm injuries by intent, such as assault (X93-X95), or undetermined intent (Y22-Y24)*, to 1.B.14, Documentation by Clinicians Other than the Patient's Provider.

Option #1 [Changing default to assault]

EXTERNAL CAUSE OF INJURY INDEX MODIFICATIONS

	Discharge (accidental)
Revise	- firearm ~~(accidental) W34.00~~ X95.9
Add	- - accidental W34.00
Revise	- - handgun (pistol) (revolver) ~~W32.0~~ X93
Add	- - - accidental W32.0
Revise	- - hunting rifle ~~W33.02~~ X94.1
Add	- - - accidental W33.02
Revise	- - larger ~~W33.00~~ X94.9
Add	- - - accidental W33.00
Revise	- - - specified NEC ~~W33.09~~ X94.8
Add	- - - - accidental W33.09
Revise	- - machine gun ~~W33.03~~ X94.2
Add	- - - accidental W33.03
Revise	- - shotgun ~~W33.01~~ X94.0
Add	- - - accidental W33.01

Revise **Gunshot wound** (see also Discharge, firearm, by type) ~~W34.00~~ X95.9

Option #2 [Changing default to undetermined, shown below].

EXTERNAL CAUSE OF INJURY INDEX MODIFICATIONS

Discharge (accidental)

Revise - firearm ~~(accidental) W34.00~~ Y24.9
Add - - accidental W34.00
Revise - - handgun (pistol) (revolver) ~~W32.0~~ Y22
Add - - - accidental W32.0
Revise - - hunting rifle ~~W33.02~~ Y23.1
Add - - - accidental W33.02
Revise - - larger ~~W33.00~~ Y23.9
Add - - - accidental W33.00
Revise - - - specified NEC ~~W33.09~~ Y23.8
Add - - - - accidental W33.09
Revise - - machine gun ~~W33.03~~ Y23.3
Add - - - accidental W33.03
Revise - - shotgun ~~W33.01~~ Y23.0
Add - - - accidental W33.01
Revise **Gunshot wound** (see also Discharge, firearm, by type) ~~W34.00~~ Y24.9

Demodex blepharitis

Blepharitis is the inflammation of the eyelids causing irritation and redness. It has classically been categorized as anterior blepharitis or posterior blepharitis with the cilium or eyelashes as the landmark. Anterior blepharitis has been further subcategorized by presumed causation into staphylococcal blepharitis, seborrheic blepharitis, and acne rosacea-associated blepharitis. Additionally, nearly 70% of all cases are due to demodex infestation, which leads to demodex blepharitis by acting as a vector for harmful bacteria [3,4,5]. Demodex blepharitis is common among all people of all ages, races, ethnicities, and genders.

Demodex, a genus of tiny parasitic mites that live in or near hair follicles of mammals, are among the smallest of arthropods with two species *Demodex folliculorum* and *Demodex brevis* typically found on humans. Symptoms of demodex blepharitis include itching and redness, ocular pain and burning, foreign body sensation, dryness, lacrimation, purulence, irritation, loss of lashes, matted or crusty lashes, and blurry vision. Research has shown that the overwhelming majority of demodex blepharitis patients have difficulty negotiating daily activities. However, it is underdiagnosed, undertreated, and often misdiagnosed despite chronically persisting signs and symptoms that often require multiple visits to an eye care practitioner.

Diagnosis is complex because demodex mites reside on the eyelids of both healthy patients and those experiencing symptoms of blepharitis. No clinical standard currently exists that determines what level of demodex infestation causes blepharitis. Additionally, the symptoms of demodex blepharitis are in alignment with those of other ocular diseases, thus making it difficult for eye care practitioners to pinpoint [6]. Current management options include lid scrubs, tea tree oil, warm compresses, antibiotic/steroid combinations and microblepharoexfoliation. However, these treatments have low efficacy and eradication rates are low. Recently, the first and only FDA-approved treatment has been developed for the treatment of demodex blepharitis [7].

The American Optometric Association is requesting new codes to capture this significant condition and to facilitate research.

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ICD-10 Coordination and Maintenance Committee Meeting
March 20, 2024

TABULAR MODIFICATIONS

B88 Other infestations

B88.8 Other specified infestation

Delete ~~Ichthyoparasitism due to Vandellia cirrhosa~~

Delete ~~Linguatulosis~~

Delete ~~Porocephaliasis~~

New code B88.81 Infestation by Demodex mites

Add Demodex follculorum infestation

Add Code also, if applicable, eyelid inflammation (H01.8-)

New code B88.89 Other specified infestation

Add Ichthyoparasitism due to Vandellia cirrhosa

Add Linguatulosis

Add Porocephaliasis

H01 Other inflammation of eyelid

H01.8 Other specified inflammation of eyelid

Add Code also, if applicable, infestation by Demodex mites (B88.81)

New code H01.81 Other specified inflammation of right upper eyelid

New code H01.82 Other specified inflammation of right lower eyelid

New code H01.83 Other specified inflammation of right eye, unspecified eyelid

New code H01.84 Other specified inflammation of left upper eyelid

New code H01.85 Other specified inflammation of left lower eyelid

New code H01.86 Other specified inflammation of left eye, unspecified eyelid

New code	H01.89	Other specified inflammation of unspecified eye, unspecified eyelid
New code	H01.8A	Other specified inflammation of right eye, upper and lower eyelids
New code	H01.8B	Other specified inflammation of left eye, upper and lower eyelids

Digital Literacy

The Gravity Project (GP) requests new codes for Digital Literacy and Housing Instability. Over the last decades growing literature has clarified and further identified the social determinants of heath and the impact on healthcare. This has sparked initiation and dissemination of national recommendations and projects. Advances have been made to collectively gain insight into social risks and social interventions; yet the terminology used to represent these concepts lags behind.

Digital literacy is widely recognized as a critical skill in an increasingly digital age. Addressing digital literacy specifically was born out of Gravity Project's previous work on health literacy given the critical role of digital skills in accessing health appointments and health information.

Furthermore, the literature shows a clear association between digital literacy and outcomes in social attainment in the sphere of education, employment, and connection. The submission for digital literacy mirrors a previous request for health literacy.

In addition, inclusion terms and instructional notes are being requested for further specificity and clarification as it relates to the codes pertaining to housing instability with a new code for "Other specified housing instability".

TABULAR MODICATIONS

Z55 Problems related to education and literacy

New code	Z55.7 Problems related to digital literacy
Add	Difficulty demonstrating basic use of digital device
Add	Difficulty demonstrating internet use skills
Add	Difficulty demonstrating safe internet use
Add	Difficulty demonstrating use of patient portal

Z59 Problems related to housing and economic circumstances

Z59.8 Other problems related to housing and economic circumstances

Z59.81 Housing instability, housed

Add Burdensome rent or mortgage payments

Foreclosure on home loan

Past due on rent or mortgage

Unwanted multiple moves in the last 12 months

Add Excludes2: extreme poverty (Z59.5)

Add financial insecurity (Z59.86)

Add low income (Z59.6)

Add material hardship due to limited financial resources, not elsewhere classified (Z59.87)

New code Z59.818 Other specified housing instability, housed

Z59.819 Housing instability, housed unspecified

Delete ~~Excludes2: extreme poverty (Z59.5)~~

Delete ~~financial insecurity (Z59.86~~

Delete ~~low income (Z59.6)~~

Delete ~~material hardship due to limited financial resources, not elsewhere classified (Z59.87)~~

Do Not Resuscitate

A Do Not Resuscitate (DNR) order consists of a statement by a physician in the medical record informing the medical staff should cardiac or respiratory arrest occur, cardiopulmonary resuscitation (CPR) should not be performed, preventing the unnecessary or unwanted invasive treatment to prolong life.

A Full Code status indicates that in the event of cardiac or respiratory arrest, all life-sustaining measures should be attempted to resuscitate the patient such as chest compressions, intubation, defibrillation, and resuscitative medications.

While there are standard definitions of a Full Code status and a Do Not Resuscitate (DNR) status, there is limited literature available regarding Limited or Partial do-not-resuscitate code statuses. A Do Not Intubate (DNI) status indicates a patient does not want to receive endotracheal intubation in the event of a medical emergency, such as cardiac or respiratory arrest. A Limited Code or Partial Code status is based on the patient's anticipated need for resuscitation and interventions can be individually declined by patients and families. It can imply only certain procedures will be done, such as allowing for chest compressions and antiarrhythmic drugs (to prevent or relieve cardiac arrhythmia) but no endotracheal intubation or mechanical ventilation. Further, it can mean the DNR is limited NOT to take one or more of the following steps/actions as requested by the patient: withhold antiarrhythmics, withhold IV vasoactive drugs, withhold defibrillation/cardioversion, withhold chest compression, withhold ventilation by mask, withhold endotracheal intubation, withhold mechanical ventilation, withhold other CPR measures.

Code status and details of CPR and chest compressions can be severely underutilized inpatient populations, and based on demographics, a not insignificant portion of patients may elect to be a limited code or partial code. The hurdle with limited codes or partial codes is when the provider is not performing compressions and providing circulation. From a clinical perspective, the patient might as well be a DNR because the provider cannot properly follow Advanced Cardiovascular Life Support (ACLS) guidelines when a limited code or partial code is in place.
Example: A patient has a code status of a Limited Code and they agree with intubation, defibrillation, and resuscitative medications, but decline chest compressions. The patient goes into ventricular fibrillation, which is a direct indication to defibrillate the patient (because it's a shockable rhythm); however, ACLS guidelines state that chest compressions should be done throughout the code in between shocks. Another factor to consider is if antiarrhythmic drugs are injected but no chest compressions are allowed per the patient's code status, the drugs are not circulated throughout the body and do not give any benefit to the patient. There is a lower likelihood of survival if the healthcare professional deviates from the ACLS guidelines.

Bon Secours Mercy Health is requesting unique ICD-10-CM codes to differentiate when a patient is a DNR versus Do Not Intubate versus partial code / limited code. These unique codes will improve data analysis, allow providers to track different code statuses, and ultimately empower patients or surrogates to give informed consent with a full understanding of code statuses.

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TABULAR MODIFICATIONS

Revise	Z66	Do not resuscitate <u>and code status</u>
Delete		~~DNR status~~
New code	Z66.1	Do not resuscitate
Add		DNR status
New sub-subcategory	Z66.2	Limited (partial) code status
Add		Excludes1: Do not resuscitate (Z66.1)

New code	Z66.20	Limited (partial) code status, unspecified
Add		Limited (partial) code status, NOS
New code	Z66.21	Do not intubate
Add		DNI status
New code	Z66.29	Other limited (partial) code status

Encounter for prophylactic removal of fallopian tube(s) for persons with no known genetic/familial risk factors

This proposal was originally presented at the September 2023 ICD-10 Coordination and Maintenance meeting, changes were made, and additional codes are being requested per public comments from the meeting. The changes are in **bold**.

Ovarian cancer (OC) is among the top 5 deadliest cancers in women. The American Cancer Society estimates that in 2023 about 19,710 new cases of ovarian cancer will be diagnosed; the vast majority of cases (70%) will have high grade serous histology.[1] This amounts to one woman diagnosed with high grade serous cancer every 40 minutes in the US. Despite the name "ovarian cancer," accumulating epidemiological, clinical, pathological, and molecular data over the past 20 years indicate that high grade serous carcinoma primarily originates from microscopic precancers in the fimbriated ends of fallopian tubes, rather than from the ovary itself.[2,3] Given the seemingly insurmountable obstacles to effectively screening for and treating the disease, the medical community and the patients are increasingly interested in the option of ovarian cancer prevention through fallopian tube removal (bilateral salpingectomy).

For the past decade, gynecologic surgeons have used the term *opportunistic salpingectomy* to describe the recommended practice of discussing salpingectomy for the primary prevention of ovarian cancer with post-reproductive women planning to undergo pelvic surgery for another indication (eg, hysterectomy) or as an alternative to tubal ligation for surgical sterilization.[4] While 20% of high grade serous cancer is attributable to genetic risk factors, and genetically high risk women are still advised to have both fallopian tubes and ovaries removed to reduce OC risk upon completion of child-bearing, opportunistic salpingectomy is designed to prevent the 80% of high grade serous cancer that affects women with no known risk factors. There is a lack of clear understanding of predisposing factors in this vast majority of cases diagnosed in women who are seemingly average risk for the disease.[5,6]

What is known is that bilateral salpingectomy substantially decreases ovarian cancer risk. Data from nested case-control and population-based retrospective cohort studies indicate that bilateral salpingectomy reduces the risk of ovarian cancer by at least 65 percent.[7,8] In 2022, Canadian researchers published the first prospective evidence that opportunistic salpingectomy may substantially decrease the incidence of high-grade serous carcinoma in the general population. At the time of 9 years follow-up, no high-grade serous carcinoma was observed among the 25,889 women who underwent opportunistic salpingectomy during hysterectomy or in lieu of tubal ligation for surgical sterilization. This is significantly less than the expected rate as well as the rate seen in the 32,080 women who did not undergo bilateral salpingectomy.[9] Studies that have compared the addition of opportunistic salpingectomy to a gynecological or pelvic procedure without

salpingectomy have not found significant differences in ovarian endocrine function, surgical complications, operative time, or length of stay.[10,11] In the US, over a million women undergo hysterectomy or surgical sterilization annually.[12,13] By current projections, universal uptake of salpingectomy during hysterectomy and in lieu of tubal ligation could prevent nearly 2000 deaths from ovarian cancer per year.[14] Expanding opportunistic salpingectomy to post-reproductive women undergoing non-gynecologic elective abdominal surgeries such as cholecystectomy, hernia repair, appendectomy, and gastrointestinal and urologic operations would at least double the impact of opportunistic salpingectomy on decreasing ovarian cancer incidence and mortality.

A 2020 study demonstrated feasibility of opportunistic salpingectomy at the time of elective laparoscopic cholecystectomy, with 60% of counseled patients accepting salpingectomy, a surgical success rate of 95 out of 105 (93.3%) of enrolled patients, and no attributable surgical complications. Mean additional operating time was 13 minutes.[15] Given that the morbidity of the procedure is low, it can be performed using all available approaches (open, laparoscopic, robotic, vaginal), there is no impact on ovarian function and the acceptance rate is high. Salpingectomy as a practical, population-level approach to ovarian cancer prevention.[4,16,17] Fallopian tube removal for ovarian cancer prevention was publicized in recent media coverage by the New York Times, Washington Post and Scientific American Magazine headlining the importance of empowering people to consider and choose opportunistic salpingectomy, especially when it comes to preventing a cancer for which there is neither adequate screening nor a dependable cure.[18,19,20] It is equally important that providers have the tools to offer it as a standard of care. Updating medical coding to the current standard of care is an immediate action item. Medical coding deficiencies for cancer-preventive surgeries like opportunistic salpingectomy need to be rectified because they endanger patient access and health care clinician engagement.[21] One of the most obvious coding deficiencies is that there is no ICD-10-CM code for an encounter for the purpose of opportunistic salpingectomy. The only code available is Z40.03, which requires risk factors such as an inherited genetic mutation.

Because 80% of ovarian cancers affect women with no known genetic or familial risk, the intervention should be available to all women following the completion of childbearing; it should not be restricted to those with genetic susceptibility for and/or a family history of ovarian cancer. At present, Z40.03 is the only ICD-10-CM code for prophylactic salpingectomy. Use of this singular ICD-10-CM code has been and is restricted to high risk people - people with genetic susceptibility or family history of ovarian cancer. There is no ICD-10-CM code for the risk factor of simply being a human with fallopian tubes. This is a major deficiency because having fallopian tubes is the only identifiable risk for ovarian cancer for most women. Establishing the epidemiological basis for prophylactic salpingectomy as a primary prevention strategy for ovarian cancer, both as a population-wide and as a targeted high-risk strategy, will only be feasible through the generation of separate ICD-10-CM diagnosis codes for prophylactic salpingectomy that differentiate people with no known genetic/familial risk of ovarian cancer from those with known genetic/familial risk.

The Gynecologic Oncology Division of the Department of Gynecology and Obstetrics at Johns Hopkins University requested the following tabular modifications. This proposal was reviewed and is supported by the American College of Obstetricians and Gynecologists.

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TABULAR MODIFICATIONS

Z15 Genetic susceptibility to disease
Z15.0 Genetic susceptibility to malignant neoplasm
Code first, if applicable, any current malignant neoplasm (C00-C75, C81- C96)
Use additional code, if applicable, for any personal history of malignant neoplasm (Z85.-)

New code **Z15.05 Genetic susceptibility to malignant neoplasm of fallopian tube(s)**

Z40 Encounter for prophylactic surgery
Excludes1: organ donations (Z52.-)
therapeutic organ removal-code to condition

Z40.0 Encounter for prophylactic surgery for risk factors related to malignant neoplasms
Admission for prophylactic organ removal
Use additional code to identify risk factor

Z40.03 Encounter for prophylactic removal of fallopian tube(s)

New code **Z40.031 Encounter for prophylactic removal of fallopian tube(s) for persons with known genetic/familial risk factors**
Add **Encounter for salpingectomy for persons with known genetic/familial risk factors**

New code **Z40.032 Encounter for prophylactic removal of fallopian tube(s) for persons with no known genetic/familial risk factors**
Add **Encounter for prophylactic salpingectomy tube(s) for persons with no known genetic/familial risk factors**
Add **Opportunistic salpingectomy**

New code **Z40.039 Encounter for prophylactic removal of fallopian tube(s), unspecified**

Z80 Family history of primary malignant neoplasm
Z80.4 Family history of malignant neoplasm of genital organs
Conditions classifiable to C51-C63
New code Z80.44 Family history of malignant neoplasm of fallopian tube(s)

Z85 Personal history of malignant neoplasm
Z85.4 Personal history of malignant neoplasm of genital organs
Conditions classifiable to C51-C63
New code Z85.4A Personal history of malignant neoplasm of fallopian tube(s)

Z86 Personal history of certain other diseases
Z86.0 Personal history of in-situ and benign neoplasms and neoplasms of uncertain behavior
Z86.00 Personal history of in-situ neoplasm
Conditions classifiable to D00-D09
New code Z86.00A Personal history of in-situ neoplasm of the fallopian tube(s)

Encounter for weaning from ventilator

The National Center for Health Statistics (NCHS) received a proposal to create a new code for ventilator status and weaning. Those patients who have life-threatening conditions that require constant monitoring and comprehensive care will usually be admitted to a hospital critical care or intensive care unit (ICU).

With Intensive Care Unit (ICU) utilization on the rise, it is vitally important that physicians adhere to the established guidelines when reporting the clinical acuity or severity of illness and the intensity of critical care services rendered to their patients.

Critical care is defined as the direct delivery by a physician(s) medical care for a critically ill or critically injured patient. A critical illness or injury acutely impairs one or more vital organ systems such that there is a high probability of imminent or life-threatening deterioration in the patient's condition. It is necessary document a complete clinical picture for the patient and cognitive work effort by the physician to support and to communicate the medical necessity for this high-level of hospital service.

When a critically ill patient is admitted to an intensive care unit (ICU), it is important that the documentation clearly depicts the clinical acuity of the patient for which the physician is caring. When a patient is intubated and put on mechanical ventilation, they are at risk for ventilator associated complications; therefore, the patient will be evaluated and monitored as well as receiving ventilator assistance and planning for trials of weaning (Nair & Niederman, 2017).

The term "weaning" is used to describe the gradual process of decreasing ventilator support. It is estimated that 40% of the duration of mechanical ventilation is dedicated to the process of weaning. Spontaneous breathing trial (SBT) assesses the patient's ability to breathe while receiving minimal or no ventilator support. The collective task force in 2001 stated that the process of SBT and weaning should start by assessing whether the underlying cause of respiratory failure has been resolved or not. Weaning predictors are parameters that are intended to help clinicians predict whether weaning attempts will be successful or not.

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ICD-10 Coordination and Maintenance Committee Meeting
March 20, 2024

TABULAR MODIFICATIONS

Z99 Dependence on enabling machines and devices, not elsewhere classified

Z99.1 Dependence on respirator

Dependence on ventilator

Z99.11 Dependence on respirator [ventilator] status

Add Ventilator status

New code Z99.13 Encounter for weaning from respirator [ventilator]

External Causes: Fishing hook and wood splitting

NCHS has received a request to add specific codes for external cause fishing hook and activity code for wood splitting. These activities were requested due to frequency in specific geographical regions.

The following tabular modifications are being requested.

TABULAR MODIFICATIONS

W45 Foreign body or object entering through skin
Includes: foreign body or object embedded in skin
nail embedded in skin
Excludes2:contact with hand tools (nonpowered) (powered) (W27-W29)
contact with other sharp object(s) (W26.-)
contact with sharp glass (W25.-)
struck by objects (W20-W22)

The appropriate 7th character is to be added to each code from category W45
A initial encounter
D subsequent encounter
S sequela

	W45.0 Nail entering through skin
New code	W45.1 Fishing hook entering through skin
	W45.8 Other foreign body or object entering through skin Splinter in skin NOS

	Y93 Activity codes
New sub category	Y93.L Other outdoor activity
New code	Y93.L1 Activity, splitting wood
New code	Y93.L9 Activity, other outdoor activity

Flank Anatomical Specificity

The “flank” (also known as “latus” or “lumbar region”) of the thorax is a unique area of the body that lies between on the lateral aspect of the thorax between the rib cage and the iliac bone of the hip (below the rib cage and above the ilium). [Alberts, D; et al. (2012). Dorland's illustrated medical dictionary (32nd ed.). Philadelphia, PA: Saunders/Elsevier. p. 714]. Simply is it “the fleshy part of the side between the ribs and the hip” [https://www.merriam-webster.com/dictionary/flank].

This proposal was presented at the March 2021, September 2022, March 2023 and September 2023 Coordination and Maintenance meeting. In response to public comments, a revised proposal is being submitted for reconsideration. Changes are noted in **bold.**

There are times when a patient will seek medical care because of “flank pain” as opposed to abdominal or back pain. Pathology specific to flank pain can include kidney stones, pyelonephritis, gall bladder or liver disease, or muscle spasm to name a few. In addition, injuries to this area can lead to different muscle or intra-abdominal pathology.

The specific anatomical locale helps determine the clinician’s evaluation process as well as resource utilization. The division of the frontal and lateral aspects of the abdomen allows for greater specificity in evaluating the patient. Currently, ICD-10-CM directs the term “flank” to the abdomen.

The American College of Emergency Physicians (ACEP) requests specific codes be added to the ICD-10-CM code set to better capture this specific anatomic region. This proposal is supported by the American Academy of Pediatrics.

TABULAR MODIFICATIONS

L02 Cutaneous abscess, furuncle and carbuncle

L02.2 Cutaneous abscess, furuncle and carbuncle of trunk
- Excludes1: non-newborn omphalitis (L08.82)
  omphalitis of newborn (P38.-)
- Excludes2: abscess of breast (N61.1)
  abscess of buttocks (L02.3)
  abscess of female external genital organs (N76.4)
  abscess of male external genital organs (N48.2, N49.-)
  abscess of hip (L02.4)

L02.21 Cutaneous abscess of trunk

Revise — L02.212 Cutaneous abscess of back [any part, except buttock and flank]

New code — L02.217 Cutaneous abscess of flank

L02.22 Furuncle of trunk
Boil of trunk
Folliculitis of trunk

Revised — L02.222 Furuncle of back [any part, except buttock and flank]

New code — L02.227 Furuncle of flank

L02.23 Carbuncle of trunk

Revise — L02.232 Carbuncle of back [any part, except buttock and flank]

New code — L02.237 Carbuncle of flank

L03 Cellulitis and acute lymphangitis

L03.3 Cellulitis and acute lymphangitis of trunk

L03.31 Cellulitis of trunk

New code — L03.31A Cellulitis of flank

L03.32 Acute lymphangitis of trunk

New code — L03.32A Acute lymphangitis of flank

R10 Abdominal and pelvic pain
Excludes1: renal colic (N23)

Add — Excludes2: costovertebral (angle) tenderness (R39.85)
dorsalgia (M54.-)

Add — flatulence and related conditions (R14.-)

R10.1 Pain localized to upper abdomen

Add — Excludes2: pain localized to flank (R10.A-)

Add — pelvic and perineal pain (R10.2-)

	R10.2	Pelvic and perineal pain
Add		Excludes2: pain localized to other parts of lower abdomen (R10.3-)
Add		pain localized to upper abdomen (R10.1-)
New code		R10.20 Pelvic and perineal pain unspecified side
New code		R10.21 Pelvic and perineal pain right side
New code		R10.22 Pelvic and perineal pain left side
New code		R10.23 Pelvic and perineal pain bilateral
New code		R10.24 Suprapubic pain
	R10.3	Pain localized to other parts of lower abdomen
Add		Excludes2: pain localized to flank R10.A-
Add		pelvic and perineal pain (R10.2-)
New subcategory	R10.A	Pain localized to flank
Add		Lateral abdomen pain
Add		Lateral flank pain
Add		Latus region pain
Add		Excludes2: pain localized to other parts of lower abdomen (R10.3-)
Add		pain localized to upper abdomen (R10.1-)
New code		R10.A0 Flank pain, unspecified side
New code		R10.A1 Flank pain, right side
New code		R10.A2 Flank pain, left side
New code		R10.A3 Flank pain, bilateral
	R10.8	Other abdominal pain
		R10.81 Abdominal tenderness
		Abdominal tenderness NOS
Add		Excludes2: pain localized to other parts of lower abdomen (R10.3-)
Add		pain localized to upper abdomen (R10.1-)
		R10.82 Rebound abdominal tenderness
Add		Excludes2: pain localized to other parts of lower abdomen (R10.3-)

Add pain localized to upper abdomen (R10.1-)
New subcategory R10.8A Flank tenderness
New code Rl0.8A1 Right flank tenderness
New code Rl0.8A2 Left flank tenderness
New code R10.8A3 Suprapubic tenderness
New code R10.8A9 Flank tenderness, unspecified
Add Flank tenderness NOS

New code R10.85 Abdominal pain of multiple sites
Add Excludes1: abdominal rigidity NOS (Rl9.3)
Add generalized abdominal pain associated with acute abdomen (R10.0)
Add generalized abdominal pain NOS (Rl0.84)
Add localized abdominal pain (Rl0.l-Rl0.4-)

R39.8 Other symptoms and signs involving the genitourinary system
New sub-category R39.85 Costovertebral (angle) tenderness Add
Excludes2: abdominal and pelvic pain (Rl0.-)
New code R39.851 Costovertebral (angle) tenderness, right side
New code R39.852 Costovertebral (angle) tenderness, left side
New code R39.853 Costovertebral (angle) tenderness, bilateral
New code R39.859 Costovertebral (angle) tenderness, unspecified side

S30 Superficial injury of abdomen, lower back, pelvis and external genitals
S30.1 Contusion of abdominal wall and flank
Delete ~~Contusion of flank~~
Delete ~~Contusion of groin~~
New code **S30.11 Contusion of abdominal wall**
New code **S30.12 Contusion of groin**
New code **S30.13 Contusion of flank (latus) region**

S30.8 Other superficial injuries of abdomen, lower back, pelvis, and external genitals
S30.81 Abrasion of abdomen, lower back, pelvis, and external genitals
New code S30.81A Abrasion of flank

S30.82 Blister (nonthermal) of abdomen, lower back, pelvis, and external genitals

New code S30.82A Blister (nonthermal) of flank

S30.84 External constriction of abdomen, lower back, pelvis and external genitals

New code S30.84A External constriction of flank

S30.85 Superficial foreign body of abdomen, lower back, pelvis, and external genitals

New code S30.85A Superficial foreign body of flank

S30.86 Insect bite (nonvenomous) of abdomen, lower back, pelvis, and external genitals

New code S30.86A Insect bite (nonvenomous) of flank

S30.87 Other superficial bite of abdomen, lower back, pelvis, and external genitals

New code S30.87A Other superficial bite of flank

S30.9 Unspecified superficial injury of abdomen, lower back, pelvis, and external genitals

New code S30.9A Unspecified superficial injury of flank

S31 Open wound of abdomen, lower back, pelvis and external genitals

S31.1 Open wound of abdominal wall without penetration into peritoneal cavity

S31.10 Unspecified open wound of abdominal wall without penetration into peritoneal cavity

New code S31.106 Unspecified open wound of abdominal wall, right flank without penetration into peritoneal cavity

New code S31.107 Unspecified open wound of abdominal wall, left flank without penetration into peritoneal cavity

New code	S31.10A Unspecified open wound of abdominal wall, unspecified flank without penetration into peritoneal cavity
Add	Open wound of abdominal wall of flank NOS without penetration into peritoneal cavity
	S31.11 Laceration without foreign body of abdominal wall without penetration into peritoneal cavity
New code	S31.116 Laceration without foreign body of abdominal wall, right flank without penetration into peritoneal cavity
New code	S31.117 Laceration without foreign body of abdominal wall, left flank without penetration into peritoneal cavity
New code	S31.11A Laceration without foreign body of abdominal wall, unspecified flank without penetration into peritoneal cavity
Add	Laceration without foreign body of flank NOS without penetration into peritoneal cavity
	S31.12 Laceration with foreign body of abdominal wall without penetration into peritoneal cavity
New code	S31.126 Laceration with foreign body of abdominal wall, right flank without penetration into peritoneal cavity
New code	S31.127 Laceration with foreign body of abdominal wall, left flank without penetration into peritoneal cavity

New code S31.12A Laceration with foreign body of abdominal wall unspecified flank without penetration into peritoneal cavity

Add Laceration with foreign body of abdominal wall of flank NOS without penetration into peritoneal cavity

S31.13 Puncture wound of abdominal wall without foreign body without penetration into peritoneal cavity

New code S31.136 Puncture wound of abdominal wall without foreign body, right flank without penetration into peritoneal cavity

New code S31.137 Puncture wound of abdominal wall without foreign body, left flank without penetration into peritoneal cavity

New code S31.13A Puncture wound of abdominal wall without foreign body, unspecified flank without penetration into peritoneal cavity

Add Puncture wound of abdominal wall of flank NOS without foreign body

S31.14 Puncture wound of abdominal wall with foreign body without penetration into peritoneal cavity

New code S31.146 Puncture wound of abdominal wall with foreign body, right flank without penetration into peritoneal cavity

New code S31.147 Puncture wound of abdominal wall with foreign body, left flank without penetration into peritoneal cavity

New code S31.14A Puncture wound of abdominal wall with foreign body, unspecified flank without penetration into peritoneal cavity

Add Puncture wound of abdominal wall with foreign body of flank NOS without penetration into peritoneal cavity

S31.15 Open bite of abdominal wall without penetration into peritoneal cavity

New code S31.156 Open bite of abdominal wall, right flank without penetration into peritoneal cavity

New code S31.157 Open bite of abdominal wall, left flank without penetration into peritoneal cavity

New code S31.15A Open bite of abdominal wall, unspecified flank without penetration into peritoneal cavity

Add Open bite of abdominal wall of flank NOS without penetration into peritoneal cavity

S31.6 Open wound of abdominal wall with penetration into peritoneal cavity

S31.60 Unspecified open wound of abdominal wall with penetration into peritoneal cavity

New code S31.606 Unspecified open wound of abdominal wall, right flank with penetration into peritoneal cavity

New code S31.607 Unspecified open wound of abdominal wall, left flank with penetration into peritoneal cavity

New code S31.60A Unspecified open wound of abdominal wall, unspecified flank with penetration into peritoneal cavity

Add Unspecified open wound of abdominal wall of flank NOS, with penetration into peritoneal cavity

S31.61 Laceration without foreign body of abdominal wall with penetration into peritoneal cavity

New code — S31.616 Laceration without foreign body of abdominal wall, right flank with penetration into peritoneal cavity

New code — S31.617 Laceration without foreign body of abdominal wall, left flank with penetration into peritoneal cavity

New code — S31.61A Laceration without foreign body of abdominal wall, unspecified flank with penetration into peritoneal cavity

Add — Laceration without foreign body of abdominal wall of flank NOS, with penetration into peritoneal cavity

S31.62 Laceration with foreign body of abdominal wall with penetration into peritoneal cavity

New code — S31.626 Laceration with foreign body of abdominal wall, right flank with penetration into peritoneal cavity

New code — S31.627 Laceration with foreign body of abdominal wall, left flank with penetration into peritoneal cavity

New code — S31.62A Laceration with foreign body of abdominal wall, unspecified flank with penetration into peritoneal cavity

Add — Laceration with foreign body of abdominal wall, flank NOS, with penetration into peritoneal cavity

S31.63 Puncture wound without foreign body of abdominal wall with penetration into peritoneal cavity

New code — S31.636 Puncture wound of abdominal wall without foreign body, right flank with penetration into peritoneal cavity

New code — S31.637 Puncture wound of abdominal wall without foreign body, left flank with penetration into peritoneal cavity

New code — S31.63A Puncture wound of abdominal wall without foreign body, unspecified flank with penetration into peritoneal cavity

Add — Puncture wound of abdominal wall without foreign body, flank NOS, with penetration into peritoneal cavity

S31.64 Puncture wound with foreign body of abdominal wall with penetration into peritoneal cavity

New code — S31.646 Puncture wound of abdominal wall with foreign body, right flank with penetration into peritoneal cavity

New code — S31.647 Puncture wound of abdominal wall with foreign body, left flank with penetration into peritoneal cavity

New code — S31.64A Puncture wound of abdominal wall with foreign body, unspecified flank with penetration into peritoneal cavity

Add — Puncture wound of abdominal wall with foreign body, flank NOS, with penetration into peritoneal cavity

	S31.65 Open bite of abdominal wall with penetration into peritoneal cavity
New code	S31.656 Open bite of abdominal wall, right flank with penetration into peritoneal cavity
New code	S31.657 Open bite of abdominal wall, left flank with penetration into peritoneal cavity
New code	S31.65A Open bite of abdominal wall, unspecified flank with penetration into peritoneal cavity
Add	Open bite of abdominal wall, flank NOS, with penetration into peritoneal cavity

Foreign Body Entering Into or Through a Natural Orifice

Foreign bodies can enter through natural body orifices. Some of which are benign and cause irritation (i.e., bead in the ear or nose). However other types of foreign bodies can have significant morbidity or mortality.

NCHS has received a request for an additional code in the recently approved expansion which was implemented October 1, 2023. The original proposal was requested by the American Academy of Pediatrics. The American Academy of Pediatrics supports the additional new code.

TABULAR MODIFICATION

	W44.H	Other sharp object entering into or through a natural orifice
	W44.H0	Other sharp object unspecified, entering into or through a natural orifice
	W44.H1	Needle entering into or through a natural orifice Dart entering into or through a natural orifice Hypodermic needle entering into or through a natural orifice Safety pin entering into or through a natural orifice Sewing needle entering into or through a natural orifice
	W44.H2	Knife, sword or dagger entering into or through a natural orifice
New Code	**W44.H9**	**Other sharp object entering into or through a natural orifice**
Add		**Shard pottery entering into or through a natural orifice**

Genetic Neurodevelopmental Disorders

This proposal is a repeat presentation for certain disorders that were previously presented separately in 2023. It provides an alternative option for creation of specific codes for a number of genetic neurodevelopmental disorders.

Neurodevelopmental disorders are coming to be more widely recognized. Many of these have a genetic basis. This current proposal considers certain specific genetic neurodevelopmental disorders, but is not exhaustive.

Detailed clinical presentations for certain specific genetic neurodevelopmental disorders were presented in 2023. At the March 2023 ICD-10 C&M meeting, there were presentations related to the ionotropic glutamate receptor (GRIN1, GRIN2A, GRIN2B, GRIN2D, GRIA1, GRIA2, GRIA3, GRIA4, and GRIK2), SCN2A-related disorders, SLC6A1-related Disorders, and STXBP1-related disorders (syntaxin-binding protein 1). Also, a detailed proposal for specific codes for DLG4-related synaptopathy was presented at the September 2023 C&M meeting. It is recognized that these neurodevelopmental disorders may involve a range of associated findings and conditions, and these may include epilepsy, autism spectrum disorders, intellectual disability, and specific learning and developmental impairments, among other things. Further clinical details are available from the prior proposals and from the references included below.

A separate category for genetic neurodevelopmental disorders is proposed.

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TABULAR MODIFICATIONS

F88 Other disorders of psychological development

Add — Excludes2: genetic neurodevelopmental disorders (F8A.-)

New category — F8A Neurodevelopmental disorders related to specific genetic pathogenic variants

Add — Code also any associated disorders, such as:
Add — attention-deficit hyperactivity disorders (F90.-)
Add — autism spectrum disorder (F84.0)
Add — epilepsy, by specific type (G40.-)
Add — intellectual disabilities (F70-F79)
Add — pervasive developmental disorders (F84.-)

New subcategory — F8A.0 Neurodevelopmental disorders related to pathogenic variants in specific genes

New sub-subcategory — F8A.01 Neurodevelopmental disorders related to pathogenic variants in certain specific genes

New sub-subcategory — F8A.010 Neurodevelopmental disorders, related to pathogenic variants in ion channel genes

New code — F8A.0101 SCN2A-related neurodevelopmental disorder

New code — F8A.0109 Neurodevelopmental disorder related to pathogenic variant in other ion channel gene

Add — SCN8A-related neurodevelopmental disorder

New sub-

subcategory	F8A.011		Neurodevelopmental disorders, related to pathogenic variants in glutamate receptor genes
New code		F8A.0111	GRIN1-related neurodevelopmental disorder
New code		F8A.0112	GRIN2A-related neurodevelopmental disorder
New code		F8A.0113	GRIN2B-related neurodevelopmental disorder
New code		F8A.0114	GRIN2D-related neurodevelopmental disorder
New code		F8A.0115	GRIA1-related neurodevelopmental disorder
New code		F8A.0116	GRIA2-related neurodevelopmental disorder
New code		F8A.0117	GRIA3-related neurodevelopmental disorder
New code		F8A.0118	GRIA4-related neurodevelopmental disorder
New code		F8A.011A	GRIK2-related neurodevelopmental disorder
New code		F8A.0119	Other glutamate receptor, ionotropic, related neurodevelopmental disorder
New code	F8A.012		Neurodevelopmental disorders, related to pathogenic variants in other receptor genes

Note: for SLC6A1 related disorder and related to the expansion for F8A.013 shown immediately below, an alternative option is also shown separately further below.

New sub-subcategory	F8A.013	Neurodevelopmental disorders, related to pathogenic variants in other transporter and solute carrier genes	
New code		F8A.0131	SLC6A1-related disorder
Add			GABA transporter 1 deficiency
New code		F8A.0139	Neurodevelopmental disorder, related to pathogenic variant in other transporter or solute carrier gene
New sub-subcategory	F8A.014	Neurodevelopmental disorders, related to pathogenic variants in synapse related genes	
New code		F8A.0141	Syntaxin-binding protein 1-related disorder
Add			STXBP1-related disorders
New code		F8A.0142	DLG4-related synaptopathy
New code		F8A.0149	Neurodevelopmental disorder, related to pathogenic variant in other synapse related gene
Add			Other genetic synaptopathy
New code	F8A.015	Neurodevelopmental disorders, related to genes associated with transcription and gene expression	
Add			FOXG1 syndrome
New code	F8A.8	Other neurodevelopmental disorders related to pathogenic variants in other specific genes	

Alternative Option for SLC6A1 related disorder / GABA transporter 1 deficiency

E72 Other disorders of amino-acid metabolism

E72.0 Disorders of amino-acid transport

New sub-subcategory	E72.05	Disorders of gamma aminobutyric acid transport	
New code		E72.050	Gamma aminobutyric acid transporter 1 deficiency
Add			GABA transporter 1 deficiency
Add			GAT-1 deficiency
Add			Gamma aminobutyric acid transporter protein type 1 deficiency
Add			SLC6A1-related disorder
Add			Code also any associated disorders, such as:
Add			attention-deficit hyperactivity disorders (F90.-)
Add			autism spectrum disorder (F84.0)
Add			epilepsy, by specific type (G40.-)
Add			intellectual disabilities (F70-F79)
Add			pervasive developmental disorders (F84.-)
New code		E72.058	Other disorders of gamma aminobutyric acid transport
Add			Other GABA transporter deficiency

Note: for this alternative for SLC6A1 related disorder, there would be a single new code for F8A.013, rather than the expansion shown previously.

Gulf War Illness

This topic was presented at the March and September 2023 ICD10 Coordination and Maintenance meeting and based on comments received during the public comment period it is being represented for consideration. Gulf War Illness (GWI) is an exposure-induced chronic multisymptom illness affecting personnel who served in the 1990-1991 Persian Gulf conflict and tied to drug/environment exposures associated with that conflict. A solid body of evidence implicates such exposures, including dose-response and gene-environment interaction data. New, unique, and excessive exposures occurred to multiple agents, distributed differently across deployed personnel. This included exposure to organophosphate nerve agents (sarin, cyclosarin), prolonged exposure to multiple pesticides (including organophosphates and carbamates, among others), pyridostigmine bromide (PB) as a nerve agent pretreatment adjunct, anthrax and botulinum toxoid vaccines and multiple vaccines, permethrin-impregnated uniforms, high concentrate DEET insect repellant, depleted uranium, ciprofloxacin, and other agents. Especially strong evidence ties acetylcholinesterase inhibiting agents including organophosphates (as pesticides and nerve agents) and carbamates (such as PB and carbamate pesticides) to GWI. Changes are indicated in **BOLD**.

Epidemiological studies affirm that deployment to the 1990-1991 conflict and associated exposures are tied to marked increases specifically in an empirically-defined debilitating multisymptom profile. Data show no difference in the fraction of individuals reporting symptoms that have persisted for at least six months in 1-2 defined symptom domains, but a marked difference in the fraction reporting concurrent symptoms across 3-6 distinct symptom domains in Gulf War-deployed personnel vs. Gulf War-era veterans (who were not deployed to the Gulf War Theater). Gulf War illness is distinguished by this identified complex of symptoms consistently characterized across 1990-91 Gulf War-deployed veteran populations.

This health condition requires deployment to the Gulf War Theater of Operations anytime between August 1, 1990, and July 31, 1991. It requires chronic symptoms for ≥ 6 months, arising during or after this deployment, in ≥ 3 of the 6 Kansas criteria questionnaire symptom domains of fatigue/sleep; neurological/cognitive/mood; pain; gastrointestinal; respiratory; and dermatologic. For a domain to qualify for this condition, symptoms in the domain must be either of at least moderate severity (not mild) and/or there must be multiple symptoms in that domain. Currently, there is no existing ICD-10-CM code which adequately captures this health condition.

The consortium of GWI clinicians, researchers, and patient advocates propose the following tabular modifications to aid clinical care of affected veterans, advance epidemiological tracking of this condition and improve health outcomes in affected veterans.

ICD-10 Coordination and Maintenance Committee Meeting
March 20, 2024

TABULAR MODIFICATIONS

T75 Other and unspecified effects of other external causes

T75.8 Other specified effects of external causes

New sub-subcategory
T75.83 Effects of war theater
Add Use additional code to identify associated manifestations

New code
T75.830 Gulf war illness
Add Gulf war syndrome

New code
T75.838 Effects of other war theater

Z77 Other contact with and (suspected) exposures hazardous to health

New subcategory
Z77.3 Contact with and (suspected) exposure to war theater

New code
Z77.31 Contact with and (suspected) exposure to Gulf War theater
Add Contact with and (suspected) exposure to Persian Gulf War theater

New Code
Z77.39 Contact with and (suspected) exposure to other war theater

Hyperoxaluria

This topic was originally presented at the September 2023 ICD10 Coordination and Maintenance meeting. Based on comments received during the public comments period, a revised proposal is being presented for consideration. Changes are indicated in **BOLD**.

Hyperoxaluria is a condition which originates from a diverse group of disorders which all ultimately result in excess levels of oxalate, a toxic metabolite which cannot be broken down by the body and which forms insoluble calcium oxalate. Primary Hyperoxaluria (PH) Types 1 (PH1), 2 (PH2), and 3 (PH3) are caused by distinct inherited genetic mutations and lead to different phenotypes of disease. Other forms of hyperoxaluria including enteric hyperoxaluria (EH) and dietary hyperoxaluria (DH) are caused by a variety of conditions which lead to accumulation of oxalate in the body.

Primary hyperoxaluria (PH) is a group of rare genetic disorders caused by mutations that cause the body to overproduce oxalate. PH1 is caused by a mutation in the AGXT gene and is the most common and severe form of PH, comprising 70-80 percent of known cases.[1,2,3] PH1 impacts both children and adults, and can present as early as infancy.[2,4] Infants with PH1 often present with failure to thrive, nephrocalcinosis, kidney failure, and kidney stones. In a survey of infants with PH1 in both developing and developed nations, the overall infant mortality rate was 52 percent.[6] Older children and adults may present with frequent kidney stones due to build-up of oxalate often requiring emergency room visits, hospitalizations, and surgery. People are often hospitalized for invasive stone-removal procedures (ureteroscopy, percutaneous nephrolithotomy), acute pain from ureteral stones or pyelonephritis, renal colic, urinary tract infection, or vomiting.[1] As renal failure progresses, oxalate accumulates and leads to systemic oxalosis (the spread of oxalate to organs and tissues outside of the kidneys), which can lead to joint damage, bone fractures, skin ulcers, vision loss, heart failure, and death.[5,7,8]

Renal outcomes are meaningfully different among the different subtypes of PH. PH2, caused by a mutation in the GRHPR gene, and PH3, caused by a mutation in the HOGA1 gene, are less prevalent and usually less severe than PH1, but can both contribute to adverse outcomes for patients and are markedly different in terms of severity and age of onset. By age 30, renal survival decreases to 27%, 92%, and 95% for patients with PH1, PH2, and PH3, respectively. By age 60, renal survival decreases further to 12% and 66% for PH1 and PH2 patients, respectively, while PH3 renal survival generally doesn't worsen. PH1 is the most severe phenotype, with up to 70 percent of patients presenting with End Stage Kidney Disease.[9,10,11] Though PH3 is generally the least severe and progresses the most slowly, it generally presents with the earliest symptoms, with a median age of 2.6 years (range from infancy to 31 years). PH1 and PH2 generally have a slightly later onset, with a median age of symptoms at 5.2 years for PH1 (range from infancy to 53 years) and 7.4 years for PH2 (range from infancy to 42 years).[10] PH1 is the only form of PH caused by a liver-specific defect, and the only form of PH where oxalate production comes solely from the liver. The enzymes defective in PH2 and PH3 are also important outside of the liver, and thus excess oxalate production occurs in extra-hepatic sources in the body.[10,12,13] As such, existing treatments such as liver transplantation or new liver-directed therapies targeting the defective AGXT gene which are, or will be, approved are appropriate only for treating PH1.[14]

Enteric hyperoxaluria (EH) occurs as a result of a variety of gastrointestinal disorders promoting fat malabsorption, leading to an excess absorption of dietary oxalate and increased urinary oxalate excretion.[15] Dietary hyperoxaluria (DH) is caused by increased ingestion of oxalate and/or its precursors such as ascorbic acid.[16] In some cases, the causes of hyperoxaluria may be unknown, or idiopathic. Patients with these non-genetic forms of hyperoxaluria can develop recurrent kidney stones or renal damage with progression to CKD or kidney failure depending on severity.[15] Treatment for EH and DH is distinct from genetically-caused PH and depends on treatment of underlying gastrointestinal disorders, intensive dietary modifications, and/or the use of hydration and crystallization inhibitors.[15] Currently, there is no effective way to capture these forms of hyperoxaluria in the coding set. If a specific type of hyperoxaluria is not indicated, coders may use R82.992 (Hyperoxaluria, a nonspecific code for abnormal findings in urine), or incorrectly use E72.53 (PH) for patients who actually have EH or DH.

Alnylam Pharmaceuticals, Incorporated is requesting the following new codes to further differentiate the various types of hyperoxaluria.

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TABULAR MODIFICATIONS

E72 Other disorders of amino-acid metabolism

E72.5 Disorders of glycine metabolism

E72.53 Primary hyperoxaluria
Oxalosis
Oxaluria

Add **Excludes1: secondary hyperoxaluria (E72.54-)**

New code E72.530 Primary hyperoxaluria, type 1

New code E72.538 Other specified primary hyperoxaluria
Add Primary hyperoxaluria, type 2
Add Primary hyperoxaluria, type 3

New code E72.539 Primary hyperoxaluria, unspecified

New sub-subcategory **E72.54 Secondary hyperoxaluria**
Add **Excludes1: primary hyperoxaluria (E72.53-)**

New code **E72.540 Dietary hyperoxaluria**
New code **E72.541 Enteric hyperoxaluria**
New code **E72.548 Other secondary hyperoxaluria**
New code **E72.549 Secondary hyperoxaluria, unspecified**

R82 Other and unspecified abnormal findings in urine
R82.9 Other and unspecified abnormal findings in urine
R82.99 Other abnormal findings in urine
R82.992 Hyperoxaluria

Revise **Excludes1: primary hyperoxaluria (E72.53-)**
Add **secondary hyperoxaluria (E72.54-)**

Hypothalamic obesity

Obesity is defined as abnormal or excessive fat accumulation that presents a risk to health, with assessments using a body mass index (BMI) over 30 kg/m^2 for determining obesity.[1] Obesity, in general, results from imbalance in energy consumption and energy expenditure. Hypothalamic obesity (HO) is a highly unique form of severe obesity characterized by the rapid onset and sustained weight gain that is unresponsive to lifestyle or traditional medical interventions. A proposal for a specific ICD-10-CM diagnosis code for hypothalamic obesity has been received from Rhythm Pharmaceuticals, Inc. HO affects approximately 5,000 to 10,000 patients in the United States and is associated with hyperphagia and decreased physical activity and energy expenditure resulting from an impairment in the MC4R signaling pathway.[2,3,4]

The hypothalamus plays an important role in the body's regulation of appetite and energy balance. The term "hypothalamic obesity" describes obesity caused by injury or damage to the hypothalamus, in contrast to other forms of obesity related to the hypothalamic dysfunction such as genetic disorders, like Prader-Willi syndrome, Bardet Biedl Syndrome, or malformations of the brain.[5] Damage to the hypothalamus disrupts the balance between caloric intake and expenditure, often leading to more calories consumed than needed for the body to properly function. This results in rapid weight gain that is very difficult to control.[5]

HO sometimes presents with hyperphagia which is the lack of feeling full (satiety) and constant insatiable feelings of extreme hunger. However, the clinical presentation may vary depending on the type and location of sustained hypothalamic injury. Some individuals, for example, do not experience hyperphagia but still gain weight rapidly because of decreased energy expenditure, both at rest and/or during physical activity leading to caloric imbalance even when food intake is restricted.[5]

A diagnosis of hypothalamic obesity is suspected when an individual experiences rapid and excessive weight gain relative to a hypothalamic injury. Providers may rely on brain imaging studies and/or other diagnostic tests to identify deficits in one or more hypothalamic/pituitary hormones. These tools help differentiate HO from other types of obesity caused by genetics, lifestyle or other factors.[5]

Specific structures within the hypothalamus that regulate both peripheral energy expenditure and satiety include the arcuate nucleus, paraventricular nucleus, dorsomedial nucleus, and dorsal hypothalamic nuclei, all found in the medial hypothalamus.[6] Injury to hypothalamic structures may occur as a result of the surgical removal or resection of a hypothalamic brain tumor, complications related to the tumor, radiotherapy treatment, and in rare cases inflammation and/or other trauma to the area.[7,8]

Craniopharyngiomas, germinomas, gliomas, hamartomas and pituitary adenomas are tumors that arise from the pituitary gland or invade the hypothalamus.[9] Up to 50% of patients who are treated for craniopharyngioma by tumor removal surgery and/or external beam radiation therapy develop HO.[7,10] Individuals who acquire HO following surgery or radiation may have been of normal weight prior to their tumor diagnosis but became obese following surgery. Individuals with hypothalamic damage due to other causes, such as traumatic brain injury or inflammation, tend to begin gaining weight at

the time of the injury or very shortly after treatment.[5] Both of these types of hypothalamic injuries cause damage of varying degree to the ventromedial nuclei (satiety center) and the lateral hypothalamic area (feeding center).

In addition to excessive weight gain, injury to the hypothalamus may also result in other disorders such as disordered sleep and circadian phase regulation, pituitary hormone deficiencies and psychosocial factors that influence healthful behaviors.[11] Some may experience difficulties maintaining normal body temperatures, have challenges regulating heart rate and blood pressure, or experience seizures. Individuals diagnosed with HO should undergo endocrinology assessment for related hypothalamic/pituitary hormone deficiencies such as diabetes insipidus (vasopressin deficiency), secondary adrenal insufficiency (ACTH deficiency), secondary hypothyroidism (TSH deficiency), growth hormone deficiency, and hypogonadism (deficiency of GNRH leading to deficiency of sex steroids or estrogen in females and testosterone in males). Monitoring for potential health consequences of obesity, such as fatty liver disease, type 2 diabetes mellitus, abnormal lipid profile, sleep-disordered breathing, pseudotumor cerebri syndrome, orthopedic problems and psychosocial/mental health disorders should also occur.[5]

Unfortunately, there are no approved therapeutics for HO, however experimental therapeutics are in development and undergoing clinical evaluation. Current treatment requires evaluating the underlying condition and attempting a variety of strategies to manage the patient's obesity through a well-coordinated, multi-disciplinary team who can implement an individualized treatment approach. This may include monitoring for early detection of tumor recurrence (for tumor survivors), pituitary hormone replacement, nutrition, physical activity and exercise, mental and behavioral health interventions and sometimes weight loss medications and/or or metabolic/bariatric surgery.[5]

In conclusion, hypothalamic obesity is a distinct condition that occurs when damage to the hypothalamus causes an imbalance between someone's energy intake from food and the amount of energy their body has used those results in adipose tissue accumulation. It is different from other forms of obesity related to lifestyle, genetic disorders, or malformations of the brain. Hypothalamic dysfunction broadly may result in impairment of the multiple body functions which the hypothalamus ordinarily helps regulate such as: body temperature, emotions, behavior, memory, growth, sex drive, and the sleep-wake cycle. In addition, hypothalamic dysfunction characterizes the impact of the hypothalamus on controlling the pituitary gland creating a hormone imbalance. In order to properly identify, diagnose and track patients with HO and the clinical interventions used to treat and manage them, including individualized specialized treatments currently available, as well as new treatments currently in development, a specific ICD-10-CM code has been requested.

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TABULAR MODIFICATIONS

E23 Hypofunction and other disorders of the pituitary gland

E23.3 Hypothalamic dysfunction, not elsewhere classified
Excludes1: Prader-Willi syndrome (Q87.11)
Russell-Silver syndrome (Q87.19)

New code	E23.31	Hypothalamic obesity
Add		Use additional code, if applicable, to identify associated manifestations, such as polyphagia (R63.2)
Add		Use additional code, if known, to identify body mass index (BMI) (Z68.-)
New code	E23.39	Other hypothalamic dysfunction

Kabuki Syndrome

Kabuki syndrome (KS) is a rare genetic disorder affecting multiple body systems and requires comprehensive medical management[1]. A specific code for KS will assure accurate diagnoses, research, and healthcare management for individuals with KS.

Kabuki syndrome is distinct and cannot be grouped with other congenital malformations. KS's unique and specific phenotypic presentation is caused by mutations in genes of histone modification, including *KMT2D* and *KDM6A*[2,3]. Patients are identified by genetic testing and characteristic phenotypes including distinct facial features, developmental delay, intellectual disability, heart defects, hypotonia, skeletal anomalies, and immune dysfunction[4]. KS requires accurate diagnosis in order for patients to receive proper treatment, particularly with precision KS therapy currently in development.

The current coding options, fails to distinguish KS from other congenital malformations, resulting in a loss of essential clinical information. It limits the ability to track and analyze KS's unique clinical characteristics, medical comorbidities, and healthcare utilization. As a result, important data related to the prevalence, treatment outcomes, and healthcare resource requirements for KS remain obscured, and medical reimbursement is delayed.

Although there are no known cures for KS, at least eight treatments specifically for KS are currently in development. One has received rare pediatric disease and orphan drug designations from the U.S. FDA[5]. Another treatment that was being developed for KS received orphan drug designation from the U.S. FDA and European Commission in 2018[6]. These designations indicate that the FDA and EMA consider KS a separate indication that cannot be grouped with others in ICD coding.

Current best estimates for the prevalence of KS are 1:32,000-86,000 live births[7,8] (figures that were estimated in 1988 and 2004, before genetic testing for KS was available). This prevalence is similar to other rare disorders that have been provided codes, including Prader-Willi syndrome (Q87.11), SYNGAP1 Encephalopathy (F78.A1) and Angelman syndrome (Q93.51); these syndromes also have intellectual disabilities and developmental delay as hallmark symptoms. Without a specific code, KS patients cannot be tracked to corroborate prevalence estimates and document morbidity and mortality of the condition, critical for informing public health initiatives and patient care.

A unique code will improve patient care by enabling accurate diagnosis, leading to better clinical management and patient outcomes. A specific code will ensure continuation of proper treatment, aiding in the early identification of potential complications and comorbidities, as well as more easily enabling potential retrospective studies for best practices in clinical care. In addition, a specific KS code facilitates the collection of evidence-based data, accelerating research on the syndrome's pathogenetic mechanisms, natural history, and therapeutic interventions, as well as helping discover and recruit participants for clinical studies/trials and patient registries. A dedicated code improves patient care, accelerates research and promotes health equity.

In summary, the creation of a dedicated ICD-10-CM code for KS has the potential to transform the landscape of KS research, treatment, and awareness. Accurate identification and documentation of this specific rare condition will advance public health knowledge and the well-being of affected individuals.

This proposal is submitted by and with contributions made from the following clinicians:

- Clara Tang, PhD, Director of Research at the KSF
- Bruce Bloom, JD, DDS, Chief Science Officer at the KSF
- Olaf Bodamer MD, PhD, Director of the Roya Kabuki Program at Boston Children's Hospital and Associate Professor at Harvard Medical School
- Margaret Adam, MD, Professor at University of Washington
- Jacqueline Harris, MD, Director of the Epigenetics Clinic at Kennedy Krieger Institute and Associate Professor at Johns Hopkins University School of Medicine
- Hans Bjornsson, MD, PhD, Associate Professor at Johns Hopkins University and Professor at the University of Iceland
- Brittany Simpson, MD, Assistant Professor at Cincinnati Children's Hospital Medical Center
- Ian Krantz, MD, Professor at The Children's Hospital of Philadelphia
- Drea K. Petersen, MD, Clinical Geneticist and Pediatrician at Randall Children's Hospital and Legacy Health

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TABULAR MODIFICATIONS

Q89 Other congenital malformations, not elsewhere classified

Q89.8 Other specified congenital malformations
Use additional code(s) to identify all associated manifestations

New subcategory	Q89.81 Kabuki (Niikawa-Kuroki) syndrome
New code	Q89.810 Kabuki syndrome, type 1
New code	Q89.811 Kabuki syndrome, type 2
New code	Q89.818 Other Kabuki syndrome
New code	Q89.819 Kabuki syndrome, unspecified

Ledderhose Disease/Plantar Fibromatosis & Plantar Fasciitis

Ledderhose Disease is a genetic disease, in the same family of diseases as Dupuytren's contacture and Peyronie's Disease. It leads to growth of painful fibrous nodules known as fibromas on the plantar surface of the feet that can be disabling to many. The nodules are most commonly found on the medial and central band of the plantar fascia in the midfoot and forefoot. Nodules are consistently painful and worsening after long periods of standing or ambulation.

Plantar fasciitis is an inflammatory and, at times, degenerative condition of the plantar fascia related to chronic or repetitive strain. Typically, symptoms present as pain first step in the morning with improvement in ambulation.

Currently, both Ledderhose Disease/Plantar Fibromatosis & Plantar fasciitis share the same ICD-10-CM code of M72.2. In addition, there is no laterality assigned to this code. The two entities are very different in presentation, different in treatment and are of different causes. There has been difficulty in tracking Ledderhose Disease with respect to incidence and prevalence due to the lack of a unique diagnosis code.

Podiatrists, Dr. Paul Carroll and Dr. Eddie Davis, are requesting new ICD-10-CM codes to distinguish between the two conditions and better track these patients.

TABULAR MODIFICATIONS

M67 Other disorders of synovium and tendon

New subcategory	M67.A	Plantar fasciitis	
New code		M67.A0	Plantar fasciitis, unspecified site
Add			Plantar fasciitis NOS
New sub-subcategory		M67.A1	Plantar fasciitis, heel
New code			M67.A10 Plantar fasciitis, unspecified heel
New code			M67.A11 Plantar fasciitis, right heel
New code			M67.A12 Plantar fasciitis, left heel
New code		M67.A9	Other plantar fasciitis
Add			Plantar fasciitis midfoot

	M72	Fibroblastic disorders
		M72.2 Plantar fascial fibromatosis
Delete		~~Plantar fasciitis~~
Add		Ledderhose disease
New code		M72.20 Plantar fascial fibromatosis, unspecified foot
New code		M72.21 Plantar fascial fibromatosis, right foot
New code		M72.22 Plantar fascial fibromatosis, left foot

Leukocyte Adhesion Deficiency Type I (LAD-I)

Leukocyte adhesion deficiency type I (LAD-I) is a rare genetic disorder caused by mutation(s) in the *ITGB2* gene that result in impaired production of CD18[1]. CD18, the beta subunit of the β2 integrins, is a glycoprotein on the surface of white blood cells that mediates their migration from inside blood vessels to sites of infection or inflammation in tissues. Because the white blood cells with *ITGB2* mutation(s) can't get to the site of infection, patients with LAD-I have an impaired ability to fight infections, especially at sites of microbial entry, and have an abnormal hyperinflammatory response to infections. Patients are especially susceptible to recurrent atypical bacterial or fungal infections such as peri-rectal infections, as well as abnormal inflammation of tissues such as periodontitis and gingivitis. The clinical severity depends on the level of preserved CD18 on the surface of neutrophils. Mortality for patients with severe LAD-I has been reported at 60-75% by the age of 2 years[2]. Diagnosis depends on an astute clinician considering the possibility of LAD-I deficiency in a child with omphalitis, other atypical infections, or recurrent infections associated with very high neutrophil counts. However, given the rarity of LAD-I, most clinicians have never made the diagnosis or treated a patient with LAD-I.

The incidence of LAD-I is estimated to be under 10 in 1,000,000 live births[3,4]. Given the impediments to diagnosis and the greatly shortened life expectancy, the exact incidence of LAD-I is not well-established and may increase as recognition of the disease continues to grow. To quote the National Organization for Rare Diseases[5]:

> *Leukocyte adhesion deficiencies often go unrecognized and may be misdiagnosed, making it difficult to determine their true frequency in the general population.*

As of 2009, one author reported approximately 300 cases of LAD-I worldwide[6].

Frequent infections (93% of patients) and poor wound healing (86%) beginning as early as the neonatal period are the two most common presenting symptoms of LAD-I[7]. Common infections in LAD-I patients include pneumonia, gingivitis, and peritonitis, all of which can be life-threatening. Patients with severe disease (caused by <2% expression of CD18) present with frequent, severe infections which can be life-threatening[8]. Patients with severe disease have very poor prognosis without hematopoietic stem cell transplant[8]. Patients with moderate disease (2% to 30% expression of CD18) have less frequent and less severe infections but still have multiple infections affecting the skin and mucosal surfaces. Although these patients can survive to adulthood with adequate treatment[8], mortality exceeds 50% by the age of 40 years[2].

Suspicion of LAD-I may be raised by inflammation (omphalitis) and delayed separation of the umbilical stump, which occurs in 58-84% of patients[2]. The diagnosis can also be suspected in infants with recurrent soft tissue infections, especially in those who present with abscess-like lesions without pus and in those who present with infections with an exceedingly high white blood cell count. A flow cytometry analysis demonstrating the absence of functional CD18 and the associated alpha subunit molecules (CD11) on the surface of leukocytes provides the definitive diagnoses of LAD-I[9]. This diagnosis is then confirmed with genetic testing to define the exact molecular defect.

LAD-I results from mutations in the *ITGB2* gene and is unique from the other two types of LAD (LAD-II and LAD-III) in molecular cause, diagnosis, and symptoms. LAD-II is caused by a mutation of the *SLC35C1* gene, is definitively diagnosed via flow cytometry analysis showing the absence of CD15a, and typically is associated with milder infections and no omphalitis. LAD-III is caused by a mutation in the *FERMT3* gene (also called kindlin-3 gene) and is differentially diagnosed from LAD-I via molecular testing. It commonly causes bleeding complications, bone marrow failure, and osteoporosis.

Treatment of LAD-I includes management of the repeated, prolonged infections that characterize the disease, and often involves hospitalization, long courses of antibiotic and antimicrobial medications, and preventative isolation or formation of limited social pods to minimize risk of infection. Allogeneic hematopoietic stem cell transplant (HSCT) is currently the only curative treatment. However, a high incidence of graft rejection and acute graft vs host disease (aGVHD) both pose barriers to HSCT success[6]. When HSCT is performed in LAD-I patients, 17% of patients experience graft failure and 24% experience grade II to IV acute GVHD by 100 days, with 8% of patients experiencing lethal GVHD[10].

An ICD-10-CM code would aid physicians in improving patient care by better ensuring appropriate patient treatment and making it possible to track clinical results of interventions. This is extremely important in addressing these patients' atypical serious infections and wound healing limitation. An appropriate code would intrinsically justify prolonged courses of antibiotics and would help identify a population of patients with an increased risk of complications related to otherwise standard management approaches. As an example, as a result of poor wound healing and excessive inflammation, patients with LAD-I can have significant complications resulting from standard-of-care procedures (such as routine incision and drainage) that are very well-tolerated in most other conditions.

In addition, it would promote effective communication across health care teams. Close monitoring is essential to ensuring proper care of LAD-I patients since infections typically require prolonged treatment and any medical intervention creating a wound, such as a surgery, biopsy, or circumcision, has the potential to be complicated by poor wound healing, abscess formation, and potentially fatal microbial dissemination.

An ICD-10-CM code can also help in identifying patients for clinical trials. Finally, an ICD-10-CM code will improve the ability to verify the prevalence and natural history of the disease, make genotype-phenotype correlations, and enable tailored and long-term monitoring of outcomes from future therapies.

Although no other curative therapies are currently available, an investigational genetic therapy for severe LAD-I being developed by Rocket Pharmaceuticals has demonstrated 100% 12-month survival in a Phase I/II study and has a PDUFA review date established with the FDA for March 31, 2024. An ICD-10 code for LAD-I will ensure that patients can be identified, and their outcomes

tracked while receiving this new therapy as well as more conventional hematopoietic stem cell transplants.

This proposal for a specific code for LAD-I is submitted by Susan Prockop, MD, Boston Children's Hospital and Magnolia Innovation with assistance from the medical team at Rocket Pharmaceuticals, Inc.

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TABULAR MODIFICATIONS

	D71	Functional disorders of polymorphonuclear neutrophils
Delete		~~Cell membrane receptor complex [CR3] defect~~
Delete		~~Chronic (childhood) granulomatous disease~~
Delete		~~Congenital dysphagocytosis~~
Delete		~~Progressive septic granulomatosis~~
New Code	D71.1	Leukocyte adhesion deficiency type I (LAD-I)
Add		LAD-I
New Code	D71.8	Other functional disorders of polymorphonuclear neutrophils
Add		Cell membrane receptor complex [CR3] defect
Add		Chronic (childhood) granulomatous disease
Add		Congenital dysphagocytosis
Add		LAD-II
Add		LAD-III
Add		Leukocyte adhesion deficiency type II
Add		Leukocyte adhesion deficiency type III
Add		Progressive septic granulomatosis

Lynch Syndrome

Lynch syndrome is the most common cause of inherited colorectal and endometrial cancer and accounts for about 4% of colorectal cancers and 3% of endometrial cancers. It is estimated to affect one in 279 people in the general population. It is caused by pathogenic variants in genes in the DNA mismatch repair pathway that maintain fidelity during replication (MLH1, MSH2, MSH6, PMS2, EPCAM). These are inherited in an autosomal dominant fashion, meaning offspring have a 50% risk of inheriting the problematic gene causing Lynch syndrome independent of sex.

Lynch syndrome is characterized by increased risk of cancer in multiple organ systems. The lifetime risk varies by gene but includes elevated risks of colorectal cancer up to 46-61%, endometrial cancer up to 34-54%, ovarian cancer up to 8-38%, urinary tract cancer up to 2.2 - 28%, gastric cancer up to 9% as well as increased but less defined risks of small bowel, pancreas, biliary tract, and prostate cancer. In addition to the high lifetime risks of these cancers, the cancers are also found to occur significantly earlier in life in comparison to sporadic cancers, with the average age of colon and endometrial cancer being in the forties. The cancer risks vary dramatically by gene.

Cancers resulting from Lynch syndrome have a characteristic feature called microsatellite instability. This is the finding of abnormal expansion or contraction of repetitive sequences in tumor DNA called microsatellite repeats. Although this can be found in sporadic cancers, it is much more common in cancers in patients with Lynch syndrome. This can be easily tested by staining pathology specimens of tumor tissue with immunochemistry techniques or through direct microsatellite instability testing. This has led to recommendations for universal testing of all colorectal cancers initially with subsequent expansion to include all endometrial cancers to identify patients at risk for Lynch syndrome. This universal testing is widely endorsed by medical societies across the world. In addition to its use in identifying patients at risk for Lynch syndrome, microsatellite instability is also the feature of tumors that indicates an increased likelihood of response to immunotherapy with immune checkpoint inhibitors and is thus also critical in optimizing cancer treatment plans. Germline genetic testing allows for testing for inherited pathogenic variants prior to a cancer diagnosis. This has been available for Lynch syndrome since the 1990s.

The creation of new ICD-10-CM codes to specifically identify patients with Lynch syndrome has been proposed by Fight Colorectal Cancer (Fight CRC) and its Genetics and Family History Advisory Council, which includes national leaders in the field of hereditary gastrointestinal cancers from multiple disciplines and institutions, with support from American Cancer Society, American College of Gastroenterology, American Society of Gastrointestinal Endoscopy, Collaborative Group of the Americas on Inherited Gastrointestinal Cancer, Lynch Syndrome Screening Network, and other groups as well as individual experts in the field. Since the cancer risks vary dramatically by gene, they have proposed ICD-10-CM codes for Lynch syndrome specifically indicating which of the five genes is responsible.

Creation of unique ICD-10-CM codes for Lynch syndrome will improve patient care in multiple ways. This includes facilitating access to multidisciplinary treatment including genetic counseling, colonoscopy, upper endoscopy, prophylactic surgery, and immunotherapy with checkpoint inhibitors.

Unique codes will also impact clinical care by making it possible to track outcomes from clinical interventions and therapies and to help provide evidence to optimize guidelines and ensure consistency between different specialists. In addition, this will aid in the implementation of approved national quality measures ensuring adequate performance of Lynch syndrome screening measures. Finally, these codes will also help aid research that is needed to measure and improve identification and clinical care of Lynch syndrome patients.

TABULAR MODIFICATIONS

Z15 Genetic susceptibility to disease

Z15.0 Genetic susceptibility to malignant neoplasm

New sub-subcategory	Z15.05	Genetic susceptibility to malignant neoplasm of digestive system	
New sub-subcategory Add		Z15.050	Lynch syndrome Hereditary nonpolyposis colorectal cancer susceptibility
New code			Z15.0500 Lynch syndrome, unspecified
New code			Z15.0501 MLH1 Lynch syndrome
New code			Z15.0502 MSH2 Lynch syndrome
New code			Z15.0503 MSH6 Lynch syndrome
New code			Z15.0504 PMS2 Lynch syndrome
New code			Z15.0505 EPCAM Lynch syndrome
New code		Z15.058	Other genetic susceptibility to malignant neoplasm of digestive system

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INDEX MODIFICATIONS

	Genetic
	- susceptibility to disease NEC Z15.89
	- - malignant neoplasm Z15.09
Add	- - - digestive Z15.05-
Add	- - - - Lynch syndrome Z15.0500
Add	- - - - - EPCAM Z15.0505
Add	- - - - - MLH1 Z15.0501
Add	- - - - - MSH2 Z15.0502
Add	- - - - - MSH6 Z15.0503
Add	- - - - - PMS2 Z15.0504
Add	Lynch syndrome – see Syndrome, Lynch
	Susceptibility to disease, genetic Z15.89
	- malignant neoplasm Z15.09
Add	- - digestive Z15.05-
Add	- - - Lynch syndrome Z15.0500
Add	- - - - EPCAM Z15.0505
Add	- - - - MLH1 Z15.0501
Add	- - - - MSH2 Z15.0502
Add	- - - - MSH6 Z15.0503
Add	- - - - PMS2 Z15.0504
	Syndrome -see also Disease
Add	- Lynch Z15.0500
Add	- - EPCAM Z15.0505
Add	- - MLH1 Z15.0501
Add	- - MSH2 Z15.0502
Add	- - MSH6 Z15.0503
Add	- - PMS2 Z15.0504

Target of (perceived) adverse discrimination and persecution

The National Center for Health Statistics (NCHS) received a request to expand the ICD-10-CM code category Z60.5, Target of (perceived) adverse discrimination and persecution from members of the American Medical Association (AMA). The proposed new code will identify patients presenting with conditions related to experiencing racism and discrimination, including systemic racism and unconscious bias. The AMA is advocating for this new ICD-10-CM code because it will provide physicians with a necessary tool within the clinical encounter to address racism and discrimination.

The existing ICD-10-CM code Z60.5, Target of (perceived) adverse discrimination and persecution, is not specific enough. A new code will be different from this existing code in that it will focus on the patient's unique experience. The new proposed new code will identify conditions related to experiencing racism and discrimination and its effect on the patient's health status. The capture of this information and later data analysis is needed to provide more fair and effective patient care. The new code will also open up an important conversation between the patient and physician about the effect of the social environment on health outcomes and help inform policy approaches.

Research is continually clarifying the full impact that racism and discrimination have on health. It is known that longstanding racism has negatively impacted communities of color. Data show higher rates of illness and death related to a range of conditions, such as hypertension, heart disease, diabetes, and asthma, among minoritized racial and ethnic groups compared to White people.[1] For example, according to the Centers for Disease Control and Prevention, Blacks and Hispanics have the highest percentage of people with asthma in the United States.[2] Studies have also shown that discrimination, specifically racial discrimination is linked to mental health issues such as post-traumatic stress disorder, anxiety, substance use disorder, and depression. A 2023 report by the Alzheimer's Association highlighted the impact of racism on health and that Black patients are about twice as likely than whites to develop dementia.[3]

Anti-racism efforts can close gaps in health for minoritized patients while also improving the health of white patients.[4] To improve health for everyone and help close the gap in health opportunity of minoritized patients, there is a need to capture data on experiences with racism and discrimination.

The AMA is requesting new ICD-10-CM codes that will identify patients presenting with conditions related to experiencing racism and discrimination, including systemic racism and unconscious bias.

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TABULAR MODIFICATION

	Z60	Problems related to social environment
	Z60.5	Target of (perceived) adverse discrimination and persecution
		Excludes1: social exclusion and rejection (Z60.4)
New code	Z60.50	Target of (perceived) adverse discrimination and persecution, unspecified
New code	Z60.51	Target of (perceived) adverse discrimination and persecution, due to racism
Add		Target of (perceived) adverse discrimination and persecution due to racial intolerance

Thyroid eye disease

Thyroid eye disease (TED) also known as Graves' Orbitopathy (Ophthalmopathy) (GO) is a sight threatening autoimmune disease occurring most commonly in hyperthyroid patients, and less frequently in hypothyroid and euthyroid patients. In patients with TED, anomalies in the immune system trigger the production of pathogenic anti- thyroid-stimulating hormone receptor (anti-TSHR) antibodies. These antibodies stimulate TSHR signaling and activate orbital fibroblasts, leading to enhanced cell differentiation and proliferation. This causes expansion of the orbital fat tissue, followed by fibrosis. Moreover, there is an upsurge in the production of inflammatory cytokines, resulting in orbital inflammation. An augmented secretion of extracellular matrix components further contributes to tissue remodeling and the enlargement of orbital muscles and overall orbital tissues.

Patients with TED can manifest signs and symptoms on a spectrum ranging from mild to severe and sight threatening unilaterally or bilaterally. The most common symptoms are dry eyes, conjunctival redness, periorbital swelling, eyelid retraction, diplopia, and proptosis. Studies have shown that the physical disfiguration and visual disruptions associated with TED diminishes patients' quality of life, psychosocial health, and result in limitations on their ability to work[1,2,3]. Until recently, the primary management of TED patients typically involved over-the-counter eye drops, while oral or intravenous corticosteroids were used for patients with persistent inflammatory symptoms. For those who developed sustained ocular changes from TED despite the resolution of inflammation, procedures such as orbital decompression surgery, strabismus surgery or eyelid surgery are necessary to mitigate symptoms and avert blindness[4]. The first biologic therapy specifically targeting TED was introduced in 2020[5], with another advanced therapy currently in phase 3 clinical trials[6].

In clinical practice, the diagnosis of TED is primarily based on a combination of medical history, presenting symptoms, radiographic imaging, and laboratory results (e.g., TSH, T3, T4, and TSH receptor antibody levels, TRAb or TSI)[7]. Several assessment tools have been devised for grading the degree of functional impairment, inflammation, and severity of TED. These grading systems include Clinical Activity Score (CAS), Gorman-Bahn diplopia score, VISA (Vision, Inflammation, Strabismus, and Appearance), and the European Group of Graves' Orbitopathy (EUGOGO) severity classifications[8].

Currently, ICD-10-CM lacks a specific code to identify TED patients, significantly complicating their identification. While scientific literature has made attempts to classify TED patients using varied coding algorithms, these methodologies differ widely across publications and lack validation, raising concerns about potentially misidentifying TED patients and misrepresenting their medical journey. The paucity and inconsistency of available data on TED incidence and prevalence further highlight the challenge of tracking the disease without clearly defined classifications. Estimates vary widely, suggesting that there are anywhere from 21 per 100,000 to 90 per 100,000[8,9]patients with TED in the United States, and that 25% to 50% of individuals with Graves' Disease will develop TED, illustrating the considerable uncertainty surrounding these figures[10,11,12].

Immunovant, Incorporated is requesting the following tabular modifications to enable improved identification of individuals with TED, improve longitudinal follow-up via EHR and facilitate focused research in this population.

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TABULAR MODIFICATIONS

H05 Disorders of orbit

H05.8 Other disorders of orbit

New sub-subcategory	H05.83	Thyroid orbitopathy
Add		Graves' ophthalmopathy
Add		Graves' orbitopathy
Add		Thyroid eye disease
Add		Code also, if applicable, any associated conditions such as: autoimmune thyroiditis (E06.3) thyrotoxicosis with diffuse goiter (E05.0)
New code	H05.831	Thyroid orbitopathy, right eye
New code	H05.832	Thyroid orbitopathy, left eye
New code	H05.833	Thyroid orbitopathy, bilateral
New code	H05.839	Thyroid orbitopathy, unspecified eye

Topical steroid withdrawal

Topical corticosteroids (TCS) are first-line therapies for Atopic Dermatitis (AD) and other inflammatory dermatological conditions [1]. While short-term low- to mid-potency TCS monotherapy is likely safe and efficacious, there are well-established adverse effects to higher-potency or longer-term us of TCS such as skin atrophy, telangiectasia, and striae, as well as systemic side effects, including Hypothalamus-Pituitary-Adrenal (HPA) Axis suppression [2][3]. In recent decades, there has been a concerning rise in severe systemic adverse reactions due to long-term use and abrupt cessation of moderate- to high-potency TCS, commonly referred to as Topical Steroid Withdrawal (TSW) [4][5][6]. TSW is the most common term used to describe this syndrome, there are alternate names such as "Red Skin Syndrome, Topical Steroid Addiction, Steroid Withdrawal Syndrome, Steroid Rosacea, and Steroid-induced Dermatitis" among others. The National Eczema Association, additionally, acknowledges TSW as a separate clinical entity from AD and highlights the absence of a formal diagnostic criteria for TSW [4]. This lack of standardized criteria and inconsistency in naming create constraints in conducting population studies.

The most up-to-date literature on TSW demonstrates an increased prevalence of TSW in adult (83.1%) females (78.9%) [4]. The primary indication for TCS use was cosmetic (61.4%), atopic dermatitis (14.2%) and acne (11.2%) and location of TCS use was also predominantly on the face (97.4%) [4]. Potency of TCS used was mostly moderate (68.9%) or high (20.85%) and duration of usage was typically 6 months or more [4]. The etiology of TSW in literature is strongly correlated with the potency and duration of topical corticosteroid (TCS) use.

Clinical findings associated with TSW that are distinct from atopic dermatitis include thermodysregulation, neurogenic pain, burning sensation, telangiectasia, skin atrophy, oozing containing a metallic smell, and specific cutaneous signs such as the "red sleeve sign" and "elephant wrinkles" [4][8]. These symptoms and morphological features are often notably distinct from the patient's primary dermatoses and may manifest in regions of the body where TCS were never applied. Two subtypes of TSW have been proposed: an erythematous-edematous subtype in patients with underlying eczematous dermatosis and a papulopustular subtype in patients who used TCS for cosmetic or acneiform conditions [4][7].

The management and treatment of TSW focus on symptom relief and promoting skin healing. Strategies include gradual tapering of TCS, regular use of moisturizers and emollients to repair skin barrier and alleviate dryness, wet wrap therapy, dupilumab, psychological support and lifestyle adjustments. However, no current therapy appears to offer more than symptomatic control.

Currently, there is no ICD-10-CM code for TSW. Diagnosis often relies on clinical evaluation, patient history of TCS use, and exclusion of other potential causes of the symptoms. The distinct clinical features and course of TSW is inadequately characterized by existing diagnosis codes as symptoms are not a flare of existing AD or other skin condition, but of another clinical condition. The availability of a specific code would increase awareness among healthcare providers about the

existence and significance of TSW as a potential complication of topical corticosteroid use, thereby guiding the judicious application of high-potency TCS in the management of atopic dermatitis. Patients with eczematous dermatitis after >4-6 months of TCS use who have additional symptoms of thermodysregulation and cutaneous neurogenic pain should be diagnosed under this new code instead of the existing codes for "atopic dermatitis" or "rash and other nonspecific skin eruption."

The International Topical Steroid Awareness Network, with the support of National Eczema Association, Allergy & Asthma Network, and National Institute of Allergy and Infectious Disease allergy training program leadership is requesting the following tabular modifications.

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TABULAR MODIFICATIONS

L30 Other and unspecified dermatitis

L30.8 Other specified dermatitis

New code	L30.81	Topical steroid withdrawal syndrome
Add		Red skin syndrome
New code	L30.89	Other specified dermatitis

Type 2 diabetes mellitus in remission

People with type 2 diabetes mellitus (T2DM) should be considered in remission after sustaining normal blood glucose (sugar) levels for three months or more, according to a new consensus statement from the American Diabetes Association® (ADA), the Endocrine Society, the European Association for the Study of Diabetes and Diabetes UK jointly published in 2021 in Diabetes Care, the Journal of Clinical Endocrinology & Metabolism, Diabetologia, and Diabetic Medicine, respectively.

The choice of terminology has implications for clinical practice and coding accuracy. Several terms have been proposed for people who have become free of a previously diagnosed disease state. In T2D, the terms "resolution," "reversal," "remission," and "cure" have been used to describe a favorable outcome of interventions resulting in a disease-free status. In agreement with the prior consensus group's conclusions [1], this expert panel concluded that diabetes remission is the most appropriate term [2]. It strikes a proper balance, noting that diabetes may not always be active and progressive yet implying that a notable improvement may not be permanent. It is important to note that the term "cure" has not been applied to T2DM, as weight regain is always a risk factor for its re-occurrence. Additionally, while the terms "reversal" and "remission" are used interchangeably, recent consensus supports the use of "remission" in the context of T2DM. Furthermore, a distinction could be made between mere reversal (return to normoglycemia) and true remission (normoglycaemia maintained for at least three months in the absence of glucose-lowering drugs)

Permanente Medicine is requesting the following tabular modification to report the clinical status of regression along the diabetes continuum in alignment with the current clinical guidelines.

References
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TABULAR MODIFICATIONS

E11 Type 2 diabetes mellitus
E11.9 Type 2 diabetes mellitus without complications

New code E11.A Type 2 diabetes mellitus in remission
Add Use additional code(s) to identify all associated manifestations

Usher Syndrome

This topic was presented at the September 2023 ICD10 Coordination and Maintenance meeting. Based on public comments, revisions to the proposal have been made for reconsideration. Usher syndrome (USH) is a hereditary disorder with well-defined genetic causation that results in impairment of both hearing and vision. USH is a recessive genetic disorder that is responsible for 50% of those with hereditary deafblindness[1]. Hearing loss varies in age of onset and severity as described further below. Retinitis pigmentosa, the visual component of USH, is a progressive and untreatable retinal degeneration that initially causes nightblindness followed by loss of peripheral vision and finally impaired central vision. USH also affects balance in some patients. Variants in at least 12 genes have been identified as causing USH with a combined estimated incidence of 4-17:100,000[1]. Three clinically identifiable categories of USH account for the majority of patient presentations and are characterized by age of onset and severity of symptoms[2]:

• Type 1: Children have profound hearing loss or deafness at birth and may have severe balance problems that may lead to delayed motor milestones. Many obtain little or no benefit from hearing aids, but early use of cochlear implants may allow for development of speech. Decreased night vision by age 10, progressing to severe vision loss by midlife.
• Type 2: Moderate to severe hearing loss at birth. Normal balance. Decreased night vision by adolescence, progressing to severe vision loss by midlife.
• Type 3: Progressive hearing loss in childhood or early teens. Normal to near-normal balance in childhood. Chance of later problems. Vision loss varies in severity and age of onset; night vision problems often begin in teens and progress to severe vision loss by midlife.

Given the current absence of a unique ICD-10-CM code that simultaneously captures the auditory and visual manifestations of USH, clinicians are forced to choose among several non-specific codes including: H35.5 (Hereditary retinal dystrophy), H35.53 (Other dystrophies primarily involving the sensory retina), H35.52 (Pigmentary retinal dystrophy), H91.93 (Unspecified hearing loss, bilateral), Q87.89 (Other specified congenital malformation syndromes, not elsewhere classified). The proposed new codes are important at a systems level as data about patients with USH cannot currently be reliably extracted from general medical databases or disease-specific registries such as the American Academy of Ophthalmology's IRIS Registry.

Usher 1F Collaborative, the Usher Syndrome Coalition, and the Usher Syndrome Society, which are all nonprofit patient advocacy organizations, are requesting the following new codes to enable better tracking of these cases and treatment outcomes.

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TABULAR MODIFICATIONS

F88 Other disorders of psychological development
Delete ~~Developmental agnosia~~
Delete ~~Global developmental delay~~
Delete ~~Other specified neurodevelopmental disorder~~

New Subcategory F88.1 Usher syndrome

Add Code also, if applicable, any associated retinal dystrophy such as:
Add other dystrophies primarily involving the sensory retina (H35.53)
Add pigmentary retinal dystrophy (H35.52)

New code F88.10 Usher syndrome, unspecified
New code F88.11 Usher syndrome, type 1
New code F88.12 Usher syndrome, type 2
New code F88.13 Usher syndrome, type 3
New code F88.19 Other Usher syndrome

New code F88.8 Other disorders of psychological development
Add Developmental agnosia
Add Global developmental delay
Add Other specified neurodevelopmental disorder

New code F88.9 Disorders of psychological development, unspecified

Xylazine-associated wounds

On April 12, 2023, the White House Office of National Drug Control Policy (ONDCP) declared fentanyl adulterated or associated with xylazine an emerging drug threat. In their response plan, the ONDCP urged the creation of ICD-10-CM codes specific to xylazine and its health consequences.[1] This proposal addresses the need for standardized diagnostic coding for one distinct and ulcerating consequence: that of xylazine-associated wounds.

Xylazine is an α-2 receptor agonist approved by the Food and Drug Administration (FDA) for sedation and analgesia in veterinary medicine.[2] Xylazine is not approved for use in humans due to unsafe hypotension and central nervous system depression. Xylazine first emerged as an illicit drug in the early 2000s in Puerto Rico and 2006 in Philadelphia, PA.[3] In 2009, Rodríguez et al. reported a higher prevalence of skin wounds among people who injected xylazine-containing drugs compared to people who injected drugs without xylazine.[4] As xylazine has become pervasive in Philadelphia's illicit opioid supply, wounds and skin and soft tissue infections have come to dominate the needs of people who use drugs. In 2021, Xylazine was present in 90% of illicit fentanyl samples tested by the Philadelphia Department of Public Health.[5] Between 2020 and 2021, there was a 39% increase in hospitalizations for skin and soft tissue infections related to injection drug use in Philadelphia.[6] Wound care providers across community, outpatient, and hospital settings in Philadelphia have developed an expertise in identifying, evaluating, and treating xylazine-associated wounds. However, the lack of standardized ICD-10-CM codes has hindered continuity of care, as well as the evaluation and monitoring of xylazine-wounds and associated sequelae.

Xylazine-associated wounds are distinctly recognizable, yet their etiology is poorly understood.[7] Xylazine-associated wounds are consistently described in peer-reviewed literature as partial to full thickness skin defects with progressive necrosis of the skin, muscle, tendon, and bone.[8,9,10,11,12,13,14,15,16] Although xylazine-associated wounds commonly develop at sites of injection, they can appear anywhere on the body irrespective of the route of xylazine administration.[7,12,17,18] Xylazine-associated wounds may initially appear as areas of blistered skin, often over reddish-purple discolored tissue which evolve into a thick layer of eschar overlying a partial or full thickness ulcer that progressively increases in size and depth.[11,19] Indeed, xylazine-associated wounds are often typified by the presence of necrotic tissue (eschar, and slough) and wound diameters greater than 10 cm.[19] Xylazine-associated wounds increase a person's risk of bacteremia, endocarditis, sepsis, limb amputation, and death.[12,18] Treatment of xylazine-associated wounds is often complicated by patients' experiences of opioid and xylazine withdrawal, co-occurring mental illness, local and systemic infections, physical disability, and health-related social needs such as housing instability and food insecurity.[9,10,14]

Catherine Tomson, Daniel Teixeira da Silva, MD, MSHP, and Rachel Neuschatz, RN, MSN of the Substance Use Prevention and Harm Reduction Division in the Philadelphia Department of Public Health are requesting the following tabular modifications for reporting, evaluation, and monitoring of xylazine-associated wounds.

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TABULAR MODIFICATIONS

T65 Toxic effect of other and unspecified substances

T65.8 Toxic effect of other specified substances

New sub-subcategory T65.84 Toxic effect of xylazine

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

Add cutaneous abscess, furuncle and carbuncle (L02.-)

Add cellulitis and acute lymphangitis (L03.-)

Add non-pressure chronic ulcer of lower limb, not elsewhere classified (L97.-)

New code T65.841 Toxic effect of xylazine, accidental (unintentional)

Add Toxic effect of xylazine NOS

New code T65.842 Toxic effect of xylazine, intentional self-harm

New code T65.843 Toxic effect of xylazine, assault

New code T65.844 Toxic effect of xylazine, undetermined

L97 Non-pressure chronic ulcer of lower limb, not elsewhere classified

L97.1 Non-pressure chronic ulcer of thigh

Add Non-pressure chronic ulcer of groin

L97.2 Non-pressure chronic ulcer of calf

Add		Non-pressure chronic ulcer of shin
	L98	Other disorders of skin and subcutaneous tissue, not elsewhere classified
	L98.4	Non-pressure chronic ulcer of skin, not elsewhere classified
New sub-subcategory	L98.43	Non-pressure chronic ulcer of abdomen
New code	L98.431	Non-pressure chronic ulcer of abdomen limited to breakdown of skin
New code	L98.432	Non-pressure chronic ulcer of abdomen with fat layer exposed
New code	L98.433	Non-pressure chronic ulcer of abdomen with necrosis of muscle
New code	L98.434	Non-pressure chronic ulcer of abdomen with necrosis of bone
New code	L98.435	Non-pressure chronic ulcer of abdomen with muscle involvement without evidence of necrosis
New code	L98.436	Non-pressure chronic ulcer of abdomen with bone involvement without evidence of necrosis
New code	L98.438	Non-pressure chronic ulcer of abdomen with other specified severity
New code	L98.439	Non-pressure chronic ulcer of abdomen with unspecified severity
New sub-subcategory	L98.44	Non-pressure chronic ulcer of chest
New code	L98.441	Non-pressure chronic ulcer of chest limited to breakdown of skin
New code	L98.442	Non-pressure chronic ulcer of chest with fat layer exposed

New code	L98.443	Non-pressure chronic ulcer of chest with necrosis of muscle
New code	L98.444	Non-pressure chronic ulcer of chest with necrosis of bone
New code	L98.445	Non-pressure chronic ulcer of chest with muscle involvement without evidence of necrosis
New code	L98.446	Non-pressure chronic ulcer of chest with bone involvement without evidence of necrosis
New code	L98.448	Non-pressure chronic ulcer of chest with other specified severity
New code	L98.449	Non-pressure chronic ulcer of chest with unspecified severity
New sub-subcategory	L98.45	Non-pressure chronic ulcer of neck
New code	L98.451	Non-pressure chronic ulcer of neck limited to breakdown of skin
New code	L98.452	Non-pressure chronic ulcer of neck with fat layer exposed
New code	L98.453	Non-pressure chronic ulcer of neck with necrosis of muscle
New code	L98.454	Non-pressure chronic ulcer of neck with necrosis of bone
New code	L98.455	Non-pressure chronic ulcer of neck with muscle involvement without evidence of necrosis
New code	L98.456	Non-pressure chronic ulcer of neck with bone involvement without evidence of necrosis
New code	L98.458	Non-pressure chronic ulcer of neck with other specified severity
New code	L98.459	Non-pressure chronic ulcer of neck with unspecified severity

New sub-subcategory	L98.46	Non-pressure chronic ulcer of face
New code	L98.461	Non-pressure chronic ulcer of face limited to breakdown of skin
New code	L98.462	Non-pressure chronic ulcer of face with fat layer exposed
New code	L98.463	Non-pressure chronic ulcer of face with necrosis of muscle
New code	L98.464	Non-pressure chronic ulcer of face with necrosis of bone
New code	L98.465	Non-pressure chronic ulcer of face with muscle involvement without evidence of necrosis
New code	L98.466	Non-pressure chronic ulcer of face with bone involvement without evidence of necrosis
New code	L98.468	Non-pressure chronic ulcer of face with other specified severity
New code	L98.469	Non-pressure chronic ulcer of face with unspecified severity

New code	L98.A	Non-pressure chronic ulcer of upper limb, not elsewhere classified
Add	Includes:	chronic ulcer of upper limb NOS
Add		non-healing ulcer of skin
Add		ulcer of skin NOS
Add	Exclude2:	gangrene (I96)
Add		pressure ulcer (pressure area) (L89.-)
Add		skin infections (L00-L08)
Add		specific infections classified to A00-B99
Add		ulcer of lower limb NEC (L97.-)
Add		varicose ulcer (I83.0-I83.93)

New sub-subcategory	L98.A1	Non-pressure chronic ulcer of upper arm
Add		Non-pressure chronic ulcer of axilla
New sub-subcategory	L98.A11	Non-pressure chronic ulcer of right upper arm
New code	L98.A111	Non-pressure chronic ulcer of right upper arm limited to breakdown of skin
New code	L98.A112	Non-pressure chronic ulcer of right upper arm with fat layer exposed
New code	L98.A113	Non-pressure chronic ulcer of right upper arm with necrosis of muscle
New code	L98.A114	Non-pressure chronic ulcer of right upper arm with necrosis of bone
New code	L98.A115	Non-pressure chronic ulcer of right upper arm with muscle involvement without evidence of necrosis
New code	L98.A116	Non-pressure chronic ulcer of right upper arm with bone involvement without evidence of necrosis
New code	L98.A118	Non-pressure chronic ulcer of right upper arm with other specified severity

New code	L98.A119	Non-pressure chronic ulcer of right upper arm with unspecified severity
New sub-subcategory	L98.A12	Non-pressure chronic ulcer of left upper arm
New code	L98.A121	Non-pressure chronic ulcer of left upper arm limited to breakdown of skin
New code	L98.A122	Non-pressure chronic ulcer of left upper arm with fat layer exposed
New code	L98.A123	Non-pressure chronic ulcer of left upper arm with necrosis of muscle
New code	L98.A124	Non-pressure chronic ulcer of left upper arm with necrosis of bone
New code	L98.A125	Non-pressure chronic ulcer of left upper arm with muscle involvement without evidence of necrosis
New code	L98.A126	Non-pressure chronic ulcer of left upper arm with bone involvement without evidence of necrosis
New code	L98.A128	Non-pressure chronic ulcer of left upper arm with other specified severity

New code	L98.A129	Non-pressure chronic ulcer of left upper arm with unspecified severity
New sub-subcategory	L98.A19	Non-pressure chronic ulcer of unspecified upper arm
New code	L98.A191	Non-pressure chronic ulcer of unspecified upper arm limited to breakdown of skin
New code	L98.A192	Non-pressure chronic ulcer of unspecified upper arm with fat layer exposed
New code	L98.A193	Non-pressure chronic ulcer of unspecified upper arm with necrosis of muscle
New code	L98.A194	Non-pressure chronic ulcer of unspecified upper arm with necrosis of bone
New code	L98.A195	Non-pressure chronic ulcer of unspecified upper arm with muscle involvement without evidence of necrosis
New code	L98.A196	Non-pressure chronic ulcer of unspecified upper arm with bone involvement without evidence of necrosis
New code	L98.A198	Non-pressure chronic ulcer of unspecified upper arm with other specified severity

New code	L98.A199	Non-pressure chronic ulcer of unspecified upper arm with unspecified severity
New sub-subcategory	L98.A2	Non-pressure chronic ulcer of forearm
New sub-subcategory	L98.A21	Non-pressure chronic ulcer of right forearm
New code	L98.A211	Non-pressure chronic ulcer of right forearm limited to breakdown of skin
New code	L98.A212	Non-pressure chronic ulcer of right forearm with fat layer exposed
New code	L98.A213	Non-pressure chronic ulcer of right forearm with necrosis of muscle
New code	L98.A214	Non-pressure chronic ulcer of right forearm with necrosis of bone
New code	L98.A215	Non-pressure chronic ulcer of right forearm with muscle involvement without evidence of necrosis
New code	L98.A216	Non-pressure chronic ulcer of right forearm with bone involvement without evidence of necrosis
New code	L98.A218	Non-pressure chronic ulcer of right forearm with other specified severity

New code	L98.A219	Non-pressure chronic ulcer of right forearm with unspecified severity
New sub-subcategory	L98.A22	Non-pressure chronic ulcer of left forearm
New code	L98.A221	Non-pressure chronic ulcer of left forearm limited to breakdown of skin
New code	L98.A222	Non-pressure chronic ulcer of left forearm with fat layer exposed
New code	L98.A223	Non-pressure chronic ulcer of left forearm with necrosis of muscle
New code	L98.A224	Non-pressure chronic ulcer of left forearm with necrosis of bone
New code	L98.A225	Non-pressure chronic ulcer of left forearm with muscle involvement without evidence of necrosis
New code	L98.A226	Non-pressure chronic ulcer of left forearm with bone involvement without evidence of necrosis
New code	L98.A228	Non-pressure chronic ulcer of left forearm with other specified severity

New code	L98.A229	Non-pressure chronic ulcer of left forearm with unspecified severity
New sub-subcategory	L98.A29	Non-pressure chronic ulcer of unspecified forearm
New code	L98.A291	Non-pressure chronic ulcer of unspecified forearm limited to breakdown of skin
New code	L98.A292	Non-pressure chronic ulcer of unspecified forearm with fat layer exposed
New code	L98.A293	Non-pressure chronic ulcer of unspecified forearm with necrosis of muscle
New code	L98.A294	Non-pressure chronic ulcer of unspecified forearm with necrosis of bone
New code	L98.A295	Non-pressure chronic ulcer of unspecified forearm with muscle involvement without evidence of necrosis
New code	L98.A296	Non-pressure chronic ulcer of unspecified forearm with bone involvement without evidence of necrosis
New code	L98.A298	Non-pressure chronic ulcer of unspecified forearm with other specified severity

New code	L98.A299	Non-pressure chronic ulcer of unspecified forearm with unspecified severity
New sub-subcategory	L98.A3	Non-pressure chronic ulcer of hand
New sub-subcategory	L98.A31	Non-pressure chronic ulcer of right hand
New code	L98.A311	Non-pressure chronic ulcer of right hand limited to breakdown of skin
New code	L98.A312	Non-pressure chronic ulcer of right hand with fat layer exposed
New code	L98.A313	Non-pressure chronic ulcer of right hand with necrosis of muscle
New code	L98.A314	Non-pressure chronic ulcer of right hand with necrosis of bone
New code	L98.A315	Non-pressure chronic ulcer of right hand with muscle involvement without evidence of necrosis
New code	L98.A316	Non-pressure chronic ulcer of right hand with bone involvement without evidence of necrosis
New code	L98.A318	Non-pressure chronic ulcer of right hand with other specified severity

New code	L98.A319	Non-pressure chronic ulcer of right hand with unspecified severity
New sub-subcategory	L98.A32	Non-pressure chronic ulcer of left hand
New code	L98.A321	Non-pressure chronic ulcer of left hand limited to breakdown of skin
New code	L98.A322	Non-pressure chronic ulcer of left hand with fat layer exposed
New code	L98.A323	Non-pressure chronic ulcer of left hand with necrosis of muscle
New code	L98.A324	Non-pressure chronic ulcer of left hand with necrosis of bone
New code	L98.A325	Non-pressure chronic ulcer of left hand with muscle involvement without evidence of necrosis
New code	L98.A326	Non-pressure chronic ulcer of left hand with bone involvement without evidence of necrosis
New code	L98.A328	Non-pressure chronic ulcer of left hand with other specified severity

New code	L98.A329	Non-pressure chronic ulcer of left hand with unspecified severity
New sub-subcategory	L98.A39	Non-pressure chronic ulcer of unspecified hand
New code	L98.A391	Non-pressure chronic ulcer of unspecified hand limited to breakdown of skin
New code	L98.A392	Non-pressure chronic ulcer of unspecified hand with fat layer exposed
New code	L98.A393	Non-pressure chronic ulcer of unspecified hand with necrosis of muscle
New code	L98.A394	Non-pressure chronic ulcer of unspecified hand with necrosis of bone
New code	L98.A395	Non-pressure chronic ulcer of unspecified hand with muscle involvement without evidence of necrosis
New code	L98.A396	Non-pressure chronic ulcer of unspecified hand with bone involvement without evidence of necrosis
New code	L98.A398	Non-pressure chronic ulcer of unspecified hand with other specified severity

New code	L98.A399	Non-pressure chronic ulcer of unspecified hand with unspecified severity

ICD-10 Coordination and Maintenance Committee Meeting
March 20, 2024

TABULAR MODIFICATIONS PROPOSED ADDENDA
All approved modifications will be effective October 1, 2025

B96 Other bacterial agents as the cause of diseases classified elsewhere
B96.2 Escherichia coli [E. coli] as the cause of diseases classified elsewhere
B96.21 Shiga toxin-producing Escherichia coli [E. coli] [STEC] O157 as the cause of diseases classified elsewhere
Revise O157:H7 Escherichia coli ~~[E.coli]~~ [E. coli] with or without confirmation of Shiga toxin-production
Revise Shiga toxin-producing Escherichia coli ~~[E.coli]~~ [E. coli]

B96.22 Other specified Shiga toxin-producing Escherichia coli [E. coli] [STEC] as the cause of diseases classified elsewhere
Revise Non-O157 Shiga toxin-producing Escherichia coli ~~[E.coli]~~ [E. coli]
Revise Non-O157 Shiga toxin-producing Escherichia coli ~~[E.coli]~~ [E. coli] with known O group

C7B Secondary neuroendocrine tumors

C7B.0 Secondary carcinoid tumors
C7B.04 Secondary carcinoid tumors of peritoneum
Revise ~~Mesentary~~ Mesentery metastasis of carcinoid tumor

C77 Secondary and unspecified malignant neoplasm of lymph nodes
Excludes1: malignant neoplasm of lymph nodes, specified as primary (C81-C86, C88, C96.-)
Revise ~~mesentary~~ mesentery metastasis of carcinoid tumor (C7B.04)

D12 Benign neoplasm of colon, rectum, anus and anal canal

D12.6 Benign neoplasm of colon, unspecified Oct 1, 2024
Adenomatosis of colon
Benign neoplasm of large intestine NOS
Polyposis (hereditary) of colon
Delete ~~Excludes1: inflammatory polyp of colon (K51.4-)~~
Add Excludes2: inflammatory polyp of colon (K51.4-)

D48 Neoplasm of uncertain behavior of other and unspecified sites
D48.1 Neoplasm of uncertain behavior of connective and other soft tissue
D48.11 Desmoid tumor
Add Aggressive fibromatosis

D68 Other coagulation defects
D68.3 Hemorrhagic disorder due to circulating anticoagulants
D68.31 Hemorrhagic disorder due to intrinsic circulating anticoagulants, antibodies, or inhibitors
D68.312 Antiphospholipid antibody with hemorrhagic disorder
Lupus anticoagulant (LAC) with hemorrhagic disorder
Systemic lupus erythematosus [SLE] inhibitor with hemorrhagic disorder

Delete Excludes1: ~~antiphospholipid antibody syndrome (D68.61)~~
Delete ~~antiphospholipid antibody with hypercoagulable state (D68.61)~~
Delete ~~lupus anticoagulant (LAC) with hypercoagulable state (D68.62)~~
Delete ~~systemic lupus erythematosus [SLE] inhibitor with hypercoagulable state (D68.62)~~

Add Excludes2: antiphospholipid antibody syndrome (D68.61)
Add antiphospholipid antibody with hypercoagulable state (D68.61)
Add lupus anticoagulant (LAC) with hypercoagulable state (D68.62)
Add systemic lupus erythematosus [SLE] inhibitor with hypercoagulable state (D68.62)

D68.6 Other thrombophilia
D68.61 Antiphospholipid syndrome

Delete Excludes1: ~~anti-phospholipid antibody, finding without diagnosis (R76.0)~~
Delete ~~anti-phospholipid antibody with hemorrhagic disorder (D68.312)~~
Delete ~~lupus anticoagulant syndrome (D68.62)~~

Add Excludes2: anti-phospholipid antibody, finding without diagnosis (R76.0)
Add anti-phospholipid antibody with hemorrhagic disorder (D68.312)
Add lupus anticoagulant syndrome (D68.62)

D68.62 Lupus anticoagulant syndrome

Delete Excludes1: ~~anticardiolipin syndrome (D68.61)~~
Delete ~~antiphospholipid syndrome (D68.61)~~
Delete ~~lupus anticoagulant (LAC) finding without diagnosis (R76.0)~~
Delete ~~lupus anticoagulant (LAC) with hemorrhagic disorder (D68.312)~~

Add Excludes2: anticardiolipin syndrome (D68.61)
Add antiphospholipid syndrome (D68.61)
Add lupus anticoagulant (LAC) finding without diagnosis (R76.0)
Add lupus anticoagulant (LAC) with hemorrhagic disorder (D68.312)

E88 Other and unspecified metabolic disorders
E88.4 Mitochondrial metabolism disorders
E88.43 Disorders of mitochondrial tRNA synthetases

Add ARS2-related mitochondrial disorders
Add LBSL
Add Leukoencephalopathy with brainstem - spinal cord involvement – lactate elevation
Add Leukoencephalopathy with thalamus – brainstem involvement - high lactate
Add LTBL
Add Mitochondrial aminoacyl-tRNA synthetase disorders

F42 Obsessive-compulsive disorder
Excludes2: obsessive-compulsive symptoms occurring in depression ~~(F32-F33)~~ (F32.-, F33.-)

F63 Impulse disorders
F63.2 Kleptomania
Pathological stealing
Revise Excludes2: depressive disorder with stealing ~~(F31-F33)~~ (F31.-, F32.-, F33.-)

G00 Bacterial meningitis, not elsewhere classified
G00.1 Pneumococcal meningitis
Revise ~~Meningtitis~~ Meningitis due to Streptococcal pneumoniae

G23 Other degenerative diseases of basal ganglia
G23.3 Hypomyelination with atrophy of the basal ganglia and cerebellum
H-ABC
Add TUBB4A-related neurologic disorders

J43 Emphysema
Delete Excludes1: ~~emphysema due to inhalation of chemicals, gases, fumes or vapors (J68.4)~~
Add Code also, if applicable, emphysema due to inhalation of chemicals, gases, fumes or vapors (J68.4)

J84 Other interstitial pulmonary diseases
J84.1 Other interstitial pulmonary diseases with fibrosis
Delete Excludes1: ~~pulmonary fibrosis (chronic) due to inhalation of chemicals, gases, fumes or vapors (J68.4)~~
Add Code also, if applicable, pulmonary fibrosis (chronic) due to inhalation of chemicals, gases, fumes or vapors (J68.4)

K75 Other inflammatory liver diseases
K75.8 Other specified inflammatory liver diseases
K75.81 Nonalcoholic steatohepatitis (NASH)
Add Metabolic dysfunction-associated steatohepatitis (MASH)

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

M36 Systemic disorders of connective tissue in diseases classified elsewhere
M36.3 Arthropathy in other blood disorders
Add Code first underlying disease

O04 Complications following (induced) termination of pregnancy
Revise Excludes2: encounter for elective termination of pregnancy~~,~~ ~~uncomplicated~~ (Z33.2)

O70 Perineal laceration during delivery
O70.1 Second degree perineal laceration during delivery
Revise Excludes1: perineal laceration involving anal sphincter (O70.2-)
O70.4 Anal sphincter tear complicating delivery, not associated with third degree laceration
Revise Excludes1: anal sphincter tear with third degree perineal laceration (O70.2-)

O90 Complications of the puerperium, not elsewhere classified
O90.4 Postpartum acute kidney failure
Delete ~~Excludes1: non-anuria and oliguria (R34)~~

O90.41 Hepatorenal syndrome following labor and delivery

O90.49 Other postpartum acute kidney failure
Add Excludes1: anuria and oliguria (R34)

P09 Abnormal findings on neonatal screening
Revise P09.6 Abnormal findings on neonatal hearing screening ~~for neonatal hearing loss~~

Q75 Other congenital malformations of skull and face bones
Q75.0 Craniosynostosis

Revise Q75.00 Craniosynostosis, unspecified
Craniosynostosis NOS
Revise Q75.001 Craniosynostosis, unspecified type, unilateral
Revise Q75.002 Craniosynostosis, unspecified type, bilateral
Revise Q75.009 Craniosynostosis, unspecified
Imperfect fusion of skull

Q75.02 Coronal craniosynostosis
Delete ~~Non-deformational anterior plagiocephaly~~
Excludes1: dolichocephaly (Q67.2)
Revise Q75.021 Coronal craniosynostosis, unilateral

Non-deformational anterior plagiocephaly
Revise Q75.022 Coronal craniosynostosis, bilateral
Revise Q75.029 Coronal craniosynostosis, unspecified

R15 Fecal incontinence
R15.0 Incomplete defecation
Delete ~~Excludes1: constipation (K59.0-)~~
Delete ~~fecal impaction (K56.41)~~
Add Excludes2: constipation (K59.0-)
Add fecal impaction (K56.41)

R39 Other and unspecified symptoms and signs involving the genitourina system
R39.0 Extravasation of urine
R39.1 Other difficulties with micturition
R39.12 Poor urinary stream
Revise Weak urinary ~~steam~~ stream

R40 Somnolence, stupor and coma
R40.1 Stupor
Catatonic stupor
Semicoma
Revise Excludes1: depressive stupor ~~(F31-F33)~~ (F31.-, F32.-, F33.-)

R53 Malaise and fatigue
R53.8 Other malaise and fatigue
Revise Excludes1: exhaustion and fatigue due to recurrent depressive episode (F33.-)

R62 Lack of expected normal physiological development in childhood and adults
R62.5 Other and unspecified lack of expected normal physiological development in childhood
R62.51 Failure to thrive (child)
Add Faltering growth

R69 Illness, unspecified
Revise Unknown and unspecified ~~cases~~ causes of morbidity

R87 Abnormal findings in specimens from female genital organs
R87.6 Abnormal cytological findings in specimens from female genital organs
R87.61 Abnormal cytological findings in specimens from cervix uteri
R87.619 Unspecified abnormal cytological findings in specimens from cervix uteri
Revise Atypical ~~endocervial~~ endocervical cells of cervix NOS

S06 Intracranial injury
S06.3 Focal traumatic brain injury
S06.34 Traumatic hemorrhage of right cerebrum
Traumatic intracerebral hemorrhage and hematoma of right cerebrum
S06.341 Traumatic hemorrhage of right cerebrum with loss of consciousness of 30 minutes or less
Revise Traumatic hemorrhage of right cerebrum with brief loss of consciousness

S12 Fracture of cervical vertebra and other parts of neck
S12.8 Fracture of other parts of neck
Revise Fracture of ~~H~~hyoid bone
Revise Fracture of ~~L~~larynx
Revise Fracture of ~~T~~thyroid cartilage
Revise Fracture of ~~T~~trachea

S37 Injury of urinary and pelvic organs
S37.6 Injury of uterus
Delete Excludes1: ~~injury to gravid uterus (O9A.2-)~~

T65 Toxic effect of other and unspecified substances
T65.3 Toxic effect of nitroderivatives and aminoderivatives of benzene and its homologues
Revise Toxic effect of ~~anilin~~ aniline[benzenamine]

V06 Pedestrian injured in collision with other nonmotor vehicle
V06.9 Pedestrian injured in collision with other nonmotor vehicle, unspecified whether traffic or nontraffic accident
V06.93 Pedestrian on standing micro-mobility pedestrian conveyance injured in collision with other nonmotor vehicle, unspecified whether traffic or nontraffic accident

V06.938 Pedestrian on other standing micro-mobility pedestrian conveyance injured in collision with other nonmotor vehicle, unspecified whether traffic or nontraffic accident

Revise: Pedestrian on hoverboard injured in collision with other nonmotor vehicle, unspecified whether traffic or nontraffic accident

W44 Foreign body entering into or through a natural orifice
W44.A Battery entering into or through a natural orifice
W44.A9 Other batteries entering into or through a natural orifice

Revise: Cylindrical battery entering into or through a natural orifice

Y07 Perpetrator of assault, maltreatment and neglect
Y07.4 Other family member, perpetrator of maltreatment and neglect
Y07.43 Stepparent or stepsibling, perpetrator of maltreatment and neglect

Revise: Y07.435 Stepbrother, perpetrator ~~or~~ of maltreatment and neglect

Persons encountering health services for examinations (Z00-Z13)

Delete: ~~Excludes1: examinations related to pregnancy and reproduction (Z30-Z36, Z39.-)~~
Add: Excludes2: examinations related to pregnancy and reproduction (Z30-Z36, Z39.-)

Z01 Encounter for other special examination without complaint, suspected or reported diagnosis

Delete: Excludes1: ~~encounter for laboratory and radiologic examinations as a component of general medical examinations (Z00.0-)~~

Add: Excludes2: encounter for laboratory and radiologic examinations as a component of general medical examinations (Z00.0-)

Z83.7 Family history of diseases of the digestive system
Z83.71 Family history of colonic polyps

Revise: Z83.718 ~~Other~~ fFamily history of colon polyps

ICD-10 Coordination and Maintenance Committee Meeting
March 20, 2024

INDEX MODIFICATION PROPOSED ADDENDA
All approved modifications will be effective October 1, 2025

Abnormal, abnormality, abnormalities - see also Anomaly
- cytology
- - anus R85.619
- - - human papillomavirus (HPV) DNA test
Revise - - - - low risk ~~postive~~ positive R85.82
- Papanicolaou (smear)
- - anus R85.619
- - - human papillomavirus (HPV) DNA test
Revise - - - - low risk ~~postive~~ positiveR85.82
- - cervix R87.619
Revise - - - thin preparaton preparation R87.619

Admission (for) - see also Encounter (for)
- adjustment (of)
- - artificial
Revise - - - eye ~~Z44.2~~ Z44.2-
- - device NEC
- - - prosthetic Z44.9
Revise - - - - eye ~~Z44.2~~ Z44.2-
- - prosthesis Z44.9
Revise - - - eye ~~Z44.2~~ Z44.2-
- fitting (of)
- - artificial
Revise - - - eye ~~Z44.2~~ Z44.2-

Algoneurodystrophy M89.00
Revise - multiple sites ~~M89.0~~ M89.09

Anemia (essential) (general) (hemoglobin deficiency) (infantile) (primary) (profound) D64.9
- due to (in) (with)
Revise - - fish tapeworm (~~D.latum~~ D. latum) infestation B70.0 [D63.8]

Angulation
Revise - coccyx (acquired) ~~- see also subcategory~~ ~~M43.8~~ M43.8X8

Aversion
- oral R63.39
Revise - - newborn ~~P92.~~ P92.8

Arthritis, arthritic (acute) (chronic) (nonpyogenic) (subacute) M19.90
- in (due to)
- - crystals M11.9
- - - specified type NEC M11.80

Revise - - - - multiple sites ~~M11.8~~ M11.89

Arthropathy (see also Arthritis) M12.9
- in (due to)

Revise - - ulcerative colitis K51.90 [~~M07.60~~ M07.6-]

Breathing
- mouth R06.5

Revise - - causing malocclusion ~~M26.5~~ M26.59

Bronchiolitis (acute) (infective) (subacute) J21.9

Delete - chronic (fibrosing) ~~(obliterative)~~ J44.89

Add - - obliterative J44.81

Conflict (with) - see also Discord

Revise - family ~~Z73.9~~ Z63.8

Cubitus

Revise - valgus (acquired) ~~M21.0~~ M21.02-

Revise - varus (acquired) ~~M21.1~~ M21.12-

Chancre (any genital site) (hard) (hunterian) (mixed) (primary) (seronegative) (seropositive) (syphilitic) A51.0

Revise - ~~Ducrey's~~ Ducreyi's A57

Chloasma (skin) (idiopathic) (symptomatic) L81.1

Revise - eyelid ~~H02.719~~ H02.71-

Revise - - hyperthyroid E05.90 [~~H02.719~~ H02.71-]

Revise - - - with thyroid storm E05.91 [~~H02.719~~ H02.71-]

Chondritis M94.8x9

Revise - ~~aurical~~ auricle H61.03-

Complication(s) (from) (of)
- circulatory system I99.8
- - postprocedural I97.89

Revise - - - following cardiac surgery (see also Infarct, myocardium, associated with revascularization procedure) ~~I97.19~~ I97.190
- transfusion (blood) (lymphocytes) (plasma) T80.92

Revise - - air ~~emblism~~ embolism

Density
- breast R92.30
Revise - - low R92.30

Depression (acute) (mental) F32.A
Revise - central nervous system ~~R09.2~~ G96.9

Derangement
- knee (recurrent) M23.9-
- - meniscus M23.30-
Revise - - - cystic ~~M23.00~~ M23.0-

Disease
- sickle-cell D57.1
- - with
Revise - - - ~~priaprism~~ priapism D57.09

- - Hb-C D57.20
- - - with
Revise - - - ~~priaprism~~ priapism D57.218

Dislocation (articular)
- vertebral (articular process) (body) (traumatic)
- - traumatic
- - - cervical S13.100
- - - - joint between
Revise - - - - C5 and C6 S13.161
Revise - - - - C6 and C7 S13.171
Revise - - - - C7-and T1 S13.181
- - lumbar S33.101
- - - joint between
Revise - - - - L1 and L2 S33.111
Revise - - - - L2 and L3 S33.121
Revise - - - - L4 and L5 S33.141

Disturbance(s) - see also Disease
- heart, functional (conditions in I44-I50)
- - due to presence of (cardiac) prosthesis I97.19-
- - postoperative I97.89
Revise - - - cardiac surgery (see also Infarct, myocardium, associated with revascularization procedure) ~~I97.19~~ I97.190
Add - - - other surgery I97.191

Dyspnea (nocturnal) (paroxysmal) R06.00
- asthmatic (bronchial) J45.909
Delete - - ~~cardiac - see Failure, ventricular, left~~

Encephalopathy (acute) G93.40
Revise - ~~spongioform~~, spongiform (subacute) (viral) A81.09

Enlargement, enlarged - see also Hypertrophy
- prostate N40.0
Revise - - without lower urinary tract ~~symtpoms~~ symptons (LUTS) N40.0

Endophthalmitis (acute) (infective) (metastatic) (subacute) H44.009
Revise - bleb associated H59.4-see also Bleb, ~~inflammed~~ inflamed (infected), postprocedural

Enthesopathy (peripheral) M77.9
Delete ~~- shoulder M77.8~~
- shoulder region -see Lesion, shoulder

Epilepsy, epileptic, epilepsia (attack) (cerebral) (convulsion) (fit) (seizure) G40.909
Revise - Lafora progressive myoclonus (see also Epilepsy, myoclonus, progressive, Lafora) G40.C09
- progressive (familial) myoclonic - see Epilepsy, myoclonus, progressive
Revise - - Lafora (see also Epilepsy, myoclonus, progressive, Lafora) G40.C09
- related to
- - alcohol G40.509
- - - not intractable G40.509
Revise - - - - without status ~~eplepticus~~ epilepticus G40.509
- - drugs G40.509
- - - not intractable G40.509
Revise - - - - without status ~~eplepticus~~ epilepticus G40.509
- - external causes G40.509
- - - not intractable G40.509
Revise - - - - without status ~~eplepticus~~ epilepticus G40.509
- - hormonal changes G40.509
- - - not intractable G40.509
Revise - - - - without status ~~eplepticus~~ epilepticus G40.509
- - sleep deprivation G40.509
- - - not intractable G40.509
Revise - - - - without status ~~eplepticus~~ epilepticus G40.509
- - stress G40.509
- - - not intractable G40.509
Revise - - - - without status ~~eplepticus~~ epilepticus G40.509

Erysipelas (gangrenous) (infantile) (newborn) (phlegmonous) (suppurative) A46
Revise - external ear A46 [~~H62.40~~ H62.4-]

Add EVALI (e-cigarette, or vaping, product use associated lung injury) U07.0

Failure, failed
- heart (acute) (senile) (sudden) I50.9
Revise - - following cardiac surgery ~~I97.13-~~ I97.130
Add - - following other surgery I97.131
Revise - - stage B -see also Failure, heart, by type as diastolic or systolic, if known I50.9
Revise - - stage C -see also Failure, heart, by type as diastolic or systolic, if known I50.9
Revise - intestinal ~~failure~~ K90.83

Fitting (and adjustment) (of)
- artificial
Revise - - eye Z44.2-
- device NOS Z46.9
- - prosthetic (external) Z44.9
Revise - - - eye Z44.2-
- prosthesis (external) Z44.9
Revise - - eye Z44.2-

Fracture, pathological (pathologic) (see also Fracture, traumatic M84.40)
- due to
- - specified disease NEC M84.60
Revise - - - hip ~~M84.65-~~ M84.659

Fracture, traumatic (abduction) (adduction) (separation) (see also Fracture, pathological) T14.8
Revise - basicervical (basal) (femoral) ~~S72.0~~ S72.04-
- femur, femoral S72.9-
Revise - - basicervical (basal) ~~S72.0 S72.04-~~

Frostbite (superficial) T33.90
Revise - leg ~~T33.9-~~ T33.99
Revise - - with tissue necrosis ~~T34.9-~~ T34.99

Fungemia NOS B49
Add - candida B37.7

History
- personal (of) - see also History, family (of)
Revise - - cardiac arrest (death), ~~successfully~~ successfully resuscitated Z86.74
Revise - - osteoporosis fractures ~~Z87.31~~ Z87.310

Hyperplasia, hyperplastic
- prostate (adenofibromatous) N40.0

Revise - - without lower urinary tract ~~symtpoms~~ symptoms (LUTS) N40.0

Impetigo (any organism) (any site) (circinate) (contagiosa) (simplex) (vulgaris) L01.00

Revise - external ear L01.00 [~~H62.40~~ H62.4-]

Infarct, infarction
- myocardium, myocardial (acute) (with stated duration of 4 weeks or less) I21.9
- - postprocedural - see also Infarct, myocardium, associated with revascularization procedure

Revise - - - following cardiac surgery ~~surgery~~ (see also Infarct, myocardium, type 4 or type 5) I97.190

Infection
- Bacillus A49.9

Revise - - ~~Ducrey's~~ Ducreyi's (any location) A57

Inflammation, inflamed, inflammatory (with exudation)

Revise - dura ~~mater~~ matter - see Meningitis

Revise - pia ~~mater~~ matter - see Meningitis

Injury -see also specified injury type T14.90
- lung - see also Injury, intrathoracic, lung

Revise - - EVALI ~~-~~ [e-cigarette, or vaping, product use associated] U07.0
(remove dash after EVALI)

Laceration

Revise - peritoneum ~~S36.893~~ S36.81

Revise Maculae ceruleae ~~--~~ B85.1
delete hyphens

Meningoencephalitis (see also Encephalitis) G04.90
- in (due to)

Revise - - Hemophilus influenzae (~~H .influenzae~~ H. influenzae) G00.0

Nephropathy (see also Nephritis) N28.9
- IgA N02.B-
Revise - - with ~~glomerular lesion N02.B1~~
Add - - - glomerular lesion N02.B1
Add - - - - focal and segmental N02.B2
Add - - - glomerulonephritis
Revise ~~-~~ - - - membranoproliferative (diffuse) N02.B3
Revise ~~-~~ - - - membranous (diffuse) N02.B4
Revise ~~-~~ - - - mesangial proliferative (diffuse) N02.B5
Revise ~~-~~ - - - mesangiocapillary (diffuse) N02.B6
Delete ~~- - - proliferative NEC N02.B9~~
Add - - proliferative NEC N02.B9
Delete ~~- - - specified pathology NEC N02.B9~~
Add - - specified pathology NEC N02.B9

Neurosyphilis (arrested) (early) (gumma) (late) (latent) (recurrent) (relapse) A52.3
Revise - dura (~~mater~~ matter) A52.13

Nodule(s), nodular
- prostate N40.2
Revise - - without lower urinary tract ~~symtpoms~~ symptoms (LUTS) N40.2

Osteonecrosis M87.9
- secondary NEC M87.30
- - due to
- - - drugs M87.10
Revise - - - - neck ~~M87.18~~ M87.188
Revise - - - - rib ~~M87.18~~ M87.188
Revise - - - - skull ~~M87.18~~ M87.188
Revise - - - - vertebra ~~M87.18~~ M87.188

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

Otitis (acute) H66.90
- externa H60.9-
- - in (due to)
Revise - - - erysipelas A46 [~~H62.40~~ H62.4-]
Revise - - - impetigo L01.00 [~~H62.40~~ H62.4-]
Revise - - - mycosis NEC B36.9 [~~H62.40~~ H62.4-]

Revise - - - parasitic disease NEC B89 [~~H62.40~~ H62.4-]
Revise - - - viral disease NEC B34.9 [~~H62.40~~ H62.4-]
Revise - - mycotic NEC B36.9 [~~H62.40~~ H62.4-]
Revise - - tropical NEC B36.9 [~~H62.40~~ H62.4-]

Revise Otomycosis (diffuse) NEC B36.9 [~~H62.40~~ H62.4-]

Overgrowth
- bacterial
- - small intestinal K63.8219
Delete - - - fungal K63.822
Add - fungal
Add - - small intestinal K63.22

Pain(s) (see also Painful) R52
Revise - rib ~~R07.81~~ R07.89

Paralysis, paralytic (complete) (incomplete) G83.9
Revise - ~~subcapsularis~~ subscapularis G56.8-

Puerperal, puerperium (complicated by, complications)
- hemiplegia, cerebral O99.355
Revise - - due to ~~cerbrovascular~~ cerebrovascular disorder O99.43
Revise - ~~retrated~~ retracted nipple O92.02

Polydactylism, polydactyly Q69.9
Add - fingers Q69.0
Add - thumb Q69.1

Polyposis - see also Polyp
Revise - coli ~~(adenomatous)~~ D12.6
Add - - adenomatous D13.91
Revise - colon ~~(adenomatous)~~ D12.6
Add - - adenomatous D13.91

Pregnancy (single) (uterine) - see also Delivery and Puerperal Z33.1
- complicated by (care of) (management affected by)
- - fetal (maternal care for)
- - - musculoskeletal anomalies
Revise - - - - trunk O35.F ~~O35.F~~

	Relapsing fever A68.9
Revise	- Obermeyers's Obermeyer's (European) A68.0
Revise	Restriction ~~of housing space Z59.19~~
Add	- growth
Add	- - fetal O36.59-
Add	- of housing space Z59.19
	Restoration (of)
	- dental
Revise	- - failure of periodontal anatomical ~~intergrity~~ integrity K08.54
	Retinopathy (background) H35.00
	- proliferative NEC H35.2-
Revise	- - ~~thaslassemia~~ thalassemia H35.2
Revise	Saber, sabre shin or tibia (syphilitic) A50.56 [~~M90.8-~~ M90.86-]
	Sepsis (generalized) (unspecified organism) A41.9
	- with
Add	- - ectopic or molar pregnancy O08.82
	- due to device, implant or graft T85.79
Delete	~~- - ectopic or molar pregnancy O08.82~~
	Short, shortening, shortness
Revise	- bowel syndrome ~~K91.2~~ K90.82-
Revise	Sin Nombre virus disease (Hantavirus) (cardio)-pulmonary syndrome~~)~~ B33.4
	Snapping
	- hip - see Derangement, joint, specified type NEC, hip
Revise	- - involving the ~~iliotiblial~~ iliotibial band M76.3-
	- knee - see Derangement, knee
Revise	- - involving the ~~iliotiblial~~ iliotibial band M76.3-
	Spasm(s), spastic, spasticity (see also condition) R25.2
Revise	- nerve, trigeminal ~~G51.0~~ G50.0
	Stasis
Revise	- foot ~~T69.0~~ T69.02-

Subluxation - see also Dislocation
- vertebral
- - traumatic
- - - cervical S13.100
- - - - joint between
Revise - - - - - C5 and C6 S13.160
Revise - - - - - C6 and C7 S13.170
Revise - - - - - C7 and T1 S13.180
- - - lumbar S33.100
- - - - joint between
Revise - - - - - L1 and L2 S33.110
Revise - - - - - L2 and L3 S33.120
- - - thoracic S23.100
- - - - joint between
Revise - - - - - T1 and T2 S23.110
Revise - - - - - T2 and T3 S23.120

Add Sudden infant death syndrome (SIDS) R99

Syndrome -see also Disease
Revise - ~~Gopalan'~~ Gopalan's (burning feet) E53.8
Revise - ~~Lemiere~~ Lemierre I80.8
Revise - tropical wet feet ~~T69.0~~ T69.02-
- wet
Revise - - feet (maceration) (tropical) ~~T69.0~~ T69.02-

Synovitis (see also Tenosynovitis) M65.9
- crepitant
Revise - - hand ~~M70.0~~ M70.04-

Syphilis, syphilitic (acquired) A53.9
Revise - dura ~~mater~~ matter A52.13

Thrombophlebitis I80.9
Revise - lower extremity – see also Phlebitis, leg ~~I80.299~~
Add - - deep (see also Phlebitis, leg, deep) I80.29-

Revise tPA (rtPA) ~~administation~~ administration in a different facility within the last 24 hours prior to admission to current facility Z92.82

Revise Tuberosity, ~~enitre~~ entire maxillary M26.07

Tumor - see also Neoplasm, unspecified behavior, by site
- carcinoid D3A.00
Revise - - ~~mesentary~~ mesentery metastasis C7B.04

Vomiting R11.10
Revise - fecal ~~mater~~ matter R11.13

ICD-10 Coordination and Maintenance Committee Meeting
March 20, 2024

ICD-10-CM EXTERNAL CAUSE OF INJURIES INDEX
All approved modifications will be effective October 1, 2025

Accident (to) X58
- nontraffic (victim's mode of transport NOS) V88.9
- - collision (between) V88.7
Revise - - - car and~~:~~
Revise - - - specified vehicle NEC and~~:~~

- traffic (victim's mode of transport NOS) V87.9
- - collision (between) V87.7
Revise - - - car and~~:~~
- watercraft V94.9
- - nonpowered, struck by
Revise - - - nonpowered vessel ~~V94.22~~ V94.21
Revise - - - powered vessel ~~V94.21~~ V94.22

Activity (involving) (of victim at time of event) Y93.9
Add - badminton (Y93.73)
Add - pickleball (Y93.73)

Asphyxia, asphyxiation
- by
- - food (bone) (seed) - see categories T17 and T18
Revise - - vomitus ~~T17.81~~ T17.810

Aspiration
- food (any type) (into respiratory tract) (with asphyxia, obstruction respiratory tract, suffocation) - see categories T17 and T18
Revise - vomitus (with asphyxia, obstruction respiratory tract, suffocation) ~~T17.81~~ T17.810

Assault (homicidal) (by) (in) Y09
- bodily force Y04.8
Revise - - sexual - see subcategories T74.0, T76.0

Revise Bean in nose ~~- see categories T17 and T18~~ T17.1

Discharge (accidental)
- firearm (accidental) W34.00
- - legal intervention
- - - injuring
Revise - - - - suspect ~~Y35.03~~ Y35.003

Drowning (accidental) W74
Revise - resulting from accident to ~~watercraftCsee~~ watercraft - see Drowning, due to, accident, watercraft

Explosion (accidental) (of) (with secondary fire) W40.9
- firearm (parts) NEC W34.19
Revise - - ~~hangun~~ handgun W32.1
Revise - handgun (parts) – see Explosion, firearm, ~~hangun~~ handgun (parts)

Exposure (to) X58
- electric current W86.8
- - taser W86.8
Revise - - - ~~undeterminedundetermined~~ intent Y33

- fire, flames (accidental) X08.8
- - controlled (in)
- - - building or structure X02.0
- - - - with
Revise - - - - - jump from building X02.5

Revise Hanging (accidental) - see also ~~category~~ ~~T71~~ T71.16-

Place of occurrence Y92.9
- school (private) (public) (state) Y92.219
Revise - - ~~trace~~ trade school Y92.215

ICD-10-CM TABLE of DRUGS and CHEMICALS
All approved modifications will be effective October 1, 2025

Substance	Poisoning Accidental (unintentional)	Poisoning Intentional self-harm	Poisoning Assault	Poisoning Undetermined	Adverse effect	Underdosing
Add Antihelminthic	T37.4X1	T37.4X2	T37.4X3	T37.4X4	T37.4X5	T37.4X6
Revise ~~Benzylhydrochlorthia-zide~~ Benzylhydrochlorthiazide	T50.2X1	T50.2X2	T50.2X3	T50.2X4	T50.2X5	T50.2X6
Add Bithionol – antihelminthic	T37.4X1	T37.4X2	T37.4X3	T37.4X4	T37.4X5	T37.4X6
Revise ~~Bromochlorosalicylani-lide~~ Bromochlorosalicylanilide	T49.0X1	T49.0X2	T49.0X3	T49.0X4	T49.0X5	T49.0X6
Calcium Revise ~~gluconogalactogluc-onate~~ gluconogalactogluconate	T50.3X1	T50.3X2	T50.3X3	T50.3X4	T50.3X5	T50.3X6
Revise Cyamopsis ~~tetragono-loba~~ tetragonoloba	T46.6X1	T46.6X	T46.6X	T46.6X	T46.6X	T46.6X
Revise ~~Diisopropylfluorophos-phonate~~ Diisopropylfluorophosphate	T44.0X1	T44.0X2	T44.0X3	T44.0X4	T44.0X5	T44.0X6
Revise Hexaethyl tetraphos-phate tetraphosphate	T60.0X1	T60.0X2	T60.0X3	T60.0X4	--	--
Revise ~~Hydroxymethylpenta-none~~ Hydroxymethylpentanone	T52.4X1	T52.4X2	T52.4X3	T52.4X4	--	--
Revise Lente ~~lietin~~ Iletin (insulin)	T38.3X1	T38.3X2	T38.3X3	T38.3X4	T38.3X5	T38.3X6
Revise MOPP (~~mechloreth-amine~~ mechlorethamine + vincristine	T45.1X1	T45.1X2	T45.1X3	T45.1X4	T45.1X5	T45.1X6

+ prednisone + ~~procarba-zine procarbazine~~)						
Revise Neosporin- ~~opthalmic~~ ophthalmic preparation	T49.5X1	T49.5X2	T49.5X3	T49.5X4	T49.5X5	T49.5X6
Revise NPH ~~lletin~~ lletin (insulin)	T38.3X1	T38.3X2	T38.3X3	T38.3X4	T38.3X5	T38.3X6
Revise Polish (car) (floor) ~~(furni-ture~~ furniture) (metal) (porcelain) (silver)	T65.891	T65.892	T65.893	T65.894	--	--

ICD-10-CM TABLE of NEOPLASMS
All approved modifications will be effective October 1, 2025

Neoplasm	Malignant Primary	Malignant Secondary	Ca in situ	Benign	Uncertain Behavior	Unspecified Behavior
Neoplasm, neoplastic						
abdomen, abdominal	C76.2	C79.8-	D09.8	D48.7	D49.89	
- wall (see also Neoplasm, abdomen, wall, skin)						
Revise - - connective tissue	C49.4-	C79.8-	- D21.4		~~D48.1~~ D48.11-	
Revise adipose tissue (see also Neoplasm, connective tissue)		C49.4	C79.89	D21.9	~~D48.1~~ D48.11-	D49.2
Revise aorta (thoracic)	C49.3		C79.89	-	D21.3	~~D48.1~~ D48.11-
- abdominal	C49.4	C79.89		-	D21.4	~~D48.1~~ D48.11-
Revise cerebrum, ~~cerebra~~ cerebral (cortex) (hemisphere) (white matter)						
Revise eye NEC	C69.9	C79.49	~~D09.2~~ D09.2-	~~D31.9~~ D31.9-	D48.7	D49.89
Revise - overlapping sites	~~C69.8~~ C69.8-	-	-	~~-~~	-	-
Revise femur (any part)	C40.2.-	C79.5-	-	-	D16.2-	-
ligament - see also Neoplasm, connective tissue	~~C57.1~~ C57.1-	C79.82	D07.39	D28.2	D39.8	D49.59
Revise - broad						
lung	C34.1-	C78.0-	D02.2-	D14.3-	D38.1	D49.1
Revise - ~~linqu~~la lingula						

Revise	meninges - ~~crainial~~ cranial	C70.0	C79.32	-	D32.0	D42.0	D49.7
Revise	odontogenic -see Neoplasm, jaw, bone						
Revise	patella	~~C40.20~~ C40.2-	C79.51	-	-	-	-