Family Doctor

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Focus:

Hospital-based Medicine for the Family Physician

FEATURE ARTICLES:

New York State Academy of Family Physicians

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From the Executive Vice President

By Vito Grasso, MPA, CAE

The 2024 legislative session in NY will present many challenges. The impact of a presidential election year will add an element of uncertainty as we endeavor to promote the interests of patients and family physicians.

Many of the issues we will face are familiar. The challenge of achieving a health care system that is current, accessible, sustainable and equitable is complex and the influence of vested interests makes change slow and often confrontational.

We continue to advocate for increased investment in primary care across a spectrum of issues including compensation, introduction of new therapies, collective bargaining for physicians, equitable treatment of patients, addressing social determinants, workforce adequacy and system reform.

The piecemeal approach to change in health care has not produced solutions to problems that are inherent in the market driven system we have relied upon to deliver health care. The prevalent perspective that health care is just another commodity, and that private enterprise is the best vehicle for providing the services that are essential to maintain good health and treat acute episodes has been revealed as impractical and unjust. Yet, somehow, we cannot find the political will to improve that system.

At its core, the essential question for health care policy makers is what is the best way to pay for a health care system that has sufficient capacity to deliver care to the entire population, to maintain a reserve of resources to address emergencies, that enables providers to form the capital they need to absorb and adapt to changes in the clinical and administrative environment, that sustains the production of new health care workers, and that supports new clinical applications to accommodate the continuing effectiveness of the health care delivery infrastructure. We have maintained that a single payer system is the best way to provide health care that meets all of these requirements.

At the national level much discussion has focused on value-based payment (VBP) and prospective payment to improve payment for primary care. Many models have been tested and none have produced the savings, efficiency or equity which have been goals of our own advocacy. A consistent deficiency in the various VBP and prospective payment models has been the elaborate methods of evaluating performance which are inextricably associated with VBP and prospective payment. It is difficult to capture the value of care provided by trusted primary care clinicians whose relationship with patients is as important a factor in determining success and relevance as any other information that can be collected from claims or clinical data.

We lack, however, the real tools to represent members more authoritatively in achieving transformation of healthcare into a patient-focused system. We have no standing to negotiate directly with payors and, thereby, to assure that family physicians have both the resources necessary to operate their practices and the independence to provide their patients with the care they need.

Some members may feel our messaging about payment should be more focused on empowerment of family physicians to be the advocates and patient partners which we know they want to be and which is part of the image we have cultivated. These members feel our collaboration in promotion of VBP implies endorsement of the essential elements of the various models including use of data identified by policy experts as the standard for measuring effectiveness. Members want their Academy to be more vocal in arguing for the value of what they do, particularly regarding those things that simply cannot be measured, such as the trust which patients place in their physician.

The data reporting and metrics used for evaluation of performance in VBP models are often criticized as too rigid and ineffective in capturing the many variations that exist among practices, communities and patients themselves. It is frustrating to know that even if you meet all of the structural and procedural requirements of a VBP system you may still lose money and forfeit bonus payments because patients do not cooperate or your own advocacy for your patients results in high cost, such as when you use more expensive drugs than a payer permits because you know that the patient is stable on the drug and complying with the payer's formulary would only disrupt that stability.

In my conversations with members on this topic, I hear many examples of actual experience with common occurrences in practice which defy measurement. Good health care and excellence in patient advocacy are inherently expensive. While we have no choice but to continue our participation in testing new payment models to improve revenue for family physicians, we do so knowing that the model we are working within is not and likely never will be ideal. We are a long way from overhauling the health care system and achieving the reform necessary to realize the goal of making health care a human right as we have previously proclaimed and which many of our members sincerely believe.

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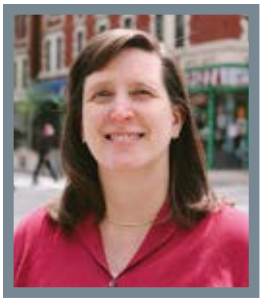

President's Post

By Heather Paladine, MD, MEd, FAAFP

This issue of the Family Doctor journal addresses an important area but often overlooked aspect of family medicine: hospital medicine. As family physicians, most of us focus our clinical care in the outpatient primary care setting. However, our training in comprehensive, patient-centered care also makes us well-suited to providing care in hospital settings. Family doctors are everywhere!

As a residency program director, I know the importance of training in the hospital as well as the office setting. Family physicians learn to manage acutely ill patients, and we excel in coordination of care and taking care of people who fall outside of the comfort zone of other inpatient specialists. What about the 22-year-old with pyelonephritis who is too old for the pediatrics service, but seems out of place in internal medicine? The pregnant patient with community-acquired pneumonia? The patient with challenging social issues who needs close outpatient follow up? These are the types of people who are perfectly within our family medicine wheelhouse.

For those family physicians who do continue to practice inpatient care after residency graduation, the broad scope of practice has benefits not only for our patients but also for ourselves. The American Board of Family Medicine found that family physicians with a broader scope of practice have lower levels of burnout.[1] The authors in this issue of Family Doctor must have the lowest levels of burnout in our state! From newborn hyperbilirubinemia to heart failure to opioid use disorder and more, the articles in this issue span the range of conditions that we encounter as family physicians in the hospital setting.

It is bittersweet that this is my last column as your NYSAFP President. I have had an amazing year of advocacy for and celebration of family medicine. It's exciting to see this great organization move forward with all of your hard work. I'm looking forward to seeing many of you at our Congress of Delegates in May. If you have not been as involved in the past, I hope you will consider joining a commission this year to lend your voice to the NYSAFP; and many congratulations to our incoming President, Rachelle Brilliant. I know that our organization will have a wonderful upcoming year.

Sincerely,

Heather L. Paladine, MD
NYSAFP President

1. Weidner A, Phillips RL, Fang B, Peterson LE. Burnout and Scope of Practice in New Family Physicians. Ann Fam Med 2018; 16(3): 200-205.

It is bittersweet that this is my last column as your NYSAFP President. I have had an amazing year of advocacy for and celebration of family medicine.

Albany Report

By Reid, McNally & Savage

Successful 2024 NYSAFP Advocacy Day

We would like to recognize the over forty family physicians, residents and students who joined us at the State Capitol in Albany on Monday, February 26th for NYSAFP's annual Advocacy Day. Led by President Dr. Paladine, President-elect Dr. Brilliant, Advocacy Chair Dr. Menendez and CEO Vito Grasso, eleven regional teams met with nearly sixty legislative offices, as arranged by our firm, to advocate for the 2024 budget and legislative priorities of NYSAFP. Special thanks to Donna Denley, Director of Finance at NYSAFP for her assistance with planning and executing the Advocacy Day.

During the day, members discussed several budget and legislative priorities as outlined below, some of which were included in Governor Hochul's Executive Budget that was released on January 16th, to promote high quality primary care for New Yorkers. NYSAFP also submitted testimony for the January 23rd Health/Medicaid Joint Legislative Budget hearing to make lawmakers aware of the Academy's budget priorities.

- Supporting primary care investments in the Governor's budget like patient-centered medical homes and a primary care rate increase under Medicaid as well as requiring health care plans to spend a minimum of 12.5% of their overall healthcare spending on primary care services.
- Supporting funding for Doctors Across NY (DANY) and Area Health Education Center (AHEC) workforce programs while urging expanded support under DANY for private practicing physicians.
- Opposing proposals to weaken the health care team by eliminating physician supervision of physician assistants (PAs) and making the law removing collaboration requirements for nurse practitioners (NPs) with over 3600 practice hours permanent.
- Opposing the Executive Budget proposal to cut funding to the State Excess Medical Malpractice program and requiring participating physicians to pay 50% of the cost.
- Expanding telemedicine abortion access and residency training opportunities; NYSAFP is seeking a "legislative add" in the State budget this year of $2 million to support these critical abortion care funding and training needs with the overturning of Roe v. Wade and recent enactment of the shield law.
- Authorizing medical aid in dying legislation.
- Reporting adult vaccines to the statewide and city immunization registries similar to pediatric vaccines.
- Promoting universal healthcare coverage through a single payer health system and much-needed insurance standardization/simplification.

State Budget Update – One House Budget Bills Released

On March 11-12th, the Senate and Assembly released their own one-house budget bills in response to what the Governor proposed and with their own priorities. Below are budget actions of particular interest to NYSAFP related to Academy priorities.

Group Photo at the Renaissance

Capital District/Hudson Valley Team with Assemblymember Shrestha

Sonya Chemouni Bach from Team 5 with Senator Myrie

Leadership Team at the NYS Assembly Chamber Podium

Leadership Team with Senator Hoylman-Sigal

The Senate and Assembly accepted DANY and AHEC funding levels and the inclusion of PCMH investment and primary care rate increase, with the Senate adding $500,000 in support of AHECs. Additionally, both houses rejected PA independent practice and the proposal for New York State to join the Interstate Medical Licensure Compact. However, both houses included a proposal to extend NP independent practice for those with over 3600 practice hours to April 1st, 2026. They also both rejected the Excess Medical Malpractice Program restructuring and funding cut and proposed to extend the program through June 30th, 2025, as NYSAFP requested.

While the Assembly rejected several of the Executive's maternal and reproductive health proposals, including authorizing a statewide standing order for doula services, they provided $5 million for medication abortion care. The Senate included $10 million in addition to the Governor's $25 million in reproductive access funding to create a Reproductive Freedom and Equity Grant program. The Senate also included language allowing any person to give consent for reproductive healthcare including contraception and abortions, without needing a reason, and authorizing any title eight health care practitioners acting within their lawful scope, to prescribe and distribute contraceptive devices or medication.

The Senate one house budget also included language to enact the wrongful death legislation (S.8445) which the Governor has vetoed the last two years and which NYSAFP will continue to strongly oppose. The Senate also included positive language to reform the Office of the Medicaid Inspector General's (OMIG) audit processes.

New proposals from the Assembly one-house budget bills include $5 million for insurance coverage for epinephrine auto-injector devices without patient copay/coinsurance, and a commitment to push for OMIG audit reforms. Both houses also included a new proposal to tax Medicaid managed care plans, which would provide billions in new revenue for Medicaid, and the Assembly added a provision to create a Medicaid investment fund to use monies generated to support healthcare delivery.

Overall, the Legislature provided substantive support in their one house proposals for many NYSAFP priorities thanks to the critical advocacy efforts of Academy members. For more information comparing the Executive Budget to the Senate/Assembly one-house budget bills in the health/mental hygiene sectors, please review our comprehensive HMH Budget Update. With this step in the budget process completed, three-way negotiations commenced between the Governor and Legislature on March 18th in an effort to reach a final budget agreement for SFY 2024-25 due April 1st.

We thank all members for your interest in NYSAFP advocacy efforts on behalf of members and your patients. We encourage all to get be involved through the COD, annual Advocacy Day and by responding to NYSAFP Action Alerts throughout the year to reach out to your state legislators to ask for their support of family medicine!

We will be keeping the pressure on over the next few weeks to advocate for inclusion of NYSAFP's priorities this year in the budget. NYSAFP will provide a member update on the final State Budget outcomes related to priority areas once the final deal comes together and the budget is passed. Following the budget's enactment, NYSAFP will continue to advocate for the advancement of its legislative priorities during the remainder of the session which is scheduled to end in June 2024.

TWO VIEWS: Caring for the Homeless

VIEW ONE

HOSPITAL-BASED CARE FOR ACUTELY-ILL ADULTS EXPERIENCING HOMELESSNESS: MAXIMIZING OPPORTUNITIES AND MINIMIZING HARMS

By Andreas Lazaris, MD, MSC; Jonathan Fricchione, MD and Sandhya Kumar, MD, MPH

Homelessness is an issue of social, racial, and health justice. Homelessness has increased in New York state by 39% from 2022 and 2023, the largest statewide increase in the United States.[1] While many different factors have contributed to homelessness for this large and diverse group of New Yorkers, common experiences include unaffordable housing,[2] structural racism,[3] unmanaged mental illness,[4] history of criminal legal involvement,[5] domestic violence,[6] addiction,[4] and forced displacement.[7] Black and Latinx neighbors are disproportionately affected by homelessness,[1] due to policies that historically excluded Black and Latinx Americans from accessing opportunities for housing, in addition to numerous societal structures that maintain racial inequality.[3] Over half of the heads of households in NYC shelters are Black, and almost one-third are Latinx.[8] Any period of homelessness is destabilizing for the individual and the family and has far-reaching impacts. Homelessness disrupts social supports,[9] contributes to student absenteeism,[10] worsens recidivism,[11] challenges recovery,[4] and negatively impacts health. Homelessness makes it harder to control chronic conditions and easier to develop new medical conditions, contributing to significant morbidity and mortality.[12] In NYC, the leading causes of death among people experiencing homelessness (PEH) include drug-related, heart disease, accidents, alcohol-related, and cancer, with the majority of deaths among PEH aged 45 to 64 years.[13]

Healthcare disparities are evident across the healthcare continuum. Many PEH are unable to engage meaningfully in primary care, substance use treatment, and specialty care services in traditional healthcare settings because of various factors, such as distrust of institutions,[14] competing demands on time or resources,[15] challenges with transportation,[16] fear of being stigmatized,[17] or difficulty tolerating long waits or crowds due to a history of trauma.[18] In hospital settings, studies have shown that up to 30% of admitted patients are experiencing either housing insecurity or homelessness at time of hospital admission.[19] Of the roughly 23 million admissions analyzed nationwide, 515,737 hospitalizations occurred among PEH, of which 447,436 were in NYS alone.[20] The disproportionate impact of homelessness on hospitalization is not just seen with adults.[21] In children, NYS saw 73.8 asthma hospitalizations per 1000 homeless children vs 2.3 per 1000 non-homeless children in 2019.[22] Despite the high medical needs requiring hospitalization in PEH across the age spectrum, those patients leaving the hospital against medical advice

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VIEW TWO

CLOSING THE GAP: THE ROLE OF PRIMARY CARE PHYSICIANS IN THE INPATIENT MANAGEMENT OF THE UNHOUSED POPULATION

By Mayra Goreja, Katarina Peck, and Carlos Swanger, MD

The U.S. Department of Housing and Urban Development (HUD) defines an unhoused individual as a person who lacks a fixed, regular, and adequate nighttime residence. More than 650,000 people experienced homelessness on a single night in January 2023, per the Annual Homeless Assessment Report (AHAR) conducted through the HUD. This represents a 12% increase from data collected in 2022. This figure includes individuals in shelters, temporary housing, and in unsheltered settings.[1]

While HUD's 2023 AHAR demonstrates homelessness as a nationwide issue, New York in particular has a high population of unhoused individuals.[1] The 5 states with the leading rates of homelessness are California, New York, Washington, Oregon, and Florida. New York, in previous years, has had a decrease in homelessness; it is theorized that this decrease was due to increased financial aid available due to the COVID-19 pandemic. In 2023 however, New York had a resurgence of homelessness at a rate of 39.1% as compared to previous data collected in 2020 and 2021. This is thought to be attributable to the recent changes in the rental housing market and the rate of rent growth combined with the conclusion of pandemic protections and programs for housing loss. Major cities tend to have the largest percentage of homeless populations, housing 52.7% of all unhoused people. New York City, NY has the largest population of people experiencing homelessness at 88,025, followed by Los Angeles City & County, CA (71,320), Seattle/King County, WA (14,149), San Diego City and County, CA (10,264), and Metropolitan Denver, CO (10,054).[2]

Homelessness is not merely a social issue, but also a medical issue.[3] Unhoused patients, whether sheltered or unsheltered, experience intense environmental, physical, and mental stressors. The higher mortality rate seen in unhoused individuals as compared to their housed counterparts appears to be related to these adverse influences.[3] In addition, these patients have a shorter life expectancy, an average of 44 years, compared to the housed general public's life expectancy of 74-80 years.[4,5]

The spike in the unhoused population is reflected in the steadily increasing rate of acute hospitalization for this population over the last few years.[6] As homelessness is a commonly encountered social barrier to ambulatory healthcare, these patients are frequent utilizers of acute hospital services.[7] The cause for recurrent hospitalizations of these patients is tied to their underlying complex medical and

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(AMA) are more likely to be homeless, and more likely to be living with diagnosed mental illness and substance use disorders.[23] In NYS specifically, homelessness is associated with significantly higher 30- and 90-day hospital readmission rates.[20] Such data highlight the importance for inpatient and outpatient family medicine colleagues to bridge care gaps while PEH are hospitalized. This includes exploring specific interventions on which hospital teams may focus in order to maximize the effectiveness of inpatient admission for PEH while also aligning individual and care team treatment goals; examining the reasons that PEH may not complete hospital treatment courses, have higher incidences of AMA discharge, and higher rates of subsequent readmission; exploring avenues for collaboration between primary care and hospitalist teams to more effectively utilize the continuum of care; and considering effective methods of interdisciplinary discharge planning to facilitate high-quality primary care post-hospitalizations in PEH and reduce readmissions.

MAXIMIZING EFFECTIVENESS OF INPATIENT ADMISSIONS

The ability to apply the following recommendations is contingent on familiarity with the local network of services for PEH within the community and available in the hospital. For example, some communities have tailored homeless healthcare organizations that deliver healthcare services where patients are residing on the street or in shelters, or when accessing services at agencies or soup kitchens. It is also critical to be familiar with the vast variability of circumstances for PEH, meaning that homelessness may refer to sleeping on the streets or it may refer to staying in a family shelter with regular access to a kitchen and bathroom. It is important to acknowledge that homelessness is a fluid social circumstance, and therefore valuable to ask patients what resources are available and what treatment recommendations are feasible in each new encounter.

Optimizing prevention and disease management – The experience of homelessness challenges the ability to access both preventive and specialty healthcare services for many people.[24] As a result, PEH have decreased rates of screening and monitoring laboratory testing, decreased prescription of and adherence to guideline-directed therapy for chronic illnesses, and decreased rates of completion of screening and diagnostic testing.[25] Utilizing the hospital course as an opportunity to complete United States Preventive Services Task Force (USPSTF) recommended screening labs as well as guideline-based monitoring labs for conditions like chronic kidney disease can provide critical information for primary care providers on discharge, while also allowing for the necessary up-titration of medications in chronic diseases including CHF and hypertension.[26] Relatedly, the hospital course can be seen as an opportunity to complete outpatient diagnostic and interventional procedures which PEH may have had difficulty executing outside of the hospital setting. This may include diagnostic colonoscopy after abnormal FIT result, routine/video EEG in the evaluation of recurrent seizures, and elective surgical procedures for conditions with high possibility for decompensation and complication in PEH.

Managing substance use and withdrawal – For PEH with opioid use disorder (OUD) and alcohol use disorder (AUD), compassionate and thoughtful management of symptoms of withdrawal during hospitalization is critical. Simultaneously, interested patients should also be offered medication treatment for substance use disorders and long-term care. In-hospital connection to substance use treatment can help to close the gap in access to treatment among PEH compared to their housed counterparts.[27] Relatedly, patients with AUD have been shown to have lower 30-day readmission rates and rehospitalization rates in those prescribed naltrexone, which highlights the benefits of hospital-based initiation of either oral or IM naltrexone in those PEH who are ready to initiate treatment.[28] Tantamount to initiation of pharmacotherapy across substance use disorders in PEH, however, is clear coordination with outpatient providers and social work teams to ensure patients have appropriate follow-up and are able to secure refills and necessary dose titration to avoid risks of overdose with re-initiation of use.

Collaborative discharges – Critical to an effective hospital course for PEH is the careful coordination of discharge, even when a patient is discharged against medical advice. Discharge planning should include strategies to overcome common challenges for obtaining and adhering to prescribed regimens, such as acquisition of prescribed medications and durable equipment on discharge, transportation to a community pharmacy, high cost of medications, and incomplete prior authorizations.[29] In response, inpatient providers should consider delivery of prescribed medicines directly to a patient's current housing site via a delivery pharmacy, or directly to a member of a patient's social support team (i.e. case worker or outreach team member) who can provide prescribed medicines to the patient.[30] In settings where an on-site hospital pharmacy can provide PEH with medications in-hand on discharge, this service should be utilized. Prior authorizations should be completed prior to discharge by inpatient teams. Further, hospital providers must consider the methods of communicating care plans and follow-up to outpatient providers. Currently, communication between inpatient and outpatient providers occurs 23–38% of the time and is often restricted to written communication through discharge summaries.[31] Outpatient providers caring for PEH would benefit from actionable to-do-lists including recommended outpatient laboratory studies, clearly reconciled medication lists, pending results, and outpatient referrals that have been placed and appointments that have been made.[31] Where possible, specialist appointments should be made prior to discharge, with appointment details provided on discharge summaries for outpatient providers or social support teams to assist patients in attending.

REASONS FOR INCOMPLETE HOSPITAL TREATMENT COURSES

Many PEH have personally experienced or witnessed harms within institutions including in healthcare, have competing demands on their time and limited resources, and also perceived stigma within the healthcare system, all of which can contribute to interruption or discontinuation of a hospital-based treatment plan.

Institutionalization – For many patients, regardless of prior experience or socioeconomic status, hospitalization for acute illness may demand a significant and distressing forfeiture of control. The hospital itself is an institution that imposes restrictions on how a patient can go about their day, including when and where they can eat, sleep, and use the bathroom.[32] Many patients have experienced

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feeling ignored, coerced, or mismanaged in the healthcare setting. In this way, the hospital may resemble other institutionalized environments with which PEH may have had experience, like jails, prisons, and homeless shelters.[33] For PEH, these other institutions can carry threats of violence, loss of agency, and loss of property.

Competing demands – As with all patients, hospitalization comes at the cost of interruption of day-to-day life. For PEH in particular, this includes caring for children or pets, employment, and attending social services or legal appointments, all while meeting various and sometimes rigid shelter requirements such as curfews. These obligations left untended may prompt discontinuation of care in favor of managing them.[32]

Stigma – The perception of treatment differences based on unhoused status is not unfounded. For example, being identified as homeless affects the administration of potentially life-saving procedures like PCI and CABG, worsening in-hospital mortality even when standardizing for risk.[34] PEH are also on the receiving end of stigmatizing behavior where assumptions are made about withdrawal or the urgency of reported pain. Stigma drives under-recognition of pain, and unmanaged pain is a strong driver of leaving against medical advice (LAMA) among PEH.[35] For those PEH who use fentanyl-containing products, higher starting doses of opioid agonists to control withdrawal are often needed and maximum daily doses of methadone as set by hospital protocols may not reflect the opioid tolerance of people with exposure to fentanyl.

MINIMIZING HARMS OF HOSPITALIZATION

The above context underlies the perceived dangers and harms that physicians often do not associate with hospital care. Exposure and re-exposure to traumatic cues, loss of control, and perceived stigmatization can inform the patient's choice to continue receiving care from a hospital team. Principles of harm reduction and trauma-informed care applied universally and consistently may help mitigate these harms, as these frameworks together recognize the patient as the expert in their life and their needs, and emphasize autonomy, safety, and shared decision-making.

Respecting autonomy – While exploring potential reasons for interruption or discontinuation of a hospital-based treatment plan may help to identify potential points of medical or social intervention to support patient needs, ultimately, it is not the physician's job to determine the validity of a patient's desire to leave a hospitalization early. Respecting a patient's decision for early discharge is essential to a successful collaborative discharge plan.

Maintaining social needs – Early involvement of inpatient social work to assist with these needs helps maintain key, post-discharge lifelines that may otherwise be cut off by the time they leave. For example, disposition and discharge planning during interdisciplinary rounds communicated to shelter-based social work or case management can help with safe transition to a shelter that best accommodates their new functional needs.

Respecting property – Patient belongings may take on outsized importance in daily life. For example, wallets contain IDs that determine access to benefits and backpacks contain key items or medications. These are often targets of theft on the street and in shelter, and they are often set aside by hospital staff when the patient enters the hospital.[36] Ensuring that those items travel with the patient or are secured in view of the patient establishes trust and safety, and decreases patient stress. If belongings are lost, often so too is trust in good intentions of the care team.

Managing pain, withdrawal, and substance use – Substance use is functional for many patients, including for management of pain. Faithfully managing withdrawal and pain are paramount to minimizing iatrogenic harm. Appropriate and early involvement of addiction medicine consult teams is important as studies have shown rapid titration of methadone to treat withdrawal decreases the rate of AMA among patients who use opioids. Caregivers unable to administer clinically appropriate methadone or buprenorphine doses to control withdrawal can consider additional agonists in the hospital setting with appropriate monitoring.[37] Appropriate standing orders for adjunctive pain medications and treatments should also be considered.

Avoiding stigmatizing language and behavior – Documentation provides an opportunity for physicians to extinguish stigmatization toward PEH, substance use and leaving against medical advice (LAMA). Prior documented episodes of LAMA can be seen by physicians as a negative behavior while patients themselves see LAMA as a reason for receiving substandard care should they return to the hospital.[38] By pursuing a collaborative discharge as described above and documenting wherever possible, the physician can dampen this feedback loop of disjointed care and lowered expectations. Lastly, physicians often witness stigmatization at the hands of their fellow providers and care team members. Utilizing "call-in" techniques like privately debriefing about the use of certain stigmatizing words or behaviors can adjust these behaviors, while "calling out" problematic behaviors may also be warranted in urgent or unsafe situations.

CONCLUSION

Medical care for PEH may require a different approach than standard hospital-based care, including thinking creatively about how to safely deviate from usual therapies to provide care that is both acceptable to the patient and feasible given the multiple competing demands on limited resources. Family medicine physicians are well-positioned to reimagine the goals and roles of hospitalization, challenge the compartmentalization of care, and employ strategies for comprehensive care across the healthcare continuum. In doing so, healthcare for PEH will honor and uphold the four pillars of medical ethics: beneficence, non-maleficence, autonomy, and justice.

Endnotes

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social needs, in addition to the lack of longitudinal access to healthcare services.[7] As this population is less likely to be integrated into a primary care system, these individuals primarily use emergency departments for the majority of their healthcare needs, leading to overutilization of hospitalization.[8]

This vulnerable patient population differs from other patients when considering underlying comorbidities and the severity of symptoms at the time of presentation for hospital admission.[9] The lack of access to regular health care and logistical difficulties in following through on medical recommendations for treatment, such as preventative lifestyle changes or regularly taking prescribed medications, culminates in more acute presentations of chronic diseases.[6]

These differences are reflected in factors such as in-hospital mortality, length of stay, and costs of services.[7] Evidence shows that the chronic street homeless population accrues a disproportionate amount of medical costs for recurrent emergency visits and prolonged hospitalizations. This further puts strain on healthcare resources and costs.[10] According to data collected by the National Hospital Ambulatory Medical Care Survey, rates of ED visits by unhoused persons increased from 141 visits per 100 people in 2010 to 310 visits per 100 persons per year in 2020.[11] However, ED visits per 100 housed persons did not significantly change over the same time. In addition to overall higher mortality, hospitalization costs are also higher. In one US study, unhoused patients often had longer inpatient stays, at an average of four days, shown to have a monetary value of $2000-$4000 per day.[12] Another study performed in Canada found that medical admissions for unhoused patients cost $2559 more than those for housed patients, even when controlling for age, gender, and required resources.[13] In addition, psych admissions for unhoused patients cost on average $1058 more than housed psych patient admissions.[13]

Homeless persons have the same medical conditions as the general population. The difference in medical management arises from their exposure to disease agents, overcrowding, unsanitary conditions, poor nutrition, sleep deprivation, violence, physical and emotional trauma, sexual abuse, crime, assault, and weather extremes. Limited education, mental illness, substance abuse, and distrust can affect their ability to respond appropriately to these adverse conditions and manage medical conditions. Based on these factors, homeless persons tend to present with advanced disease, and the approach to therapy is different depending on each person's situation.[14]

Management of this patient population extends beyond the acute medical care that is provided to all patients. Identifying reversible social causes that contribute to the worsening of chronic conditions is vital; social barriers should be addressed in a multidisciplinary fashion while patients are in the inpatient setting. A thorough assessment of social determinants of health should be conducted as a part of the history and physical at the time of admission. This is pertinent to finding the appropriate measures that need to be addressed for the duration of the patient's stay. Questions vital for the social history of these patients include current living conditions, prior homelessness, prior primary care providers, most recent hospitalizations, history and risk of abuse, education level, and available support systems to name a few.[15]

Along with addressing social factors with the help of a multidisciplinary team, other screenings should also be considered while patients are in the hospital setting. Patients from this population are at higher risk for certain diseases less commonly found among housed populations. Homeless patients are at an increased risk for TB, HIV, sexually transmitted infections, hepatitis A and B, malnutrition, alcoholism, substance abuse, skin and foot diseases, and psychoses.[8] It is also recommended that clinicians in the inpatient setting work with admitted patients to complete preventative measures, such as updating vaccinations. In particular, hepatitis B and TDAP vaccines are indicated for this patient population.

Adapted clinical guidelines available through the National Health Care for the Homeless Council are available to further outline the treatment of these patients. These guidelines give special consideration to management when patients are transitioning out of acute care at the time of discharge, as well as simplifying steps to positive outcomes; this can include reducing the number of medications prescribed and ensuring access to outpatient follow-up. Great effort should be made to enact preventative health maintenance to help avoid rehospitalization. An example of accommodations made for the discharge of unhoused diabetic patients is giving insulin injection pens as opposed to standard insulin vials and syringes because insulin vials often require refrigeration, while syringes might be used for substance abuse or sold to others.[9]

Special considerations for prescriptions given at the time of discharge include avoiding medications that have significant sedative effects unless tried for an extended period in a safe environment, as this can diminish alertness and may directly affect patient safety in shelters or on the streets. Diuretics and commonly prescribed medications with gastrointestinal side effects, namely diarrhea, can prove a problem when there is limited access to bathroom facilities and can cause dehydration in the warmer months. Other considerations include patients prescribed statins, as the unhoused population has a higher prevalence of hepatitis. As such, liver function tests should be followed closely. When discharging patients with hypertension, precaution should be taken when prescribing beta blockers, as these can prove dangerous for individuals who are cocaine users; an additional alpha blocker, or combination alpha and beta blockers may be a better choice for individuals with a known substance abuse history. Careful education regarding medications prescribed to patients, as well as their side effects should be addressed.[16]

It has been found that the use of medical respite programs following acute inpatient discharge significantly reduced rehospitalizations and ED visits. Researchers proposed that these resource reductions allowed for an estimated hospital care cost savings of $6,307. These programs are particularly useful as they allow unhoused patients to be discharged to a stable environment for an average of two to four weeks with varying nursing care available to allow for healing that would be otherwise difficult to achieve if the patient were to return to a shelter or the streets.[17]

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Family physicians, are well-suited for setting up unhoused patient populations for success at the time of discharge from an inpatient setting with outpatient follow-up, allowing for longitudinal, cost-effective preventative care to improve the long-term health of individuals; this allows for closing the metaphorical gap in healthcare witnessed in regards to this patient population.[18] Primary care physicians (PCPs) have long been lauded for their wide breadth of knowledge, as well as for their ability to coordinate holistic, patient-centered care.[19,20,21] The UK has had great success in utilizing inpatient PCPs in special programs to support homeless patients.[18] They review patients, collaborate across specialties, advocate for patients with inpatient teams, educate patients on the healthcare system, and act as a facilitator with external officials. Primary care providers are also able to write letters supporting patient needs to housing councils. Across the three hospital pathways in this program, an average of 50% of patients have been rehomed, in addition to a significant number of instances of the teams preventing patient evictions or removal of their homes. These providers also hold educational events for their colleagues with the express intent of transforming prejudice and hospital culture. Coding homeless status, improving hospital reimbursement and more accurately documenting the work of hospital staff have also improved, in addition to better addressing the complex needs of these patients. This homeless consult service also allows for consults in the emergency department to help ensure longitudinal care. Their program hosts "frequent attenders' meetings" where the homeless team can discuss patient circumstances and work towards securing housing for their frequent attenders.[18]

Creating a compassionate, goal-oriented care plan for the homeless would reduce reliance on acute hospital services, and primary care providers have been proven to be the ideal physicians to do so. Devoting time and attention to providing an empathetic patient care visit better builds rapport with patients in this vulnerable population, making them more likely to continue returning for care. This continued care ultimately leads to more continuous patient care and a decrease in the readmission rate for the unhoused population.[22]

As medical students partaking in and learning from the providers at the Health Reach for the Homeless Outreach Program and Clinic, we have witnessed the difference simple preventive interventions can provide in the quality of care these patients receive. By pairing hypertension screening clinics across shelters in the city of Rochester with virtual telemedicine clinics with Health Reach's medical director, we have been able to help mitigate medical exacerbations with early intervention and easier access to healthcare services.[23] For instance, numerous instances of hypertensive urgencies have been caught through the blood pressure screening clinics held around the city. A common response from patients, when asked the reason for not continuing their prescribed antihypertensives, was because their prescribed medications had been stolen at shelters. Through telemedicine visits, medications are re-prescribed and can be picked up at the pharmacy closest to the shelter, with a bus pass provided when necessary. The experience of supporting individuals through this process has been significant in forming our mindsets as future clinicians.

The University of Rochester's Highland Hospital also offers a consultation service for inpatient management for people experiencing homelessness. The service emphasizes a complete, holistic inpatient visit for this population including a thorough history with attention to vision and hearing difficulties, preferred primary language, English literacy, and education level. The detailed discharge plans include current housing, insurance details, assigned care manager, accessibility to a cell phone or internet access, and method of transportation anticipated for follow-up visits. Medication adherence and discharge instructions are discussed, and written guidelines are provided at a fourth-grade level per national recommendations.

As medical students, we have also had the unique experience of participating in the efforts of the Regional Health Reach Street Medicine team. These healthcare workers regularly take their efforts directly to the unhoused population in the streets of Rochester, and a strong kinship between providers and patients is evident in every interaction. The care and attention that underlies each patient encounter, whether in the back of a van or in a shelter for transitional housing, is tangible. Knowing individuals on a personal level has strengthened trust with this population, resulting in a decrease in the overall rate of preventable acute hospital admissions and subsequent hospital readmissions. Street medicine programs have been shown to produce significant cost savings and represent a cost-effective delivery model that improves health outcomes in underserved populations.[23]

This population often exists without underlying medical, social, or psychiatric concerns being adequately addressed. It is only at the time of a critical problem that individuals from this population are brought to the attention of the healthcare system, often for short-term interventions with subsequent discharge back to the social arrangements that created their previous environmental stressors, reinforcing the vicious circle of continued homelessness.[24] Policy efforts should address barriers to the use of ambulatory care services and behavioral health services in particular, to help reduce acute care use and improve the long-term health of homeless individuals.[8] Holistic inpatient treatment of these patient's acute medical conditions, while addressing the social factors that create barriers to their health and well-being, are priorities for the care of this vulnerable population.

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Antibiotic Resistance:
Exploring the Negative Impact, Risk Factors, Guidelines, Prevention, and Treatment Approach for Multidrug Resistant Bacteria

By Lily Sitisa, MMS; Rebecca Cyrek; Colton Davis and Elizabeth Loomis MD, FAAFP

Overview

Antibiotics are credited for saving millions of patients from premature death due to bacterial infection; however, the exceptional progress the healthcare system has made with the introduction of antibiotics has been threatened by the growth of resistant bacteria. Antibiotic resistance is a growing health concern in the United States with an estimated 2.8 million antibiotic resistant infections per year.[1] Specifically, hospital-acquired multidrug resistant bacteria have an increased mortality, length of stay, and hospital costs.[2,3] This article will focus on the current risk factors for developing antibiotic resistance and will provide guidelines for empiric treatment of common infections encountered in the inpatient setting. These include community and hospital acquired pneumonia, urinary tract infections, intra-abdominal infections, catheter site, and skin infections. The article will also discuss the risk factors for developing multidrug resistant bacteria including poor antibiotic stewardship, improper de-escalation of antibiotics, and not adhering to empiric guidelines. Additionally, this paper will cover how to properly approach patients who have known multidrug resistance and important prevention techniques.

Introduction and the negative impact of resistance on the hospital system

Prior to the discovery of antibiotics in the 20th century, the morbidity and mortality rates of infectious diseases were significantly higher than they are today.[4] Before antibiotics were introduced, herbs, honey, animal feces, and moldy bread were utilized to treat infections and of these treatments, the use of moldy bread was the most successful.[4] In 1928, a bacteriologist, Alexander Fleming noticed a zone around an invading mold where bacteria did not grow and after isolating and extracting the mold, he named the active agent penicillin.[5] Fleming found that penicillin had an antibacterial effect on gram positive bacteria such as staphylococci.[5] The years following the discovery of penicillin became known as the "golden age" of antibiotics as new agents such as streptomycin and vancomycin were introduced and available for patient use.[4] However, the efficacy of antibiotics in treating bacterial infections have decreased with their large-scale use as bacteria began to develop antibiotic resistance.[5]

The negative impact of antibiotic resistance on the hospital system includes increased mortality, length of stay, and hospital costs.[2,3] In patients with infections caused by multidrug resistant bacteria, options for treatment with antibiotics that the bacteria are susceptible to are limited. With limited antibiotic treatment options, patients are at increased risk of complications such as sepsis or dissemination of disease which increases risk of mortality.[6] During 2019-2020, the Centers for Disease Control and Prevention (CDC) estimated that antibiotic resistance increased by 15%, leading to 29,400 additional deaths from infection in the United States and of that, 40% were due to hospital acquired infections.[7] Patients that develop complications from infections require a longer length of stay at the hospital to ensure hemo-dynamic stability, clearance of infection, and completion of treatment course. A longer length of stay leads to increased hospital cost and increased risk of nosocomial infections.

Given the increase in antibiotic resistant infections, it is important that health-care professionals are aware of the negative impact of large-scale antibiotic use and multidrug resistant bacteria on mortality risk, length of hospitalization, and hospital costs for patients with infections.

Risk factors for the development of resistance to antibiotic

Antibiotic resistance is the ability of bacteria to resist or develop defense mechanisms against the antibiotics that were designed to kill them (bactericidal) or suppress their growth or activity (bacteriostatic).[8] Multidrug resistant bacteria have posed several challenges in treating infections in the inpatient setting since there are limited antibiotic treatment options. The risk factors associated with developing multidrug resistant bacteria are poor antibiotic stewardship, improper de-escalation of antibiotics, and not adhering to empiric guidelines.

Antibiotic stewardship refers to efforts in monitoring and improving the prescription and usage of antibiotics including appropriate selection, dosage, and duration of antibiotic treatment for optimal efficacy in treating infections.[9] The inappropriate selection of antibiotic, dosage, and duration will support bacterial genetic alterations or increase virulence, allowing the bacteria to become resistant to many antibiotics.[10] In addition, even if the appropriate antibiotic was chosen, suboptimal dosage or duration of antibiotic can promote the development of multidrug resistance since it gives the bacteria the opportunity to tolerate, adapt, and survive the effect of the antibiotic.[10]

De-escalation of antibiotics refers to the discontinuation of one or more components of the combination empirical therapy or switching from a broad-spectrum to a narrower spectrum antibiotic to decrease the risk of antibiotic resistance.[11] While blood cultures or urine cultures are pending, broad-spectrum antibiotics are utilized to ensure proper coverage for all possible causes of infection. However, once culture and susceptibility results are available, there is no longer the need for a broad-spectrum antibiotic as a narrow spectrum antibiotic will ensure that the bacteria causing the infection is targeted. Improper de-escalation of antibiotics or continuing with broad-spectrum antibiotics will allow other bacteria in the microbiome to tolerate and adapt to the effects of the broad-spectrum antibiotic and eventually develop resistance.[11]

Empiric guidelines were developed to assure effective treatment, decrease treatment diversity, and reduce the unnecessary use of broad-spectrum antibiotics.[12] When a patient exhibits signs and symptoms of an infection but the cause of the infection is unknown, empiric therapy with broad-spectrum antibiotics is utilized to ensure adequate coverage of bacteria and prevent complications. Once the cultures and susceptibility results are available, the broad-spectrum antibiotic can be discontinued and the proper antibiotic with the proper dosage and duration can be utilized. If however, empiric guidelines are not adhered to, not only will the infection not be successfully treated, but other bacteria in the microbiome will have the opportunity to adapt and survive the antibiotic and develop resistance to many antibiotics over time.[12]

For example, with an elderly patient from a nursing home presenting the emergency department with pneumonia, the empiric antibiotic is piperacillin-tazobactam to include *Pseudomonas aeruginosa* coverage.[12] If empiric guidelines are not adhered to and the patient is treated with ceftriaxone, the unnecessary exposure to this antibiotic may allow the bacteria causing the pneumonia to develop resistance. If this has happened many times, the bacteria may become multidrug resistant. Hence, appropriate antibiotic stewardship, proper de-escalation of antibiotics, and adherence to empiric guidelines can help limit unnecessary exposure to broad-spectrum antibiotics and reduce multidrug resistance.

Empiric antibiotic guidelines

Empiric antibiotic guidelines have been created based on experience and observation. Many physicians follow these guidelines on a daily basis using clinical judgment and reasoning. Early treatment with antibiotics can be lifesaving in many patients who cannot wait for bacterial cultures to return. For example, patients that meet sepsis criteria rely on accurate infection localization and initiation of appropriate empiric antibiotics. The following sections discuss empiric antibiotic guidelines based on the Infectious Disease Society of America (IDSA) for several of the most common infections including community and hospital acquired pneumonia, urinary tract, intra-abdominal, and skin infections. While this article provides a broad overview of empiric antibiotics, it is important to also consult local antibiograms to better tailor empiric treatment. Antibiograms provide a summary of antimicrobial susceptibility based on data from a local hospital or health care system.

Community and hospital acquired pneumonia

Typical community acquired pneumonia (CAP) is most commonly caused by *Streptococcus pneumoniae*, *Moraxella catarrhalis*, and *Haemophilus influenzae*. Atypical CAP should include coverage of *Chlamydophila pneumoniae* and *Legionella pneumoniae*. Inpatient empiric treatment of CAP includes combination therapy with a beta-lactam and a macrolide or monotherapy with a respiratory fluoroquinolone. Pneumonia that is acquired within 48 hours of admission to a hospital is defined as hospital acquired pneumonia (HAP). This includes bacteria that are not incubating at the time of admission. When the patient meets criteria for HAP it is important to cover for organisms including *Pseudomonas aeruginosa* and methicillin-resistant *Staphylococcus aureus*. Risk factors for MRSA and *P. aeruginosa* include prior colonization or hospitalization and parenteral antibiotic exposure in the past 90 days.[13] Vancomycin and linezolid

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are empiric options for patients with risk factors for MRSA. For patients with risk factors for *P. aeruginosa*, current recommendations include piperacillin-tazobactam, cefepime, ceftazidime, aztreonam, meropenem, or imipenem.[13]

Urinary tract infection

Uncomplicated cystitis is diagnosed clinically and treated empirically for the most common pathogens including *Escherichia coli*, *Klebsiella pneumoniae*, *Proteus mirabilis*, and *Staphylococcus saprophyticus*. Empiric antibiotics for uncomplicated cystitis include nitrofurantoin, trimethoprim sulfamethoxazole (TMP-SMX), and fosfomycin. Beta-lactams can be considered when there are contraindications to the previously stated medications. Amoxicillin and ampicillin are not recommended for the treatment of cystitis due to poor efficacy and widespread resistance.[14] Fluoroquinolones such as ciprofloxacin are typically reserved when pyelonephritis is suspected.

Intra-abdominal infection

Intra-abdominal infections are defined as infections of the peritoneal space that can be categorized into primary (localized), secondary (breakdown of surgical anastomosis, perforation, traumatic injury, or ischemic necrosis), or tertiary (hospital acquired post-operatively).[15] Severe infections should be treated promptly with broad spectrum antibiotics with enteric gram-negative coverage and surgical source control. Initial empiric treatment of extra-biliary complicated intra-abdominal infection should include ceftriaxone, cefazolin, cefotaxime, ciprofloxacin, or levofloxacin in combination with metronidazole. For those at high risk including those with advanced age or immunocompromised state, cefepime, ceftazidime, ciprofloxacin, or levofloxacin in combination with metronidazole can be used. Initial empiric treatment of biliary infection should include cefazolin, cefuroxime, or ceftriaxone.[16] Data suggests mild, hemodynamically stable patients should be treated based on culture and sensitivity data.[15]

Catheter-site infection

Catheter-site infections (CRI) may be localized presenting signs of inflammation, purulence or cellulitis. Infections originating from catheter sites may lead to bacteremia and subsequent sepsis. Empiric antimicrobial therapy should be initiated if the catheter is not removed, in the presence of central venous or surgically implanted catheters, patients with severe sepsis, neutropenia, suppurative phlebitis, embolization and acute endocarditis.[17] Empiric antibiotics should include coverage of staphylococci with vancomycin and the addition of an antipseudomonal agent if the patient is neutropenic, a burn victim, or is septic. These agents include a fourth-generation cephalosporin, carbapenem, or beta-lactam/beta-lactamase combination, with or without an aminoglycoside. One should include coverage of candidemia with an echinocandin when a patient has had a prolonged course of broad-spectrum antibiotics, malignancy, femoral catheterization, or receiving total parenteral nutrition.[18] These therapies should be de-escalated as soon as blood cultures and sensitivities return.[17]

Skin infection

Bacterial skin infections are a common reason for hospitalization in the United States with the most common infectious organism being *Staphylococcus aureus*. Higher rates of abscesses, infection recurrence, treatment failure and hospitalization has occurred with the increase of community-associated methicillin-resistant *Staphylococcus aureus* (MRSA). Current guidelines suggest vancomycin, linezolid, tigecycline, daptomycin, ceftaroline, and telavancin for severe purulent infections. For moderate purulent infections, trimethoprim-sulfamethoxazole and doxycycline are recommended.[19]

Prevention techniques

One of the most powerful prevention techniques is educating patients, the general public, and healthcare professionals. The public should be educated on appropriate hygiene, hand washing, symptomatic treatment, and taking medications as prescribed. Healthcare professionals should remain up to date with guidelines on antibiotic stewardship in addition to maintaining hygiene, hand washing, and disinfection to protect themselves and their patients. Globally, there are several different phenomena that contribute to antibiotic resistance. Increased antibiotic usage in agriculture has been a growing concern as animals account for 10% more in antibiotic sales compared to humans. U.S. citizens are exposed to antimicrobial resistant bacteria through direct contact with animals, water supply, vegetation, and consumption of animal products.[15] Another global health concern is easy access to antibiotics in developing countries without medical guidance. Antibiotics are taken inappropriately or at inadequate levels of active ingredients increasing the amount of antibiotic resistance bacteria.[15] While correction of global health concerns is difficult, physicians can do their best to combat this issue with patient education and keeping up with evidence based antibiotic stewardship guidelines.

Approach to patients with known resistance

Multidrug-resistant bacteria are often difficult to treat effectively. It is imperative to know the treatment strategies against multidrug-resistant bacteria which are commonly seen in the hospital setting as these infections often have drastic consequences on morbidity and mortality of patients. When approaching a patient with an infection caused by a multidrug-resistant-bacteria, treatment including infectious disease specialists, pharmacists, and members of the local antibiotic stewardship program is recommended. As discussed above, empiric treatment should be guided by the most common pathogens for the presenting infection. A physician should also consider previous organisms identified along with susceptibility, antibiotic usage in the last month, and local community bacterial susceptibility. The most commonly seen drug

resistant bacteria include methicillin-resistant *Staphylococcus aureus* (MRSA), carbapenem-resistant *Enterobacteriaceae* (CRE), vancomycin-resistant *Enterococcus* (VRE), extended-spectrum b-lactamase-producing *Enterobacterales* (ESBL-E), and multidrug-resistant *Pseudomonas aeruginosa*. Below are the standard treatments in the hospital setting barring the multidrug-resistant bacteria is not susceptible to standard empiric treatment.[20]

MRSA infections are one of the leading causes of soft tissue skin infections. The drug of choice for combating MRSA infections in the hospital setting is intravenous vancomycin. Other options include oral or intravenous linezolid, intravenous daptomycin, oral or intravenous clindamycin, and intravenous telavancin. One can consider combination therapy when the infection is refractory to standard treatment.[21]

The drugs of choice for combatting pyelonephritis and complicated urinary tract infections caused by CRE in the hospital setting include ceftazidime-avibactam, meropenem-vaborbactam, imipenem-cilastatin-relebactam, and cefiderocol. Aminoglycosides can also be used. When CRE infections are outside of the urinary tract, ceftazidime-avibactam plus aztreonam, or cefiderocol as monotherapy can be used prior to culture data.[20]

The drug of choice for combating VRE is ampicillin. When bacteremic infections are resistant to ampicillin, daptomycin or linezolid can be used. When a patient has a VRE uncomplicated urinary tract infection, nitrofurantoin and fosfomycin can also be used.[22]

The drugs of choice for combating ESBL-E in cases of complicated urinary tract infections include TMP-SMX, ciprofloxacin, or levofloxacin. Carbapenems are preferred agents when TMP-SMX or fluoroquinolones have resistance. Lastly, aminoglycosides can be used. When ESBL-E infections are located outside of the urinary tract system treatment should include meropenem, imipenem-cilastatin, or ertapenem.[20]

The drugs of choice for combating multidrug-resistant *Pseudomonas aeruginosa* include traditional non-carbapenem b-lactam agents such as piperacillin-tazobactam, ceftazidime, cefepime, and aztreonam. Secondary options include fluoroquinolones or carbapenems.[20]

Conclusion

Antibiotic resistance is a growing health concern in the United States leading to increased mortality, mortality, length of stay, and hospital cost. The most common risk factors for developing multidrug-resistant bacteria include poor antibiotic stewardship, improper de-escalation of therapy, and not adhering to empiric treatment guidelines. Staying up to date and referring to empiric treatment guidelines can aid in the reduction of the consequences of multidrug-resistant bacteria. The most important technique in preventing multidrug-resistant bacteria is education to providers, patients, and the general public. When approaching a patient with known multidrug resistance it is imperative to have a basic understanding of the options of drugs to help combat the bacteria. Working with an infectious disease team and pharmacist can help in your management. Further advances in technology as well as antimicrobials are an important topic of research to be further evaluated in treating and reducing multidrug-resistant bacterial infections.

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Review of Updated Guidelines on Deep Vein Thrombosis Diagnosis and Management for Family Physicians

By Janice C. Lau, MD; Christine Ly, MD, PhD and Sandy Wang, MD, MPH

Introduction

Deep vein thrombosis (DVT) is a common venous thromboembolic event (VTE) with an incidence rate of 1.6 per 1000.[1] Multiple studies suggest that many DVT diagnoses are missed, with one study estimating that more than 50% of cases are undiagnosed.[2] In addition, at least one-third of these individuals will experience a recurring thromboembolic event in the next ten years. DVTs can lead to complications, such as venous ulcers, pulmonary embolisms (PEs), which can lead to sudden deaths, and up to 20-50% can develop post-thrombotic syndrome (PTS), a painful disability due to chronic venous insufficiency.[3] Like other chronic pain syndromes, PTS and other DVT adverse sequelae can lead to hospitalizations and have negative effects on quality of life, resulting in social and financial costs to both the healthcare system, with estimates from the Centers for Disease Control and Prevention as high as $10 billion annually, and to the individuals themselves, costing over $15,000 for each event.[4] Thus, it is important for family medicine physicians to be able to identify, treat, and prevent DVTs in high-risk patients, such as those that present in the emergency and hospital settings.

Pathophysiology, Risk Factors, and Clinical Presentation of DVTs

A DVT is a blood clot that can form in the deep veins of the upper, but more often, the lower extremities (LE). According to Virchow's Triad, there are three main contributing factors in developing clots: impaired venous states or blood flow turbulence, hypercoagulable states, and vascular endothelial injury. The locations of the deep veins of the legs and their valves are prime locations for DVTs to form because of the decreased changes in blood flow, potentially leading to venous stasis.[5] Common locations include the distal calf, femoropopliteal, and iliofemoral veins.[6] Other types of blood clots include upper extremity DVT, splanchnic thrombosis, cerebral vein thrombosis, or portal vein thrombosis.

Immobility is one of the main risk factors that would increase an individual's risk of developing DVTs, especially if a patient is hospitalized, or lying in bed for multiple days. It is also important for family physicians to study a patient's full medical history including smoking, birth control use, history of cancer, and any condition that could fit into Virchow's triad (Table 1).

Clinical Presentation and Determining Pretest Probability

In addition to the severity and location, DVTs vary in clinical signs and symptoms, which are often non-specific, making their diagnoses challenging. The classic clinical symptoms of DVTs include unilateral or asymmetrical extremity edema, pain, warmth, and skin color changes (often red or purple). With our diverse patient population, it is important to be attentive to patients of darker skin tones, as erythema may not be as obvious. Additional signs may include fever, peripheral cyanosis, and a positive Homan's sign (pain upon passive dorsiflexion of the foot). The more proximal the location of the DVT, the more severe the symptoms. However, approximately 50% of DVTs do not present with any clinical signs, which can further make diagnoses challenging.[7]

Lower Extremity DVT Diagnostic Strategies

To begin, we first determine the pretest probability (PTP). One commonly used and validated score in the outpatient setting is the Wells score (Table 2), which takes into consideration risk factors to stratify patients by likelihood of having a LE DVT: 0 or less is low, 1-2 is moderate, and 3 or more is high risk for DVT. Multiple studies have shown overall good correlation between predicted and observed probabilities of DVT based on the Wells score, with ≤ 1 being unlikely and > 1 being likely. However, even with a score of -2, the prevalence of DVT in this group was still 5%.[8] Additionally, multiple studies have shown that compared to in the outpatient setting, using the Wells score in the inpatient setting has a higher

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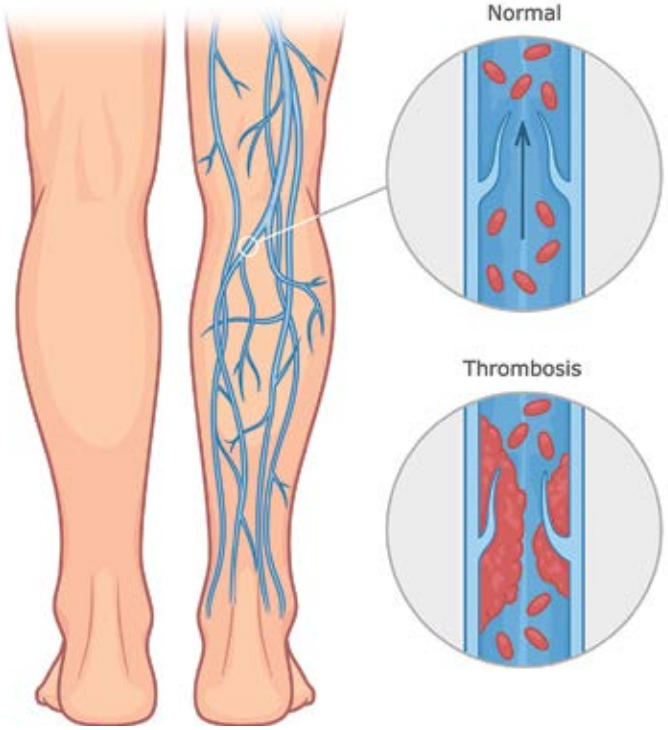

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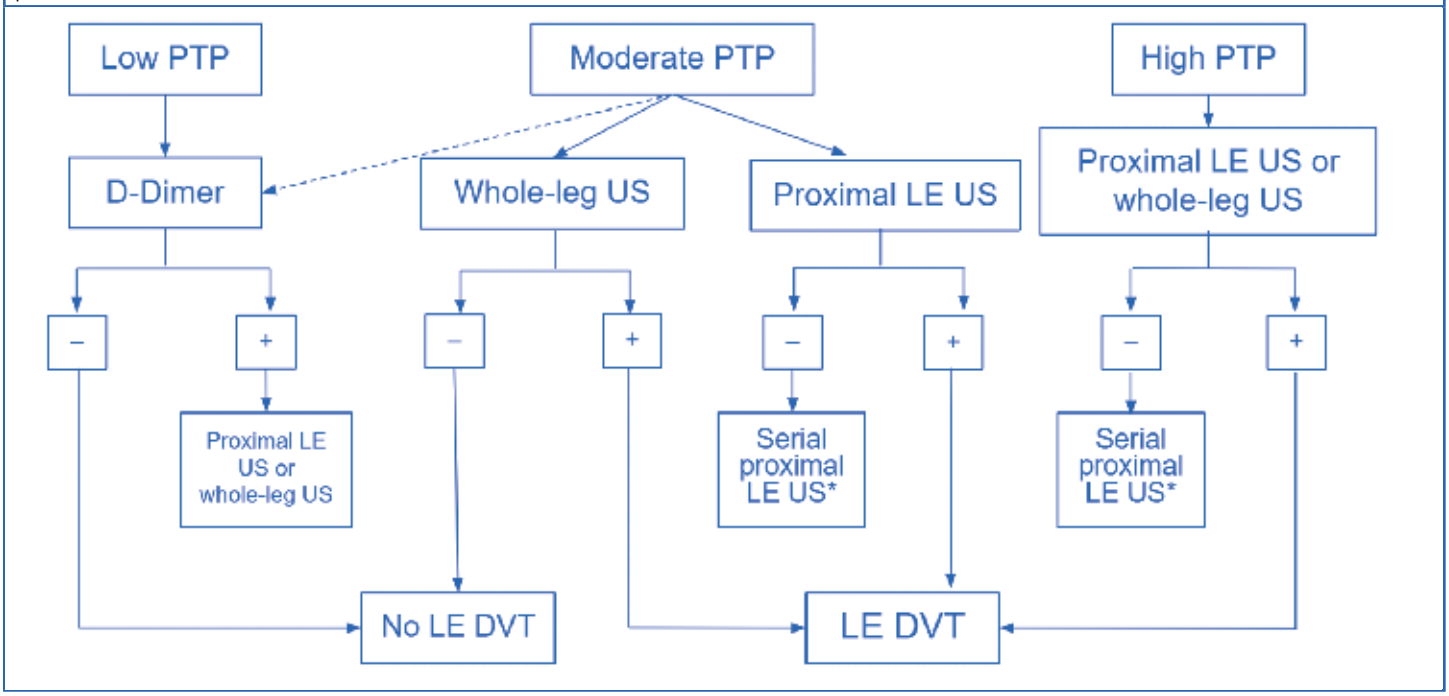
Figure 1: Schematic of ASH 2018 guidelines for LE DVT diagnostic workup[10]
PTP = pretest probability; LE = lower extremity; DVT = deep vein thrombosis; US = ultrasound; – represents negative; + represents positive. Arrow lines represent direction of flow. Dashed arrow line path for low-moderate PTP with consideration for initial D-dimer test. * repeat proximal LE US in 1 week from initial US.



failure rate and a lower efficiency. This may be due to increased prevalence of recent immobilization, surgical procedures, active cancer or routine use of VTE prophylaxis.[9] Therefore, the Wells score alone in both the outpatient and inpatient settings was not enough to rule out DVT.

In 2018, the American Society of Hematology (ASH) published diagnostic workup guidelines for suspected first DVT episode in the LE, as detailed in Figure 1 above.

For concerns of recurrent DVT, ASH guidelines recommend starting with a D-dimer test in those with low PTP to rule out recurrent DVT. If there is a positive D-dimer or high PTP, then proceed with proximal LE US. If the US is negative, but clinical suspicion remains high, then follow up with a repeat US in 1 week.

Upper Extremity DVT Diagnostic Strategies

UE DVTs account for about 4-11% of all DVTs, and are more commonly encountered in inpatient medicine than in outpatient settings as a result of patients who tend to be more acutely ill and with increased risk factors, such as presence of central venous catheters, recent procedures and surgeries, cancer, or prolonged immobility.[11,12]

Constans et al. developed the Constans score to help assess likelihood of a patient having a UE DVT by focusing on 4 key points:

1. presence of indwelling catheter (+1)
2. localized pain (+1)
3. unilateral edema (+1)
4. another plausible diagnosis (-1).[13]

Per ASH guidelines (Figure 2), a Constans score less than or equal to 1 suggests a UE DVT is unlikely. In this case, obtain a D-dimer. If negative, then most likely negative for UE DVT. If positive, then obtain a duplex US. If the initial US is negative, but clinical suspicion for DVT remains high, then obtain a serial duplex US one week later. If Constans score is 2 or more, then it is likely. ASH guidelines recommend either starting with a D-dimer followed by imaging, or imaging alone.[14]

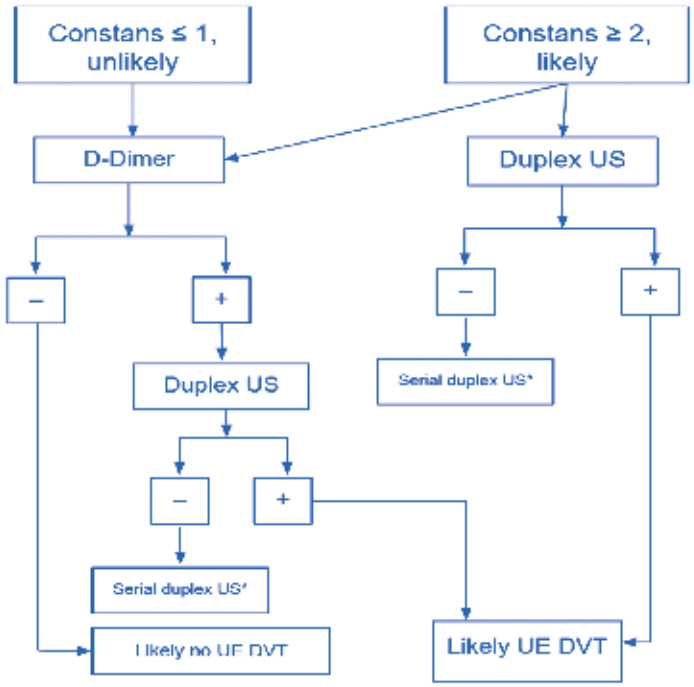
Adverse Complications of DVTs

With treatment, many incidences of DVTs can fully resolve and do not result in any complications. However, there are high rates of recurrence, which increases the risk of morbidity and mortality, with approximately 6% resulting in death.[16] The two major complications of DVTs are PEs and PTS. PEs occur when DVTs detach and travel to the pulmonary vascular system, resulting in impaired gas exchange and circulation. Approximately 12% of PEs occur within a month of a DVT diagnosis.[17] Overall, PEs are associated with very high mortality and morbidity.

We wish to raise awareness of post-thrombotic syndrome (PTS), although less commonly seen, is also associated with significant morbidity, resulting in a markedly decreased quality of life. It can develop within two years of a DVT diagnosis, with symptoms of PTS (Table 3) differing among individuals, can be intermittent or persistent, and can progress throughout the day. The chronic nature of PTS symptoms can be disabling and can involve multiple hospitalizations for pain control. Despite the many treatments for PTS symptoms (e.g. compression therapy, exercise, surgical or endovascular procedures, etc.), the most ideal method is to prevent the initial DVT, if possible, and to promptly treat and prevent recurrent DVTs, especially in the ipsilateral extremity.

Figure 2: Schematic of ASH 2018 guidelines for UE DVT diagnostic workup[15]

UE = upper extremity; DVT = deep vein thrombosis; US = ultrasound; – represents negative; + represents positive. Arrow lines represent direction of flow. * repeat duplex US in 1 week from initial US.



The Basics of DVT Management

The goal of DVT treatment is to prevent negative complications, such as PEs, chronic venous insufficiency syndromes, and PTS. Duration of treatment can vary depending on the cause, initial versus recurrence, and kidney function. The recommended first-line treatment of choice for DVTs is the direct oral anticoagulant (DOAC). According to the ASH guidelines, acute DVT associated with transient risk factors can be treated for 3-6 months, whereas DVTs associated with a chronic risk factor often require indefinite anticoagulation. Of note, it is not recommended to use testing, imaging, or prognostic calculators to determine the duration of treatment. In some cases of limb-threatening DVT, thrombolysis can be a treatment option. When anticoagulation is contraindicated, then an inferior vena cava filter can be considered to prevent PEs.

DVT Prophylaxis in the Hospital

Hospitalization with an acute illness is one of the most important risk factors for developing a DVT, estimated to increase the risk by 10 times.[18] In 2006, the National Quality Forum and the Joint Commission issued a recommendation for hospitals to have policies regarding the risk assessment, prevention, diagnosis, and treatment of DVTs and VTEs in the hospital.[19] DVT treatment should also take the following factors into consideration: comorbidities/drug interactions, mode of administration, and cost (Table 4).

DVT in Pregnancy

Family medicine physicians care for pregnant patients, a unique population at high risk for hypercoagulation. During pregnancy, increased venous stasis, decreased venous outflow and altered levels of coagulation factors result in an increased thrombogenic state. While DVT in nonpregnant individuals more commonly occur in the distal veins, pregnancy-related DVTs tend to occur in the iliofemoral (64%) and iliac (17%) veins.[20] Compared to the first and second, the third trimester has a higher VTE risk. Highest risk is during the postpartum period, especially the first week. Additionally, since D-dimer levels increase progressively during pregnancy, the American College of Obstetricians and Gynecologists (ACOG) guidelines suggest it is not helpful in predicting VTE risk in pregnant individuals.[21] Given that pregnancy-associated changes are not incorporated into the risk assessment tools discussed above, diagnosis and management of DVT during pregnancy is challenging.

ACOG published clinical management guidelines regarding thromboembolism in pregnancy. Unlike in non-pregnant individuals, there is no widely accepted scoring system to assess risk for DVT during pregnancy. Rather than obtaining a D-dimer if a new DVT is suspected, start with a compression US of the proximal veins. If the initial US is negative or equivocal and concerns remain high, then obtain doppler US of iliac vein, venography, or MRI. Empiric anticoagulation can also be considered based on the clinical scenario. If the initial US is negative, then consider repeat imaging in 3 and 7 days.[22]

Pregnancy-related DVT should be treated with anti-coagulation for a total duration of 3-6 months with at least 6 weeks of postpartum therapy. Anticoagulation should be resumed no sooner than 4-6 hours post vaginal delivery or 6-12 hours after cesarean delivery. Pneumatic compression devices should be used until the patient is ambulatory and anticoagulation therapy is restarted. Warfarin, low-molecular weight heparin, and unfractionated heparin do not accumulate in breast milk and are safe to take during breastfeeding.[23]

Conclusion

Family physicians are uniquely trained to treat DVTs both in the outpatient and inpatient settings. Inpatient patients already have a higher pretest probability of DVT due to their immobility, so physicians must be extra vigilant in suspecting a DVT and should also balance cost effectiveness of possible expensive further work up. It is important to review established guidelines to help guide management but also know patients' medical histories well to further substantiate less common risk factors that would help deciding when to begin work up (Virchow's triad). We also remind our colleagues of the unique pregnant patients who do not fall under pretest probabilities and would then rely on a more expensive work up, such as imaging, that would not be performed for non-pregnant patients. Moreover, starting early treatment for DVTs is imperative to help prevent the sequalae of chronic pain syndromes, such as PTS and recurrent DVTs. With all of this, we can help improve quality of life, alleviate the socio-economic burdens of DVT, and improve the mortality and morbidity of our patients.

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Table 1: Risk Factors in Developing DVTs

Venous Stasis or Blood Flow Changes	Hypercoagulability	Vascular Endothelial Injury (Damage to Vessel Wall)	Constitutional Risk Factors
Immobility Travel Stenosis Anatomic variants in venous anatomy Obesity (BMI > 30kg/m2)	Hypertension Diabetes Heart Failure Pregnancy Malignant neoplasms Thrombocytosis Polycythemia vera Sepsis Vasculitis Inflammatory bowel disease (Crohn's, ulcerative colitis) Genetic conditions/mutations (Factor V Leiden, prothrombin 20210, deficiencies in antithrombin, protein C, and protein S) Myocardial infarction Systemic Lupus Erythematous, Lupus anticoagulant Nephrotic syndrome Oral estrogen therapy (birth control pills, hormone replacement therapy) Smoking	Major trauma Major surgery (pelvic, orthopedic, etc.) Fractures Central venous catheters Pacemakers Intravenous drug use	Age > 60 years Obesity (BMI > 30kg/m2) Critical care admission Dehydration Personal or family history of DVTs Nursing home residency

Table 2: Variables used to Calculate Wells Score for risk Assessment of Lower Extremity DVT

Variable	Point
Active cancer – treatment of palliation within 6 months	1
Bedridden for more than 3 days or major surgery within 12 weeks	1
Calf swelling > 3cm compared to other leg and measured 10cm below tibial tuberosity	1
Collateral (non-varicose) superficial veins present	1
Whole leg swelling	1
Localized tenderness along deep veins	1
Unilateral pitting edema	1
Paralysis, paresis, or recent plaster immobilization of LE	1
History of documented DVT	1
Alternative diagnosis to DVT as likely or more likely	-2

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Table 3: Risk Factors and Symptoms of Post-Thrombotic Syndrome (PTS)[24]			
Pathophysiology	Damaged vein valves and venous obstruction result in venous hypertension, which causes a reduction in muscle perfusion, leading to PTS		
Risk Factors	**At time of DVT Diagnosis**	**Related to DVT Treatment**	**Post-DVT Treatment**
	DVT location (proximal > distal) Prior ipsilateral DVT Age > 60 years Obesity (BMI > 30kg/m²) Pre-existing primary venous insufficiency	Anticoagulant choice Subtherapeutic oral anticoagulant	Ipsilateral DVT recurrence Persistent venous symptoms 1 month after DVT event Residual thrombosis as detected on US Persistently elevated D-dimer
Symptoms	• Varies among individuals, but can include: • Chronic pain and swelling of the affected extremity • Fatigue • Sensations of extremity pulling or heaviness • Skin changes: – changes in skin color – Stasis hyperpigmentation – Varicose veins – Telangiectasias – Dermatitis – Skin thickening (lipodermatosclerosis) – Painful and slow-healing ulcers in severe cases		

Table 4. Pros and Cons of Anticoagulants			
Anticoagulant	**Dose**	**Pros**	**Cons**
Unfractionated heparin (i.e Lovenox)	1mg/kg Q12H	- Fast Acting	Available only subQ Expensive
Direct oral anticoagulants	**Apixaban:** 10mg BID for 7 days, followed by 5mg BID daily **Rivaroxaban:** 15mg BID for 21 days, followed by 20mg daily **Dabigatran:** 150mg BID	Can take orally Daily dosing No need INR titration Quick acting	- Expensive
Vitamin K antagonists (i.e. warfarin)	Warfarin dosing is individualized and often follows institutional protocols based on the patient's goal INR	Cheap Can take orally (outpatient friendly)	- Must draw INR frequently - Takes time to titrate to therapeutic dose
Fondaparinux	Weight-based dosing: <50kg: 5mg 50 to 100kg: 7.5mg daily >100kg: 10mg daily	- Fast acting	- Expensive - SubQ
Graduated compression stockings		- Cheap, removable	Uncomfortable, must be correct compression Can still have DVT
Ambulation		Cheap Least invasive	Painful for PTS patients

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Cyber-attacks:
The New "Silent Threat" in Healthcare

By Saige Bree Greenwell, DO

Two community hospitals in the Hudson Valley fell victim to cyber- attacks in late 2023. The perpetrators had access to patients' protected health information from these facilities for nearly two months before the breach was detected and resolved.[1] In response, HealthAlliance Hospital of the Hudson Valley (HAHV), underwent a planned shutdown which required staff to vacate nearly every patient bed on the wards in order to resolve the situation.[1,2] The breach sent shockwaves through the surrounding community and severely damaged public trust in HAHV, a common consequence of data breaches.[3] Patients and even members of hospital staff struggled to understand how something like this could have occurred, because cyber-attacks, though rampant, are rarely discussed openly in the context of healthcare. In fact, "during 2014 – 16, 90% of hospitals and clinics experienced at least one data breach, and 45% experienced at least five data breaches."[3] Some may expect that community hospitals, like HAHV, are more prone to cyber security breaches because they have fewer resources and less security scaffolding when compared to larger facilities, but this assumption is false. Even Boston Children's Hospital, currently ranked second on the Children's Hospital Honor Roll, had to shut down their internet for over two weeks as a result of a cyber-attack in 2014.[3]

These days, technology is integral to day to day operations in medicine. Eighty- eight percent of outpatient doctors use an electronic record,[4] and in light of widespread, lasting changes made to healthcare delivery in response to Covid-19, cyber threats pose tremendous risks to the profession of family medicine. Covid rapidly normalized telehealth care and working from home in family medicine and other specialties, which ingrained technology even more drastically into the daily practice of most physicians.[3] One publication showed that the rates of telehealth utilization grew dramatically during Covid, "[increasing by] 766% in the first 3 months of the pandemic."[5] Alongside an increased reliance on technology in patient care came an increased awareness of our global cybersecurity deficits. Martin Ignatovski, PhD, Chief Information Officer of the online EHR company *SimplePractice*, writes that, "The US healthcare industry saw a 25 percent increase in successful cybersecurity attacks during the pandemic."[6]

The natural question is what can be done to prevent and manage cyber security breaches? At the broadest level, as a profession, we must increase awareness and research. One study found that "Only 17% of the most widely-published studies [about cybersecurity] were included in health journals, the other 83% in engineering journals", suggesting that current levels of attention to cyber security amongst physicians may be low.[3] This assumption is substantiated by survey data collected by Alhuwail, et al. which showed that doctors have poor cyber practices as compared to other healthcare professionals such as nurses and administrators.[7] Given that cyber-attacks are costly, violate patients' right to privacy, and may impact patient safety and outcomes,[3] cyber- attack incidences and effective management strategies need to be shared in order to learn and evolve as a medical community.

At a systems level, the key is to be proactive. Wasserman and Wasserman found that "training staff in cybersecurity principles has been shown to reduce the number of attacks."[3] The research of Saira, et al. found that most cyberthreats occur when employees fall prey to phishing attempts or mishandle data.[8] Thus, facilities can protect themselves from attacks by training their staff, preparing for the breaches, and having policies and disaster planning in place *before* they need to use it. One additional tool in preparation which may be more challenging to address, is funding. Lack of resources is an attributing factor to IT departments, especially in hospitals, being unprepared and fragile.[3] However, considering that in 2020 primary care accounted for <5% of health care expenditure in the United States, it may be difficult to shunt any portion of this limited

funding towards avenues that may not seem explicitly related to patient care.[9] Despite the preexisting lack of funding within family medicine, it is likely that increasing spending on IT prophylactically may save money and better protect patient interests in the long run.

At an individual level, mindfulness is crucial. Before pitching certain electronic or online options to patients, consider whether it is truly the best option or just the convenient one.[3] For instance, a cybercriminal accessing and using data through an online patient portal is much less likely to be detected and stopped than someone trying to steal a paper record from a file cabinet.[3] This is not to say that physical charts do not come with their own set of issues, but it is worth taking the extra time to reflect on alternative options. For example, a phone call or follow up visit may be a safer avenue for discussing lab results than an online message if the patient is not confident in their ability to safely use a portal. Telehealth visits are another such example. When patients are at home, the burden is on them to protect themselves from invisible threats. Just as hospital employees may not recognize phishing endeavors, patients may also be unable to see through cleverly disguised faux emails or messages, a method which cyber criminals have used with increasing frequency since the Covid pandemic.[10] Additionally, patients may be using insufficient security measures such as inadequately complex passwords, or they may physically lose a device which provides easy access to their healthcare-related accounts.[3] Just as physicians may anticipate medication adjustments for patients' dietary lapses around the holidays or prophylactic vaccine needs when they travel internationally, we should encourage patients to use preventative security measures by requiring two-factor authentication, suggesting timely software updates, and ensuring patients have a safe and private location to participate in virtual visits where no potentially dangerous passers-by can potentially listen in.[11]

To help prevent patients from getting scammed, providers should give specific instructions when discussing or scheduling telehealth appointments. Whenever possible, this should include specific dates/times to expect communications from the office as well as a list of safe phone numbers and email addresses that patients can trust.[11] Additionally, as with any patient encounter, providing good patient education and answering any questions they may have can go a long way in making them feel comfortable and capable of taking the right steps.[11] Providers can find numerous and specific instructions for a variety of ways to protect patients and themselves when it comes to using technology in practice directly on the AMA website.[12] Easy, quick answers to questions about anything from the legal responsibilities required by HIPAA, to how to assess potential areas of vulnerability, to Covid-specific technology changes can be found there.[12] Additionally, the National Institute of Standards and Technology's Computer Security Resource Center recently published a comprehensive list of other resources with embedded hyperlinks included, which can be found here: https://csrc.nist.gov/files/pubs/sp/800/66/r2/final/docs/sp800-66r2-cybersecurity-resources.pdf .[13]

At this time, all healthcare workers need to be prepared to face situations related to cyber security and breaches. Technology and its associated threats can, and likely will, impact all providers at some point given the expanding level of reliance on computers and software in healthcare and the fact that such breaches have been steadily increasing annually for a number of years.[14] Specific steps must be taken at both systems and individual levels to prevent and curtail cyber-attacks in order to limit dangers to patients and their privacy. A number of resources are already in existence and easily accessible to help hospitals and providers. Like hypertension and cardiac disease, cyber-attacks have become a prevalent "silent threat" in medicine that physicians must be diligent in addressing.

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Incidental Findings of Hepatic Steatosis or Cirrhosis on Imaging

By Julia Cooper, MD, AAHIVS

Case: You begin a week of rounding at the hospital and recognize one of the admitted patients - a medically complicated 62-year-old woman with advanced COPD (on 2L O2 at baseline), paroxysmal atrial fibrillation and flutter (on rivaroxaban), type 2 diabetes (on insulin), well controlled HIV (on tenofovir alafenamide / emtricitabine and dolutegravir), and a colostomy which was created after an episode of bleeding diverticulitis. Her colostomy was not intended to be permanent, but due to her comorbidities, she has never pursued a reversal, and has since developed a large parastomal hernia. While she is hospitalized for COPD exacerbation, a CT scan is obtained overnight for severe abdominal pain. The scan rules out ischemia or incarceration of the hernia. However, the radiologist incidentally notes "The liver is enlarged with nodular contour. Moderate splenomegaly." You note with confusion that this patient has had a prior abdominal ultrasound with similar findings, and has very mild thrombocytopenia, but there is no mention of liver disease in her problem list.

Questions: Do you have adequate information to diagnose cirrhosis? What risk factors does this patient have for cirrhosis? What workup can you offer her? What can be done to reduce her risk of complications?

Epidemiology and etiologies of cirrhosis: The majority of patients with cirrhosis are unaware of their diagnosis,[1] and most remain asymptomatic until decompensation occurs, by which time they have missed the opportunity to improve or stabilize their liver health. As metabolic dysfunction-associated steatotic liver disease (MASLD) [formerly known as "non-alcoholic fatty liver disease" (NAFLD), but recently renamed by AASLD[2]] surpasses hepatitis C and alcohol use disorder as the leading cause of cirrhosis,[3] it becomes increasingly important to be vigilant for evidence of hepatic fibrosis and cirrhosis in patients who lack "traditional" risk factors like alcohol or drug use. It is also important to recognize that concomitant sources of inflammation such as diabetes plus hepatitis C, or heavy alcohol use plus HIV, can synergistically contribute to fibrosis and cirrhosis.[2] Regardless of etiology, liver diseases tend to progress along the same continuum, from fatty infiltration or *steatosis*, to the inflammatory phase *steatohepatitis*, to scarring or *fibrosis* and, eventually, cirrhosis.[2]

In June 2023, an international panel of experts renamed "NAFLD" to MASLD (metabolic dysfunction associated steatotic liver disease) and MetALD (a subset of steatotic liver disease in patients with both metabolic dysfunction and heavy alcohol use). "NASH" is now MASH (metabolic dysfunction associated steatohepatitis). Read more at https://www.aasld.org/new-masld-nomenclature.]

Diagnostic tests for hepatic steatosis, fibrosis and cirrhosis: Unfortunately, both liver enzymes and imaging can be deceptively normal at any stage of hepatic steatosis, fibrosis or in compensated cirrhosis.[1] Even noninvasive calculators such as the "NAFLD fibrosis score" or the FIB-4 may underestimate fibrosis in its early stage. True "screening," at least for early disease, is therefore limited. What fibrosis scores can do is to help rule out advanced fibrosis, and when fibrosis cannot be ruled out, the best confirmatory test for both fibrosis and steatosis, without the risks of biopsy, is vibration-controlled transient elastography (VCTE or FibroScan).[4] VCTE is similar to ultrasound in terms of patient experience – it is done by holding a vibrating probe over the right upper quadrant – but its output is a measurement of liver stiffness, not a radiographic image. The AASLD in 2023 released a new recommendation that patients with diabetes, obesity with other features of metabolic syndrome, or a family history of metabolic-associated liver disease be screened for fibrosis[5] with fibrosis scores and then, if abnormal, with transient elastography. They also recommend this MASLD workup for patients with abnormal liver enzymes, incidental steatosis on imaging, or other clinical suspicion for metabolic-associated liver disease, including in patients with concomitant alcohol use. Unfortunately, access to transient elastography is still limited. [For interested readers: training in transient elastography as a family physician is not difficult, but insurance reimbursement presents a hurdle – or perhaps an opportunity for advocacy.]

On the other hand, hospitalized patients are highly likely to undergo abdominal imaging, with incidence of diagnostic abdominal CT increasing steadily.[6] And although the sensitivity of US, CT or MRI for hepatic steatosis and fibrosis are not excellent, hepatic steatosis and cirrhosis are still the *most common* incidental parenchymal findings on ultrasound and CT.[7] Because hepatic fibrosis is difficult to screen for and easily slips under the radar in primary care, the hospitalist has a critically important opportunity to notice radiographic evidence of undiagnosed liver disease and to facilitate workup.

Although cirrhosis is not definitively diagnosed on imaging, the American College of Radiology (ACR) lists several suggestive characteristics on ultrasound or CT, including increased caudate-to-right lobe ratio, nodular surface contour, presence of abdominal varices, ascites, portal vein dilation >13mm, and/or presence of splenomegaly. Surface nodularity in particular has a high specificity for cirrhosis.[8] When multiple radiographic features are present, especially if they can be corroborated by laboratory findings (Table 1a and 1b), a working clinical diagnosis of cirrhosis is appropriate. Physical exam findings such as distal erythema, "Terry nails," spider angiomata, or findings of portal hypertension such as caput medusa, splenomegaly and ascites, are similarly nondiagnostic but can corroborate a radiographic impression of cirrhosis. Recall that the cirrhotic liver may be enlarged *or* shrunken.

Table 1a. Laboratory abnormalities which can support a diagnosis of cirrhosis

Laboratory values	Relevance to cirrhosis
INR	In cirrhosis, elevated INR reflects impaired synthetic function of the liver (but INR may also be elevated by medications or by hypovitaminosis K, e.g. due to malabsorption or during broad-spectrum antibiotic use)[9]
Thrombocytopenia	In cirrhosis, portal hypertension leads to hypersplenism which leads to increased destruction of platelets; impaired synthetic function can also decrease hepatic production of thrombopoietin[10] (in addition to broad ddx of thrombocytopenia)
Hypoalbuminemia	In cirrhosis, low albumin reflects impaired synthetic function of the liver (but albumin may also be lost via nephrotic renal disease)

Recommended workup for incidentally diagnosed liver disease: Any finding of hepatic steatosis, fibrosis or cirrhosis should trigger a workup. Although it is not urgent to determine the etiology during the hospitalization, it is of critical importance to communicate the findings to the PCP, since many etiologies can be improved or stabilized with treatment and since patients deserve education about this potentially life-threatening diagnosis. The degree of fibrosis can be approximated by fibrosis scores and confirmed by outpatient transient elastography or liver biopsy. If there is adequate evidence to make a clinical diagnosis of cirrhosis, elastography is not necessary, but biopsy may still be indicated in cases of uncertain etiology.

Case conclusion: This patient's imaging findings are indeed sufficient to give her a clinical diagnosis of cirrhosis, which was communicated both to her and to her PCP. Her inpatient team did not complete her workup during the hospitalization, but her medication list was reviewed for anything requiring a hepatic dose adjustment.

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Table 1b. Fibrosis scores calculated from lab results which can support a diagnosis of cirrhosis or advanced fibrosis. If any/all of calculated fibrosis scores are indeterminate, a more definitive measurement such as transient elastography or biopsy should be considered. Note that none of these scores are well studied in alcohol-related liver disease.

Fibrosis scores	Components	Interpretation and sensitivity/specificity	Best used for
APRI (AST to Platelet Ratio Index)	-AST -Platelet count	Low (<0.5) has good negative predictive value against cirrhosis. Upper cutoff varies in PPV for fibrosis or cirrhosis based on etiology[11]	In patients with HCV, HBV or MASLD, APRI and FIB-4 can be used together to rule cirrhosis in or out if the scores are very high or low. They are not useful for fibrosis staging.
FIB-4	-AST -ALT -Platelet count -Age	FIB-4 < 1.45 has negative predictive value of 90% for advanced fibrosis. FIB-4 > 3.25 has good (but slightly varying) positive predictive value for advanced fibrosis or cirrhosis in HCV, HBV or MASLD.[1,12]	
NAFLD fibrosis score	-AST -ALT -Platelet count -BMI -Fasting glucose, A1c or glucose tolerance (normal/abnormal) -Albumin -BMI -Age	< -1.455: F0-F2 (no, mild, or moderate fibrosis) -1.455 – 0.675: Indeterminant score > 0.675: F3-F4 (advanced fibrosis to cirrhosis)	NAFLD fibrosis score considers metabolic factors such as glucose tolerance. It is useful in patients with metabolic syndrome, both for estimating fibrosis stage in patients with hepatic steatosis and for corroborating a suspicion of cirrhosis.
Fibrosure, Fibrometer	Varies by test. Calculation is proprietary.		These tests are validated for fibrosis staging in hepatitis C specifically.

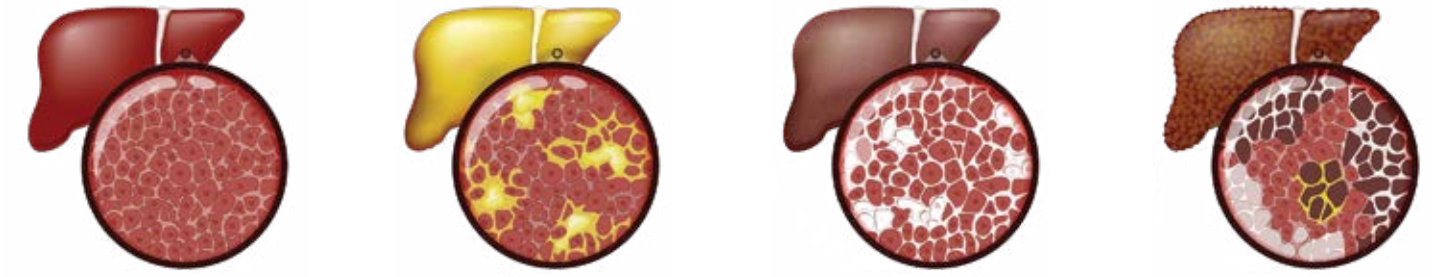
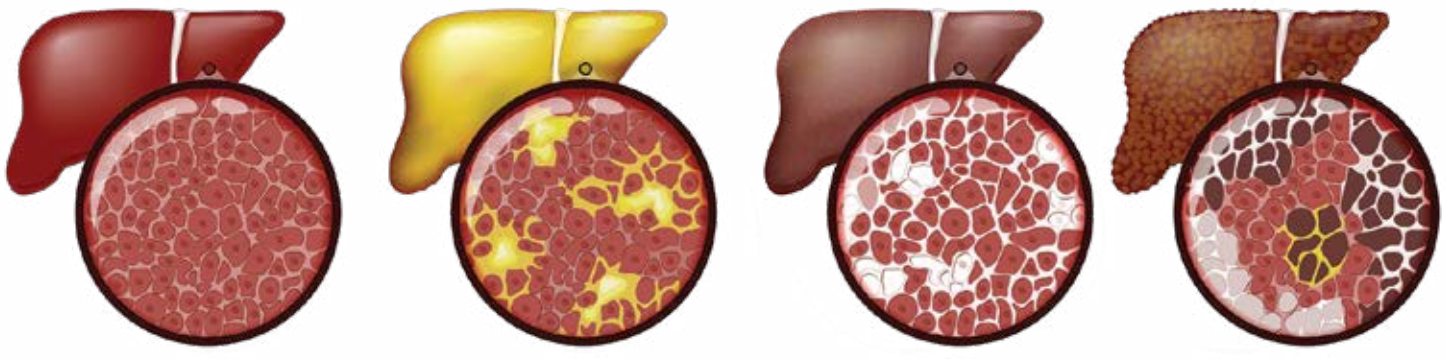


Table 2. Determining the etiology of incidentally discovered steatosis, fibrosis or cirrhosis. Tests in the left column can be collected at a single visit to rule out common causes.

Recommended diagnostic tests	Results and their interpretations	Management
Ruling out viral hepatitis		
HCV antibody with reflex HCV viral load	Reactive antibody and negative VL – **resolved HCV**	Update problem list and re-screen periodically if there are ongoing risk factors.
	Detectable VL – **active HCV**	Ensure follow-up with a PCP who treats HCV or with hepatology.
Hepatitis B surface antibody, surface antigen, and core antibody	Negative antibodies and antigen - unvaccinated and uninfected	Vaccinate
	Positive surface antibody and core antibody, with negative antigen – **previously infected and now under immune control**	Ensure that resolved hep B infection and cirrhosis are in the patient's problem list to alert providers to low, persistent risk of reactivation.
	Positive surface antigen - **active HBV infection**; obtain HBV DNA level	Ensure follow-up with a PCP who is comfortable with HBV management, or hepatologist.
	Solely core antibody positive – could be resolved HBV or occult HBV infection with waned antigen production. False positives do also occur. (Lone core antibody positivity is also seen in HBV window period, but acute HBV would not cause radiographic findings of fibrosis or cirrhosis.)	Given that this is a high-risk patient with liver damage on imaging, HBV antibodies and antigen and/or DNA should be retested in 2 months.
HIV screening	Positive antibody / antigen or positive HIV viral load – **HIV infection**	HIV can compound the inflammatory effects of viral hepatitis or metabolic liver disease. Ensure follow-up with a PCP who manages HIV or with ID.
	Negative HIV screening	If HIV-negative but with ongoing risk factors, ensure follow-up with a PCP who can provide PrEP. Note that all three approved PrEP regimens can be used in cirrhosis, but in severe disease (Child Turcott Pugh C), Descovy (TAF/FTC) and Apretude (LA-CAB) are unstudied.
Ruling out autoimmune liver disease		
Antinuclear antibody	ANA or anti-smooth muscle antibodies > 1:80 may suggest **autoimmune hepatitis**; can order total IgG for further evidence	Refer to hepatology May require biopsy to confirm diagnosis
Anti-smooth muscle antibody	Anti-mitochondrial antibody is most associated with **primary biliary cirrhosis**	
Anti-mitochondrial antibody	ANA, other antibodies, p-ANCA, and alkaline phosphatase may be positive/elevated in **primary sclerosing cholangitis**	

Table 2, *continued*. Determining the etiology of incidentally discovered steatosis, fibrosis or cirrhosis. Tests in the left column can be collected at a single visit to rule out common causes.		
Ruling out inborn metabolic abnormalities		
Iron studies (ferritin, iron level, total iron binding capacity) Transferrin saturation = [iron x 100]/TIBC	If ferritin >250 ng/mL in men, or >200 ng/mL in women, or if transferrin saturation >45%, order human **hemochromatosis** protein gene mutation analysis Recall that ferritin is an acute phase reactant and may be elevated by other inflammation	Refer to hematology
Optional, based on clinical suspicion: Alpha-1 anti-trypsin activity assay	Abnormal α1AT activity, especially with concomitant emphysema, suggests **alpha-1 anti-trypsin deficiency**	Refer to hepatology
Optional, based on clinical suspicion: ceruloplasmin	Low ceruloplasmin suggests **Wilson's disease**	Confirmatory testing can include serum copper level, urinary copper excretion, liver biopsy, and genetic testing. Refer to hepatology or genetics.
Assessing for other factors		
Liver enzymes GGT Thorough alcohol history	AST elevated to twice the level of the ALT is suggestive of **alcohol-induced injury**; greater than 3x even more so. GGT may also be elevated in alcoholic liver disease. The AASLD suggests diagnosing "MetALD" instead of pure "MASLD" if the patient consumes 140-350 g alcohol/week (20-50 g/day) for women or 210-420 g/week (30-60 g/day) for men. However, recall that alcohol-induced liver disease can occur in non-daily or "binge" drinkers, and in patients who do not experience dependence or withdrawal.	Encourage abstinence from alcohol (for any patient with liver disease, but especially those with alcohol-induced damage). Offer treatment if applicable for alcohol use disorder. Recall that cirrhosis is not a contraindication to treatment, including treatment with naltrexone if the liver enzymes are below five times normal.
Routine metabolic tests (fasting glucose, A1c, lipid panel)	Patients with concomitant insulin resistance, hyperlipidemia (especially hyperTG), HTN or other features of metabolic syndrome are more likely to have a component of **metabolic dysfunction associated steatotic liver disease or MASLD**. MASLD can affect lean patients. MASLD can contribute to liver disease even if a second cause (like alcohol use or viral hepatitis) also exists. Only a liver biopsy can definitively diagnose metabolic dysfunction-associated steatohepatitis (MASH), but clinicians who suspect MASLD can recommend the relevant lifestyle changes, which are universally healthy.	In diabetic patients, glycemic control is critical, especially with pioglitazone or GLP1 agonists if possible, since these may stabilize or improve steatosis and fibrosis.[13,14] Dietary changes should focus on reducing processed carbs and red meats in favor of more plant-based foods and whole grains. Even 5-10% weight loss can help, but healthy dietary changes are beneficial irrespective of weight loss, therefore patients should not feel pressured to reach a target weight.



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This patient's main risk factors for cirrhosis are her diabetes and HIV infection, which contribute synergistically to her risk of metabolic steatotic liver disease or MASLD. As a person living with HIV, she fortunately had recent testing for hepatitis B and C and was negative for both. Her history with alcohol was reviewed, and although she rarely drinks alcohol, she was counseled to abstain completely. Autoimmune hepatitis and hemochromatosis were ruled out (although it should be noted that a patient with multiple inflammatory conditions and with COPD is likely to have a slightly elevated ferritin and possibly a low-positive ANA; these tests must be interpreted thoughtfully). Her severe COPD also made alpha-1 anti-trypsin deficiency a consideration, but she had a normal result. Because she had multiple radiographic and laboratory findings of cirrhosis, there was no need for confirmation with transient elastography. See Table 2 for the recommended workup in step-by-step form.

Going forward, she should undergo liver ultrasound every 6 months for hepatocellular carcinoma screening, and should have an endoscopy to screen for varices. She should be caught up on tetanus, pneumonia and hepatitis A&B vaccines as needed. Her PCP should monitor her for signs of decompensation such as ascites, hepatic encephalopathy, or jaundice. Her MELD score should be calculated periodically; many patients with cirrhosis can be managed by their PCP (and endoscopist, if applicable), but when the MELD score reaches 15 (or sooner if the patient experiences complications), the patient should be introduced to a hepatologist for transplant evaluation and advanced management.[1] MELD scores greater than 17 can be helpful for prognostication.

Receiving an unexpected diagnosis of cirrhosis, especially based on incidental imaging findings, can be bewildering. However, it is a much easier discussion to have before a patient experiences decompensation. For this patient, understanding her liver disease played a role in her decisions about insomnia treatment, the surgical risk of having her hernia repaired, and her advanced directive.

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Did Hospitals Kill the House Call?

By Thomas C. Rosenthal, MD

Daniel Chapin, MD (1761-1821) was a Yale Medical College graduate practicing in Buffalo, New York in 1821. He did not like to ride horseback and was known to walk miles to make home visits with a pipe clenched firmly in his teeth. Patients recalled seeing their doctor arrive with a rifle resting on his shoulder, a dog trotting at his feet, a plume of smoke circling his cap, and a large leather bag over his shoulder. He was sixty in the winter of 1821 when he was summoned to visit a feverish child with measles. The child was seizing when Dr. Chapin arrived and the family struggled to understand what the doctor could not explain, but he was certain seizures meant inflammation of the brain. As he feared, the little girl did not survive. The time for hand holding, hugging and sharing tears had exhausted itself when Doc Chapin left for home in the darkest hours of night. Somewhere along his hike, the sleet turned to a windblown snow and got the best of him. Ole Doc Chapin died of exposure, wanting little more that morning than to have breakfast with his wife.[1]

Most doctor visits for acute care happened in the home. Nineteenth-century hospitals were places of last resort for the poor, itinerant laborers, and sailors and who had no family to care for them. The earliest hospitals evolved from almshouses that were plagued by contagions little understood at the time. At best, they provided forced rest and nourishment, but few cures.

If you had a fever and could not muster the strength to get to the outhouse, you wouldn't risk falling off a horse going to the doctor. After every home remedy had failed, a messenger was sent to fetch the family doctor. As late as the 1920s, if you had an accident, you were taken home to recover and wait for the doctor to show up. Your chances were just as good at home as in a hospital bed where you risked adding a hospital fever to your problems. Armed with a smile, a hat and his leather bag, the family doctor would treat you for everything from a broken arm to a case of influenza.[2] Night calls were so common that many doctors had a pipe installed from their front door to the bedroom so dispatched messengers could holler in it, saving broken glass caused by stones being thrown at an upstairs window.

But the 1870s and 1880s saw an understanding of germ theory and by the 1930s procedures that might have occurred on the kitchen table were best done in a sterile operating room. Hospitals became a source for antibiotics, imaging, and complex medical processes. Then, in 1946, the Hill-Burton Act funded construction of over 6,800 hospitals nationwide.

The hospital's emergency room became the go-to place for a broad range of services and was obligated by law to provide care to patients regardless of health insurance. People in crisis found the 24/7/365 ER to be a reliable safety net for all kinds of conditions. The more complicated and stressed our health system became, the more people relied on the hospital, and the more difficult it was to remember a time when Americans avoided hospitals at all cost. Very sick patients may get triaged to immediate attention, but one could argue that less acute patients can spend hours in contagion sharing waiting rooms. Still, patients keep going to the hospital because real scientific progress has cemented the connection between rapid medical treatment and chances of survival.

Hospitals facilitated and encouraged specialization. Physicians became highly skilled at performing a narrow range of services. Today, many specialists require the hospital setting to maximize their skills and, in turn, they have become reliant on the range of other specialists to provide all the services a patient may need. This knowledge concentration, so essential in modern, high technology hospitals, means fewer doctors can handle a broad scope of illness. It also means fewer doctors who can handle house-calls and explains why the modern medical student considers home visits a quaint idea.

House calls dropped from 40% of physician encounters in 1930 to 10% by 1950 and less than 1% by 1980.[3] But it came with a huge financial burden.

Physicians who make house calls now are almost exclusively practicing in a primary care field where a talent for comprehensive care is valued. A survey of Virginia Medicaid providers found that physicians who make routine house calls were most likely to be family physicians. They are women and men who collaborate with home health agencies and consider patient transportation difficulties, chronic disease, end-of-life care, death pronouncement, and their own personal enjoyment as indications.[3] Besides performing a clinical assessment, house calls may involve

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observing the patient performing daily activities, reconciling medication discrepancies, and evaluating home safety.[4] Today, many hospice programs are well accepted examples of the value of physician home visits.

Home-based primary care programs in Veteran Affairs Medical Centers, and several other programs begun since the passage of the Affordable Care Act, have proved that house calls can lower costs and keep patients out of emergency rooms. Home visit programs built as part of a primary care practice may eliminate barriers, particularly for disabled and chronically ill patients. These programs build in principles of continuity, chronic disease management, patient self-care, and care-giver relationships that become critical to their success.[4]

The need for physician house calls will grow as the US population ages. In 1998, Medicare established new billing codes that increased reimbursements for physician house calls by nearly 50%. Medicare reimbursements for physician visits to homes and domiciliary facilities are now over $213 for complex new patient visits, and the value of the most commonly used code rose to over $100.[3] Because today's visits involve chronic disease management, they can be scheduled in advance and planned by neighborhoods to minimize driving time.[4]

So, have hospitals killed the home visit? What we can say is that medicine has evolved into two domains. One is the 'intensely acute' domain, which includes much of the high technology capable of saving lives in crisis. The second is the 'personal care' branch that returns the longer-term commitment of relationships and continuity to medicine. The indications for a house call for acute care have waned. But house calls for ongoing personal care remain essential in a well-balanced health system.

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Upcoming Events

2024

May 11 (Open virtually)
May 18-19, 2024
Reconvene Congress of Delegates
Albany

July 27-28, 2024
Summer Cluster
Troy
Hilton Garden Inn

November 3, 2024
Fall Cluster
(Board meeting only commissions meet virtually)
Hilton Garden Inn
Albany Medical Center

Winter Weekend
January 23-26, 2025
Lake Placid, details TBA

For updates or registration information for these events go to www.nysafp.org

Updates in Management of Newborn Hyperbilirubinemia

By Surabhi Aggarwal, MD and Lovedhi Aggarwal, MD

Overview

Hyperbilirubinemia is a prevalent condition in neonates, especially within the first week of life.[1] It affects 8-11% of newborns, with 60-80% developing idiopathic neonatal jaundice, characterized by a yellowish discoloration of the skin and sclera due to elevated bilirubin levels. Hyperbilirubinemia is defined as a total serum bilirubin level exceeding the 95th percentile for age in the first week of life.[2,3] Although rare, acute bilirubin encephalopathy and kernicterus can have devastating effects on patients and their families.[4] Therefore, careful monitoring and treatment of neonates affected by hyperbilirubinemia are crucial to mitigate these risks.

The American Academy of Pediatrics (AAP) initially published clinical guidelines in 2004 for infants ≥35 weeks' gestation and an updated commentary was issued in 2009. In 2022, AAP published revised guidelines for treatment of hyperbilirubinemia aimed at preventing both overtreatment and the rare necessity of escalating care to an exchange transfusion.[5,6] Changes included an increased threshold for initiating phototherapy treatment and exchange transfusion, varying based on gestational age. For monitoring, emphasis was placed on the importance of evaluating for hemolysis both prenatally and postnatally, including conducting a direct antiglobulin test in infants born to mothers with either unknown or O blood type. A recommendation was also provided to check rebound bilirubin based on age at onset of phototherapy and risk factors such as hemolysis, and to wait at least 12-24 hours before obtaining a level particularly for low-risk infants. New guidelines also placed importance on advocating for breastfeeding and enteral supplementation over intravenous fluids (IV) unless the infant meets specific criteria for the escalation of care.[5] Changes were also made in the post-discharge follow-up recommendations. As many family physicians encounter this common neonatal condition in the inpatient setting and outpatient follow up, we aim to provide a brief summary of the updated guidelines in this article.

History of the Guidelines

AAP initially published clinical guidelines in 2004 for infants ≥35 weeks' gestation. This gestational age range included most newborn infants who were cared for and followed by general pediatricians and family physicians on well newborn services. In 2009, a commentary describing several clarifications and modifications[7,4] to the 2004 clinical practice guideline was published. This included clarifying the distinction between "hyperbilirubinemia risk factors," which increase the risk of subsequent hyperbilirubinemia, and "hyperbilirubinemia neurotoxicity risk factors," which increase the risk of bilirubin neurotoxicity. A new recommendation was made for universal predischarge bilirubin screening with measures of total serum bilirubin (TSB) or transcutaneous bilirubin (TcB) linked to specific recommendations for follow-up.

Changes under the 2022 AAP guidelines

1) Prevention and early detection of at-risk infants for hyperbilirubinemia:

A. Evaluation of hyperbilirubinemia – All infants require visual assessment of jaundice every 12 hours after birth and laboratory assessment with total serum bilirubin (TSB) or transcutaneous bilirubin (TCB) at 24-48 hours after birth. A rapid rate of increase (≥0.3 mg/dL per hour in the first 24 hours or ≥0.2 mg/dL per hour thereafter) is unusual[10] and suggests hemolysis, prompting a direct antiglobulin test (DAT), or Coombs test to be performed. It is among the most widely used assays in laboratory medicine, first described over 70 years ago and introduced by Robin Coombs in 1945.

B. Risk factors clarification – Infants with risk factors (see Table 1) require more careful monitoring. Risk factor determination can be made through infants' examination, laboratory values, and family history. The presence of neurotoxicity risk factors (see Table 2) further lowers the threshold for phototherapy treatment. Neurotoxicity risk factors are modified with newer guidelines to assist physicians in clinical assessment (Table 3). Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a significant cause of hyperbilirubinemia leading to kernicterus in the United States.[14,15] In New York State, diagnostic testing is mandatory for infants at high risk of G6PD deficiency.[11] It is an X-linked disorder that is challenging to identify because sometimes there is no family history, but genetic ancestry from certain populations (e.g., Sub-Saharan Africa, Middle East, Mediterranean, Arabian Peninsula, and Southeast Asia) can aid in predicting risk.

C. Clarifying direct antiglobulin test (DAT) negative status – DAT helps identify infants at risk for hyperbilirubinemia attributable to hemolysis. However, infants with a DAT-positive status due to maternal RhIG (Rh immunoglobulin) status can be considered at no risk for hemolysis.[16]

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TABLE 1: Risk Factors for Developing Significant Hyperbilirubinemia – Adapted from Kemper et al, Pediatrics, 2022

- Lower gestational age (ie risk increases with each additional week less than 40 wk)
- Jaundice in the first 24 h after birth
- Predischarge transcutaneous bilirubin (TcB) or total serum bilirubin (TSB) concentration close to the phototherapy threshold
- Hemolysis from any cause, if known or suspected based on a rapid rate of increase in the TSB or TcB of >0.3 mg/dl per hour in the first 24 h or >0.2 mg/dl per hour thereafter.
- Phototherapy before discharge
- Parent or sibling requiring phototherapy or exchange transfusion
- Family history or genetic ancestry suggestive of inherited red blood cell disorders, including glucose-6-phosphate dehydrogenase (G6PD) deficiency
- Exclusive breastfeeding with suboptimal intake
- Scalp hematoma or significant bruising
- Down syndrome
- Macrosomic infant of a diabetic mother

TABLE 2: Hyperbilirubinemia Neurotoxicity Risk Factors – Adapted from Kemper et al, Pediatrics, 2022

- Gestational age <38 wk and this risk increases with the degree of prematurity
- Albumin <3.0 g/dl
- Isoimmune hemolytic disease (i§, positive direct antiglobulin test), G6PD deficiency, or other hemolytic conditions
- Sepsis
- Significant clinical instability in the previous 24 h

a Gestational age is required to identify the phototherapy thresholds (Figs 2 and 3; Supplemental Tables 1 and 2, and Supplemental Figs 1 and 2) and the exchange transfusion thresholds (Figs 5 and 6; Supplemental Tables 3 and 4, and Supplemental Figs 3 and 4).

Table 3

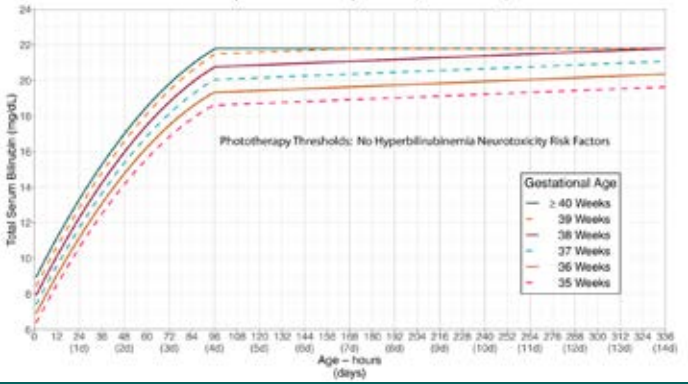
2004AAP	2022AAP
• Hemolytic disease	• Hemolytic disease
• Asphyxia • Significant lethargy • Temperature instability • Acidosis	• Significant clinical instability in the previous 24 hours
• Sepsis	• Sepsis
• Albumin< 3.0 g/Ql	• Albumin< 3.0 g/Ql

D. Raised phototherapy and exchange transfusion thresholds- Please refer to https://www.bilitool.org/ for updated phototherapy thresholds.

New thresholds graphs for phototherapy initiation and escalation of care have been formulated based on expert opinion. They consider gestational age, the hour-specific total serum bilirubin (TSB), and bilirubin neurotoxicity risk factors (see Figures 1-4).

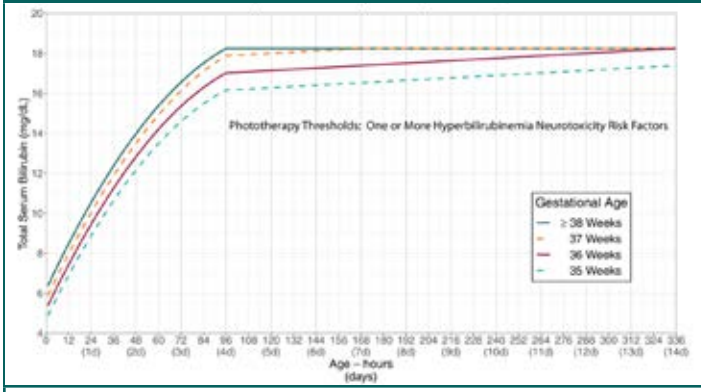
The goal of phototherapy is to prevent an increase in TSB to a level that would warrant escalation of care to exchange transfusion, rather than aiming to prevent subtle neurodevelopmental outcomes. The recommended treatment levels for hyperbilirubinemia are well below overt neurotoxicity or kernicterus levels.[12,13,17,18]

FIGURE 1- Adapted from Kemper et al, Pediatrics, 2022



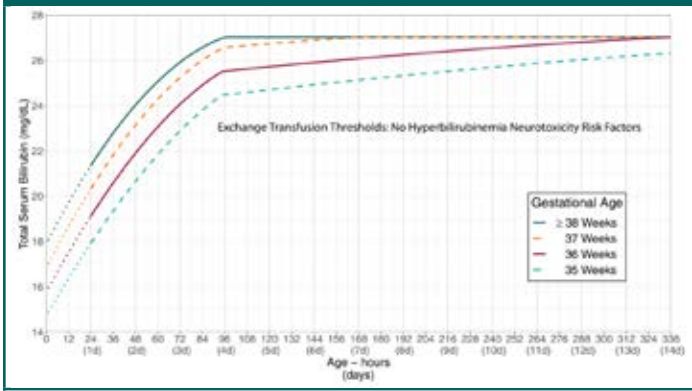
Phototherapy thresholds by gestational age and age in hours for infants with no recognized hyperbilirubinemia neurotoxicity risk factors other than gestational age. These thresholds are based on expert opinion rather than strong evidence on when the potential benefits of phototherapy exceed its potential harms. Use total serum bilirubin concentrations; do not subtract direct-reacting or conjugated bilirubin from the total serum bilirubin. In rare cases of severe hyperbilirubinemia in which the direct-reacting or conjugated bilirubin exceeds 50% of the TSB, consult an expert. Note that infants <24 hours old with a TSB at or above the phototherapy threshold are likely to have a hemolytic process and should be evaluated for hemolytic disease.

FIGURE 2 - Adapted from Kemper et al, Pediatrics, 2022



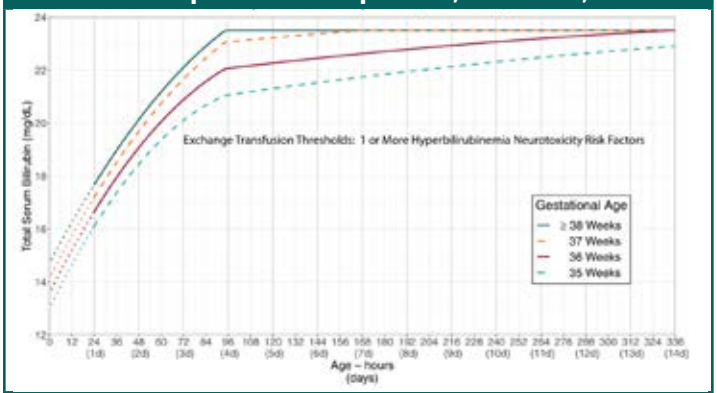
Phototherapy thresholds by gestational age and age in hours for infants with any recognized hyperbilirubinemia neurotoxicity risk factors other than gestational age. These thresholds are based on expert opinion rather than strong evidence on when the potential benefits of phototherapy exceed its potential harms. Use total serum bilirubin concentrations; do not subtract the direct-reacting or conjugated bilirubin from the total serum bilirubin. In rare cases of severe hyperbilirubinemia in which the direct-reacting or conjugated bilirubin exceeds 50% of the TSB, consult an expert

FIGURE 3 - Adapted from Kemper et al, Pediatrics, 2022



Exchange transfusion thresholds by gestational age for infants with no recognized hyperbilirubinemia neurotoxicity risk factors other than gestational age. These thresholds are based on expert opinion rather than strong evidence on when the potential benefits of escalation of care exceed its potential harms. The stippled lines for the first 24 hours indicate uncertainty because of the wide range of clinical circumstances and responses to intensive phototherapy. Use total serum bilirubin concentrations; do not subtract direct bilirubin from the total serum bilirubin. In rare cases of severe hyperbilirubinemia in which the direct-reacting or conjugated bilirubin exceeds 50% of the TSB, consult an expert.

FIGURE 4 - Adapted from Kemper et al, Pediatrics, 2022



Exchange transfusion thresholds by gestational age for infants with any recognized hyperbilirubinemia neurotoxicity risk factors other than gestational age.These thresholds are based on expert opinion rather than strong evidence on when the potential benefits of escalation of care exceed its potential harms. The stippled lines for the first 24 hours indicate uncertainty because of the wide range of clinical circumstances and responses to intensive phototherapy. Use total serum bilirubin concentrations; do not subtract direct bilirubin from the total serum bilirubin. In rare cases of severe hyperbilirubinemia in which the direct-reacting or conjugated bilirubin exceeds 50% of the TSB, consult an expert.

2) Phototherapy discontinuation thresholds:

New guidelines recommend discontinuing phototherapy if the total serum bilirubin (TSB) has decreased by at least 2 mg/dL below the hour-specific threshold at the initiation of phototherapy. However, if there are risk factors for rebound hyperbilirubinemia (e.g., gestational age <38 weeks, age <48 hours at the start of phototherapy, hemolytic disease), a longer duration of treatment should be considered.[8,9]

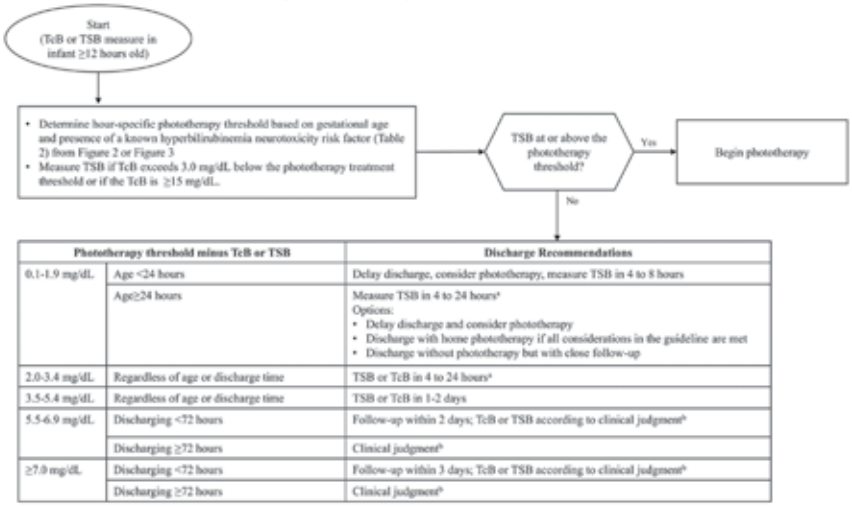
3) Rebound TSB testing after discontinuation of phototherapy:

The timing of follow-up bilirubin testing after discontinuation of phototherapy should be at least 12-24 hours later and based on risk of rebound hyperbilirubinemia (Refer to Table 1). Sufficient time should be allowed for bilirubin concentration to demonstrate the presence or absence of rebound hyperbilirubinemia.[19] Rebound hyperbilirubinemia should be treated with the previous recommendations for initiation of phototherapy.

4) Post discharge follow up:

New guidelines recommend using the difference between the TSB and the phototherapy threshold at the time of measurement to determine the interval between discharge and follow-up and the need for additional TSB or TcB measurements (Figure 5). Both gestational age and hyperbilirubinemia neurotoxicity risk factors are used in this decision, as opposed to previous guidelines which used Bhutani nomograms based on postnatal age in hours and TSB.

FIGURE 5 - Adapted from Kemper et al, Pediatrics, 2022



Phototherapy threshold minus TcB or TSB		Discharge Recommendations
0.1-1.9 mg/dL	Age <24 hours	Delay discharge, consider phototherapy, measure TSB in 4 to 8 hours
	Age≥24 hours	Measure TSB in 4 to 24 hours[a] Options: • Delay discharge and consider phototherapy • Discharge with home phototherapy if all considerations in the guideline are met • Discharge without phototherapy but with close follow-up
2.0-3.4 mg/dL	Regardless of age or discharge time	TSB or TcB in 4 to 24 hours[a]
3.5-5.4 mg/dL	Regardless of age or discharge time	TSB or TcB in 1-2 days
5.5-6.9 mg/dL	Discharging <72 hours	Follow-up within 2 days; TcB or TSB according to clinical judgment[b]
	Discharging ≥72 hours	Clinical judgment[b]
≥7.0 mg/dL	Discharging <72 hours	Follow-up within 3 days; TcB or TSB according to clinical judgment[b]
	Discharging ≥72 hours	Clinical judgment[b]

Flow diagram for infants during the birth hospitalization to determine postdischarge follow-up for infants who have not received phototherapy. [a]Use clinical judgment and shared decision making to determine when to repeat the bilirubin measure within this 4 to 24 hour time window.
[b]Clinical judgment decisions should include physical examination, the presence of risk factors for the development of hyperbilirubinemia or hyperbilirubinemia neurotoxicity risk factors, feeding adequacy, weight trajectory, and family support.

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5) Home phototherapy breast-feeding associated jaundice:

Home LED-based phototherapy is an option for some infants who develop hyperbilirubinemia instead of being readmitted to the hospital.[20,21] Low-risk infants meeting the following criteria can be considered for home phototherapy: gestational age ≥38 weeks, age ≥48 hours, clinically well with adequate feeding, absence of known neurotoxicity risk factors (refer to table 2), no history of previous phototherapy, and total serum bilirubin (TSB) concentration no more than 1 mg/dL above the treatment threshold (refer to Figure 1).

6) Breast-feeding associated jaundice:

There is an increased association of jaundice in exclusively breast-fed infants. There can be two types of jaundice in breast-fed infants and distinguishing them is important to guide management.

A. Breastfeeding jaundice: This can occur from suboptimal intake of human milk, typically peaks on days 3 to 5 after birth and is frequently associated with excess weight loss. Breastfeeding fewer than 8 times per day has been associated with higher TSB concentrations.[22]

B. Breast milk jaundice: This jaundice can last up to 3 months despite optimal intake of human milk and adequate weight gain and is non pathologic indirect hyperbilirubinemia.[23]

The AAP recommends implementation of maternity care practices that promote comprehensive, evidence-based, family-centered breastfeeding support: breast milk feeding within the first hour after birth with frequent feeding on demand.[24]

Conclusion

Neonatal hyperbilirubinemia is a common condition in newborns and proper diagnosis and treatment of this condition is vital for a newborn's health. The new AAP guidelines that were published in 2022 have recommended increased thresholds for initiating phototherapy treatment, different treatments by gestational age, emphasis on evaluating for hemolysis (including a direct antiglobulin test in infants born to mothers with O blood type who require phototherapy), waiting at least 12-24 hours before obtaining a follow-up bilirubin level after phototherapy discontinuation for infants at low risk for rebound hyperbilirubinemia, and encouraging breast feeding and enteral supplementation over intravenous fluids unless criteria for escalation of care is met.

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Diagnosing and Managing Heart Failure with Preserved Ejection Fraction

By Awais Ur Rahman, DO; Saskia Levine, MD and Gregory Faughnan, MD

Heart failure (HF) affects an estimated 64 million people worldwide.[1] Its prevalence has been increasing and is projected to continue increasing with a total of 8 million expected cases by 2030 in the United States.[2] This may be partially attributable to an aging population and improvements in treatment, leading to increased survival; however, these figures may be an underestimation due to many patients with left ventricular dysfunction remaining asymptomatic or having comorbidities contributing to their clinical presentation which confounds their diagnosis. Indeed, several studies have noted an increasing incidence of heart failure with preserved ejection fraction (HFpEF) with prominent articles noting lifetime risk of developing HF at age 40 for both males and females, being one in five.[3] The universal definition of HF describes it as a clinical syndrome with signs or symptoms of HF caused by a structural and/or functional cardiac abnormality, which presents with an elevated natriuretic peptide level and/or objective evidence of pulmonary or systemic congestion.[4] The clinical entity is divided into categories based on ejection fraction (EF) with HF with *reduced* ejection fraction (HFrEF) with patients having an EF ≤ 40%, HF with *mildly reduced* EF (HFmrEF) with patients having an EF of 41-49%, and HFpEF, where the EF is > 50% or higher.

In addition to the categorizations, stages and classes of HF have also been developed by the American Heart Association (AHA)/ American College of Cardiology (ACC) and The New York Heart Association (NYHA) respectively, in order to help characterize disease severity and progression. Stage A is reserved for patients that are at risk for HF but who do not have any clinical signs or symptoms and who do not have any structural disease or elevation in biomarkers. Stage B is where patients do not have signs or symptoms of HF though do have evidence of one of the following: structural heart disease, abnormal cardiac function or elevated natriuretic peptide or cardiac troponin levels. Stage C is consistent with a classical understanding of HF, where the patient has a structural and/or functional cardiac abnormality causing signs or symptoms. Stage D simply refers to severe signs or symptoms in a HF patient.[5] The (NYHA) functional classification creates classes based on symptoms with Class I patients having no limitation of physical activity where ordinary physical activity does not cause symptoms of HF; Class II patients having slight limitation of physical activity, being comfortable at rest, but with ordinary physical activity resulting in symptoms of HF; Class III patients having marked limitation of physical activity, being comfortable at rest, but with less than ordinary activity causing symptoms of HF; and class IV patients being unable to perform any physical activity without symptoms of HF, or patients having symptoms of HF at rest.

Of the different divisions of HF, over 50% of cases are attributable to HFpEF; however, it remains an elusive diagnosis leading to undertreatment and an increased burden on the healthcare system.[6] HFpEF is diagnostically challenging due to variations in its clinical presentation. Patients often present with concurrent comorbidities or with a presentation that, in fact, fits the universal definition of HF but is attributed to a non-HF mimicking clinical entity; thus, confounding the clinical diagnosis.[6] Major signs and symptoms of HF are summarized by the major and minor criteria in the Framingham HF Diagnostic Criteria[7] with the most common presenting symptoms being dyspnea, exercise intolerance, and/or edema. Their presence should prompt clinicians to suspect HFpEF, especially in the presence of certain highly associated conditions, such as obesity, hypertension, coronary artery disease, atrial fibrillation, diabetes and chronic kidney disease.[8]

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Initial studies often include chest radiograph, an electrocardiogram (ECG), and echocardiography. Unfortunately, these standard evaluations have limitations in applicability for the diagnosis of HFpEF. A chest radiograph is used to assess for signs of pulmonary edema or to search for other explanations of dyspnea, however, most patients with HFpEF have normal chest radiographs.[8] An ECG may be helpful if it shows prior infarctions or atrial fibrillation, both of which may be implicated in the pathophysiology of HFpEF, but ECG findings are generally nonspecific.[8] An echocardiogram can detect abnormal diastolic function and elevated pulmonary artery systolic pressure, both key findings in HFpEF patients; however, diastolic dysfunction on an echocardiogram is non-specific for HF, and echocardiographic findings cannot diagnose HF alone as mentioned above.[6] Elevated natriuretic peptides are included in the universal definition of heart failure and can be measured in suspected HFpEF; however, in a significant portion of HFpEF patients, natriuretic peptide levels are normal.[9,10]

Non-HF mimics can also make it difficult to ascertain the diagnosis of HFpEF as noted above. Some important mimics that must be considered in contributing to patients presenting with classical symptoms of dyspnea and/or edema include non-cardiac entities, such as nephrotic syndrome, liver failure, anemia, severe obesity, and lung disease. These may be assessed with urinalysis to look for proteinuria, abdominal ultrasound to evaluate the liver, and pulmonary evaluation. Alternatively, cardiac mimics to consider, include infiltrative cardiomyopathy, hypertrophic cardiomyopathy, valvular disease, pericardial disease, and high-output HF. Most patients, however, do not require extensive testing to rule out cardiac mimics, but certainly findings suggestive of these diagnoses warrant further workup. The complexity of presentations of HFpEF and the labyrinthine diagnostic workup of HFpEF has no doubt led to difficulty in recognition of HFpEF and resulted in insufficient medical treatment.

In order to clarify a diagnosis of HFpEF, two important diagnostic scoring systems—HFA-PEFF and H_2FPEF—have been developed and are supported by the European Society of Cardiology and the American College of Cardiology.[6] The H_2FPEF score has taken clinical precedence as it lacks invasive measurement requirements making it more clinically applicable , while also providing higher diagnostic accuracy when compared to the HFA-PEFF score.[11] The H_2FPEF score was developed by assessing the ability of non-invasive clinical criteria to serve as predictive tools to discern between HFpEF versus noncardiac causes of dyspnea. These criteria were compared with HFpEF diagnosed through invasive exercise hemodynamic measurements considered as gold-standard for diagnosis. A BMI greater than 30 kg/m^2 (H_1), treatment with 2 or more antihypertensives (H_2), atrial fibrillation (F), pulmonary hypertension with pulmonary artery systolic pressure greater than 35 mmHg (P), age greater than 60 (E), and filling pressures with echocardiographic E/e' ratio greater than 9 (F) were found to be predictive of HFpEF diagnosis. A composite weighted score (H_2FPEF score) between 0-9 was developed using each variable and a score greater than or equal to 6 was deemed to be highly suggestive of HFpEF. A score of 0-2 suggested low probability, and a score of 2-5 suggested intermediate probability.[12]

Utilization of the score can assist clinicians with supporting the diagnosis of HFpEF, in the setting of confounding clinical pictures. Consider a 65-year-old patient presenting with acute dyspnea and/or edema with a medical history of obesity, hypertension and/or chronic kidney disease, chronic liver disease or chronic lung disease—a fairly common patient encountered in the acute inpatient setting. The symptoms of acute dyspnea and edema can be attributed to several of these etiologies; in addition, these etiologies can be propagating the pathophysiology of underlying HFpEF. Utilizing the H2FPEF score can help elucidate the likelihood of HFpEF in such patients, or alternatively can suggest the need for work-up of HF mimics as noted above and necessitate the involvement of cardiovascular specialists in those with intermediate scores, hastening accurate diagnosis and management.[12]

The overall recommendations for management of HFpEF per ACC guidelines include risk stratifications, and management of comorbidities—which is especially important in the outpatient setting to limit disease pathophysiological progression, but will not be covered herein given the breadth of appropriate recommendations, non-pharmacological management, symptomatic management and modifying the overall disease process via guideline directed medical therapy (GDMT).[6] Despite initial unfavorable data supporting GDMT in HFpEF, deeper pathophysiological understanding, prognostic predictions, and several landmark trials have provided support for the benefit of medical therapy in HFpEF with some GDMT receiving ACC Class 1 and Class 2 recommendations.[13] In addition, specifically for the inpatient clinicians, the STRONG-HF trial has provided pivotal evidence for initiation of GDMT prior to hospital discharge. When adhered to a regime of rapid up-titration with close follow up post discharge after GDMT was started while patients were in the hospital, patients had a higher likelihood of achieving GDMT target doses, had greater improvement in health status, and reduction in heart failure readmission rates or death in 180 days irrespective of baseline ejection fractions.[14] This evidence serves as a powerful impetus for utilization of new recommendations and diagnostic scores to achieve timely HFpEF diagnosis and initiate appropriate GDMT for hospital medicine practicing clinicians.

Of the GDMT available, loop diuretics and sodium-glucose cotransporter-2 inhibitors (SGLT2is) receive the strongest recommendations. Loop diuretics have long been a mainstay of therapy in HF to manage symptoms of congestion and achieve euvolemia, and remain so in HFpEF. The treatment goal with a loop diuretic should be to achieve clinical evidence of decreased fluid retention at the lowest possible dose.[15] Usage of a thiazide diuretic in conjunction with a loop diuretic may be considered when congestion is unresponsive to the loop diuretic alone (ACC). Though they do not have a mortality benefit, they still receive ACC Class 1 recommendation for HFpEF patients with fluid retention and NYHA class II-IV patients. SGLT2is significantly reduce the risk of hospitalization for HF and cardiovascular death.[6] Unless otherwise contraindicated, all patients with HFpEF should be started on an SGLT2i. Two trials, the DELIVER and the EMPEROR-Preserved trial both evaluated the effects of dapagliflozin and empagliflozin,

respectively, on several outcomes in patients with HF and left ventricular ejection fraction (LVEF) greater than or equal to 40%. These trials found improvements in health status, improvements in baseline symptomatic impairment, and in reduction in hospitalizations for HF. In these trials and others, treatment with SGLT2is appeared to be well-tolerated and safe leading to ACC Class 2a recommendation.

The evidence for the use of mineralocorticoid antagonists (MRAs) comes from the TOPCAT trial and its respective post-hoc analyses. TOPCAT[16] was a randomized, double-blind trial in which 3445 patients with symptomatic heart failure with LVEF of 45% or greater were recruited and assigned to receive either spironolactone or placebo. The primary composite outcome of death from cardiovascular causes, aborted cardiac arrest or hospitalization for heart failure was found to be significant post-hoc analysis in the subset of population validated to be taking spironolactone.[17] The analyses also showed benefit in appropriately selected patients, including those with lower natriuretic peptide levels (BNP < 166 pg/ml or NT-proBMP < 682 pg/ml), those with LVEF < 60 percent, and in women.[6] As such, MRAs have received ACC Class 2b recommendation for HFpEF patients without contraindications - women of all ejection fractions (EF), men with EF < than 55 to 60%, and those with fluid retention.[6]

The role of angiotensin receptor-neprilysin inhibitor (ARNI) in HFpEF was gleaned from analyzing the data from the PARAGON-HF trial. This was a large randomized, double-blind trial in which 4822 patients with LVEF greater than or equal to 45%, elevated natriuretic peptides, and evidence of structural heart disease were assigned to receive either sacubitril/valsartan or valsartan alone with the primary composite endpoint of total hospitalizations for heart failure and death from cardiovascular causes.[6] Although the data showed no statistically significant benefit in the primary composite endpoint, post-hoc analysis showed a potential benefit in patients with LVEF between 45% to 57% as well as in women when compared to men. ACC has subsequently provided a Class 2b recommendation for the careful use of sacubitril/valsartan to decrease hospitalizations for women and men with LVEF < 55% to 60%.[6,15]

While there is a growing body of evidence for renin-angiotensin-aldosterone system inhibition for the treatment of heart failure with a reduced ejection fraction, studies in HFpEF have not shown similar benefits. The CHARM-Preserved trial was a large double-blind trial with 3023 patients with an EF greater than 40% who were randomized into either an angiotensin receptor blocker (ARB) (candesartan) or placebo group. The study found no statistically significant rates of cardiovascular death or inpatient admissions for heart failure at 3.5 years.[18] A post-hoc analysis did however, reveal possible benefit of candesartan compared to placebo when patients were further stratified into groups based on EF for the lower end of the LVEF spectrum.[19] Further meta-analysis evaluating ARBs have subsequently been performed including data from 4 trials and 7694 patients with HFpEF and did not show significant benefit on cardiovascular mortality, all-cause mortality, or hospital admission for acute decompensated heart failure.[20] The ACC has subsequently provided a Class 2b recommendation for ARB use in select patients with HFpEF to potentially reduce risk of hospitalization in patients with an EF on the lower end of the normal EF spectrum, especially those who are eligible for ARNI but are intolerant.[6]

Lifestyle modification remains an important aspect of HFpEF management. Poor exercise tolerance and obesity have been associated with poor quality of life and worse long-term prognosis. Dyspnea on exertion and poor activity tolerance are common factors leading to decreased quality of life. Two observational studies[21,22] have demonstrated as little as 3 months of regular endurance or resistance training to have positive impacts on VO2- a measure of cardiac and skeletal muscle oxidative function. Additionally, both studies also showed improvements in patient centered outcomes of quality of life. Calorie restriction in addition to structured exercise programs have shown further benefit to improvement in cardiac fitness.[23] Lastly, data review from AHA that evaluated the benefit of supervised exercise training in HFpEF has shown that it improves exercise capacity. This provides inpatient clinicians another valuable opportunity to improve patient outcomes by initiating rehabilitation referrals at time of discharge in addition to initiation GDMT as above.[24]

With advancements in understanding of HFpEF disease process, and improvements in understanding of medical therapies, it is more imperative than ever that clinicians are able to recognize the diagnosis of HFpEF in order to initiate comprehensive therapeutic management. With the conclusions of the STRONG-HF trial showing that initiation of aggressive management prior to discharge has improvements in overall health, readmission rates and mortality, the clinician in the hospital, with the help of the H2FPEF score, should aim to become skilled at recognizing, working-up and treating HFpEF.

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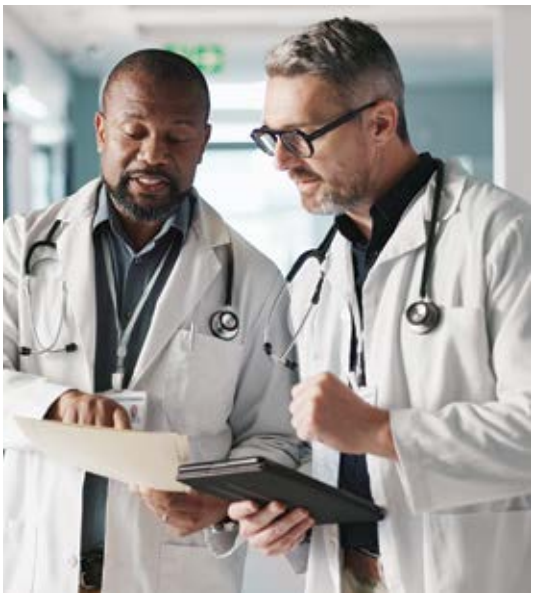
Questions and Answers on Community Acquired Pneumonia

By Joshua Steinberg, MD

As a student in medicine, a resident on night float, and now a family physician in office, hospital, and all the other places we work, you have managed plenty of community acquired pneumonia (CAP). Much in the diagnosis and treatment of pneumonia has not changed. CAP hospitalizes more than one million adults per year, more than all other causes aside from childbirth.[1] Until dwarfed by COVID-19's arrival, CAP has been the ninth leading annual cause of death and the leading infectious cause of death.[2] And CAP remains a domain of empiric treatment for an infection where we rarely identify the pathogen.[3] But there are a few changes with updated evidence and guidelines of the past few years. Let's review some relevant to hospital care in question-and-answer style.

Have the basic categories of pneumonia changed?

Yes, they have. Current categories are community acquired pneumonia, hospital acquired pneumonia, and ventilator associated pneumonia. Many will remember a fourth category, health care associated pneumonia (HCAP). HCAP is no longer a clinical entity since the 2019 American Thoracic Society and Infectious Diseases Society of America (ATS-IDSA) joint guideline[4] on CAP. The idea was that certain community dwelling patients might have so many interactions with health care (wound care, dialysis, nursing home, etc.) that their colonizing and infecting pneumonia pathogens would be more like hospital patients than community dwelling patients. But that turned out not to be true. Thus, the three current pneumonia categories are CAP, HAP, and VAP.



How can I decide which patients need hospitalization for pneumonia?

Use a mortality prediction tool to admit those with significant risk.[5,6] The Pneumonia Severity Index (PSI) is a valid, evidence-based mortality prediction tool. But the PSI requires factors that no physician has in the office (BMP, CXR, and ABG), and only a few have on typical cases in the emergency department such as arterial pH and PaO_2. Plus, the PSI's point system is complex, requiring a point-of-care decision support calculator or spreadsheet to implement. In contrast, the CURB-65 and CRB-65 are effective and practical in nearly every setting. Factors that receive 1 point each are **C**onfusion, **U**rea (BUN > 19 mg/dL), **R**espiratory rate ≥ 30, **B**lood pressure low (SBP < 90, DBP < 60), and age ≥ **65** years. In the office without labs, CRB-65 will do. A score of 2 or more on CURB-65 or 1-2 or more on CRB-65 identifies a patient with a mortality risk of at least 5% and should prompt consideration of hospital admission. Other factors also influence hospitalization such as oxygen requirements and ability to take antibiotics and other medicines by mouth.

What diagnostic testing should hospitalized pneumonia patients undergo?

After thorough history and physical exam, a chest x-ray (CXR) really is necessary in the hospital setting. As much as I cheer the power of careful history and examination, there simply are no signs and symptoms powerful enough to rule in pneumonia.[7] Plus, CXR can also reveal complications like effusion and empyema, and comorbidities like heart failure and COPD.

After CXR, much testing is surprisingly discretionary. The ATS-ISDA 2019 guideline points out that most testing is of low yield; therefore, they recommend neither for nor against a great deal of testing except in certain situations. Blood cultures and sputum gram stain and culture should be obtained for patients with severe pneumonia (in the ICU), when covering for MRSA or Pseudomonas, when previously infected anywhere with MRSA or Pseudomonas, and when the patient was hospitalized to receive IV antibiotics within the last 90 days. Pneumococcal urinary antigen only for those with severe (ICU) pneumonia. Legionella urinary antigen again only if in the ICU or if the patient is at risk for exposure to an outbreak such as by travel history. Influenza testing should be done on all when influenza is circulating. And here's one more new recommendation. Nares should be swabbed for MRSA for the same patients noted above who get blood and sputum studies because MRSA nasal carriage testing helps antibiotic coverage decisions with test-and-escalate and cover-and-deescalate treatment strategies. See the included Table 1 on inpatient diagnostic testing.

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Table 1: Inpatient Pneumonia Diagnostic Testing

clinical indication	Blood culture	Sputum gram stain & culture	Pneumococcal urinary antigen	Legionella urinary antigen	Influenza molecular assay	Procalcitonin	MRSA nasal swab PCR
ICU admission (severe CAP)	yes	yes	yes	yes	yes [3]	no [4]	yes
Hospitalized and					↓	↓	
- covered for MRSA	yes	yes					yes
- covered for Pseudomonas	yes	yes					
- previously infected MRSA [1]	yes	yes					yes
- previously infected PAerug [1]	yes	yes					
- was hospitalized and had IV ABX ≤ 90 days ago [2]	yes	yes					yes
Travel ≤ 2 weeks hotel, cruise, Legionella outbreak				yes			
Influenza circulating in community							
All others							

1. infected anywhere, particularly but not exclusively in respiratory tract
2. hospitalization and IV ABX not necessarily same event
3. test all when flu circulating in community, nucleic acid amplification test assay preferred over antigen test
4. does not distinguish bacterial vs. viral well enough to guide antimicrobial therapy initiation decisions

Adapted from ATS-IDSA CAP guideline 2019 by Drs. Michael Putnam, Hilary Mount, and Joshua Steinberg.

I've heard great things about procalcitonin testing, so how about testing that, too?

Yeah, I've heard great things about procalcitonin (PCT) testing too! Elevated PCT correlates with bacterial infection while normal PCT levels correlate with absence of bacterial infection. The ATS-ISDA guideline acknowledges how promising this sounds. But then they review the evidence that studies of using PCT testing in diagnosis and treatment have failed to improve the care and outcomes of CAP patients. Perhaps we don't yet know how best to use the test. Perhaps we doctors run IV antibiotics for CAP in the hospital out of caution or tradition or stubbornness, regardless of PCT results. Whatever the cause, since we haven't figured out collectively how to use PCT in the care of CAP, ATS-IDSA recommends against procalcitonin testing.

What's new in antibiotic management of hospitalized community acquired pneumonia patients?

ATS-IDSA divides empiric antibiotic management recommendations of CAP patients into hospital ward vs. ICU ward, and divides again further into 4 situational sub-categories for each. Probably the easiest and most helpful thing to explain is their updated paradigm for antibiotic management. For hospital ward and ICU ward, they recommend a standard antibiotic regimen and a broad antibiotic regimen. And then for each of the 4 situational sub-categories, they recommend generally one of two schemes:

1. Treat standard, do some testing (MRSA, Pseudomonas), and broaden coverage if positive
2. Treat broad, do some testing (MRSA, Pseudomonas), and narrow coverage if negative

See the guidelines or point-of-care resources for the fine details of antibiotic recommendations.

What about the special case of aspiration pneumonia?

It turns out the special case of aspiration pneumonia is not so special. Previous research with invasive sampling techniques in non-representative, non-generalizable populations suggested anaerobic organisms were common. More recent studies show that anaerobes are uncommon. Further studies show how common aspiration itself is in ill and normal patients. Thus, aspiration pneumonia is no longer the special entity it was previously. ATS-IDSA recommends standard community acquired pneumonia coverage without anaerobic coverage for patients suspected of aspiration pneumonia, except in two special situations: lung abscess and empyema.

What about the special case of community acquired pneumonia patients who test positive for influenza?

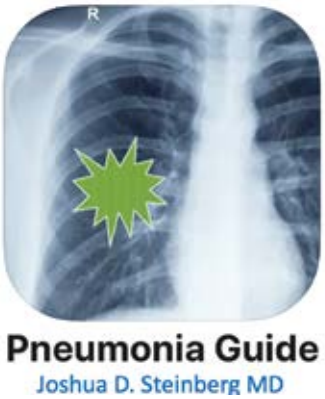
We in primary care see mild, moderate, and severe cases of influenza all flu-season long. But a patient with CAP who also has influenza is different. The ATS-IDSA guideline recommends covering these patients with an influenza antiviral like oseltamivir regardless of duration of illness. And they recommend CAP standard anti-bacterial antibiotics as well because influenza is a strong risk factor for co-infection presently or complicating co-infection subsequently with staph aureus, pneumococcus, h. flu, and more.

If you double-cover pneumonia and influenza with antivirals and antibiotics, how about pneumonia and COVID-19?

There was no COVID-19 when the latest ATS-IDSA community acquired pneumonia guideline was issued in 2019. When COVID-19 blazed across the healthcare landscape in 2020, the assumption was that coronavirus pneumonia might be akin to influenza pneumonia, thus antibacterial antibiotics were heavily used empirically early on. But now we have evidence. IDSA maintains an outstanding, oft-updated guideline on COVID-19 management online.[8] The "Bacterial Co-Infections and Antibiotic Use" section reviews how rarely co-infection occurs, 1-4% at most depending on study. Given the risk of IV antibiotic use itself and the potential of empiric antibiotics to cultivate resistance in whatever late-developing superinfections may occur, IDSA does not recommend antibiotic coverage for COVID-19 patients.

My patient is getting better, going home from hospital after CAP. Should I get a follow-up chest x-ray?

Older physicians may remember the recommendation to check a chest x-ray weeks after pneumonia to make sure that cancer was not obscured on chest x-ray during the acute infection. ATS-IDSA now says not to bother, and the reasons are interesting. It is true that some pneumonias occur in the setting of undiagnosed cancer, estimated at 1-4% of the time, almost always in smokers. But CXR isn't any good at finding those cancers. The ATS-IDSA guideline reminds us that the best way to find these occasional cancers in smokers is to offer low dose lung CT screening. If you are going to check for cancer, do the right test.



Any good point-of-care decision-support resources to help me keep all this straight?

Glad you asked. I can't keep it all straight, either. So, I've written a free iPhone app called *Pneumonia Guide*[9] to help a clinician, especially students and residents, get quick accurate answers to questions like these on the fly during patient care. I'm always happy for feedback and suggestions which can be given via email link within the app. The app is available at the Apple Store.

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Options for Inpatient Buprenorphine Induction

By Julia Cooper, MD, AAHIVS and Talia Urdanigo, MD

According to the 2022 NYS DOH opioid annual report[1], there were 13,976 hospital discharges in NYS in 2020 where opioid use was the primary diagnosis, and an unknown additional number of hospitalizations complicated by opioid use disorder. A study of a large hospital system in Delaware corroborated many clinicians' observations that patients with opioid withdrawal are disproportionately likely to leave AMA compared to other patients, and are increasingly likely to do so each year (from 2017 – 2020.)[2] Buprenorphine can be a very flexible tool in the inpatient management of opioid withdrawal and pain, and its use can reduce both AMA discharges and post-discharge overdose risk. The main barrier to buprenorphine use is the fear of precipitating withdrawal. This article complements last issue's excellent piece by Drs. Ghayalod and Aggarwal, and aims to make clinicians comfortable with multiple ways of safely starting buprenorphine, with emphasis on the hospital setting.

Buprenorphine is a semi-synthetic opioid which has a moderate analgesic effect but which causes less euphoria, sedation, or respiratory suppression than other opioids.[3] Buprenorphine does not induce tolerance or hyperalgesia to the same extent as other opioids.[4] Finally, buprenorphine's minimal effect on respiratory drive also appears not to be dose-dependent but instead has a "ceiling effect," even when dosed generously.[3] It is a partial agonist at the mu-opioid receptor, which it binds with extremely high affinity. This means that buprenorphine can suddenly displace other opioids from the receptor, and replace their effect with its own slightly weaker effect (an effect called buprenorphine-precipitated opioid withdrawal or BPOW, a more jarring and painful experience than the gradual withdrawal symptoms of abstinence). In order to avoid rapidly displacing other opioids, buprenorphine cannot simply be administered; instead, we use the phrase "buprenorphine induction" to describe the careful timing of the first doses. The standard protocol for buprenorphine induction advises waiting until a patient is already withdrawing from their last dose of opioid, and waiting at least 12 hours since the last use of heroin or up to 72 hours from the last dose of fentanyl. (Although clinicians may think of fentanyl as short-acting, fentanyl's lipophilicity prolongs its presence in the body and, therefore, its window for potential precipitated withdrawal.) The longest-acting opioid of all is methadone, which is why a standard transition from methadone to buprenorphine requires a long period in withdrawal which patients rarely tolerate.

Inversely, once opioid receptors are flooded with buprenorphine, its high affinity makes it unlikely that another opioid will displace it. When patients who already use buprenorphine take additional opioids, they are therefore somewhat protected against respiratory suppression. For this reason, many outpatient buprenorphine prescribers have adopted a harm-reduction approach whereby they will continue prescribing buprenorphine to patients at ongoing overdose risk. In both the clinic and the hospital, for patients who require chronic or acute-on-chronic pain control, buprenorphine can be used as a less-sedating baseline, and also reduce the risk of respiratory suppression if additional opioids are required for breakthrough pain. This approach has performed well for patients with chronic pain, including elderly patients, patients with sickle cell disease[5], and hospice patients.[6]

General concepts in buprenorphine induction

In order to minimize the risk of precipitated withdrawal, many protocols rely on waiting until the patient is in at least "mild withdrawal" according to the COWS score. Note that patients can have a COWS score of 5 without any objective or observable signs, and note also that being too irritable to participate in the questionnaire is itself worth 4 points; therefore, nurses or clinicians must be careful not to underestimate a patient's level of withdrawal by skipping the subjective questions, whether due to time pressure or due to a patient's irritability. Patients also recognize their own withdrawal symptoms, so simply asking, "do you feel as though you're in withdrawal now?" can validate a clinical suspicion of withdrawal without calculating a precise score.

It is important to have medications on hand for symptomatic management of withdrawal, and if the risk of precipitated withdrawal seems high, it is reasonable to pre-treat before introducing buprenorphine. Symptomatic management typically includes clonidine, hydroxyzine, diphenhydramine, ondansetron, loperamide, and/or gabapentin. (See Table 1.) Hospitalists or ED providers may wish to create a "withdrawal management" order set to encourage symptom management at their institutions. Treating the symptoms of withdrawal should not delay buprenorphine induction but it can reduce the risk of patients leaving "against medical advice." Finally, although severe intractable BPOW is very rare, hospitalists should be aware that it can be managed with larger doses of buprenorphine (see high-dose section) and, if BPOW persists, benzodiazepines, ketamine or dexmedetomidine may be added.[7]

Standard buprenorphine induction

The standard buprenorphine induction taught in X-waiver courses requires waiting until a patient is in moderate withdrawal and gradually introducing sublingual buprenorphine 2-4mg at a time. Because patients must tolerate several hours (or even a day) of withdrawal before starting, plus 1-2 days of gradual titration to the full dose, and because there is still some risk of BPOW, some patients have negative experiences with the standard induction and are unwilling to do it again.

Table 2 shows the standard buprenorphine induction protocol. Note that each section is read from top to bottom, with "yes" and "no" at each level arranged to the right and left.

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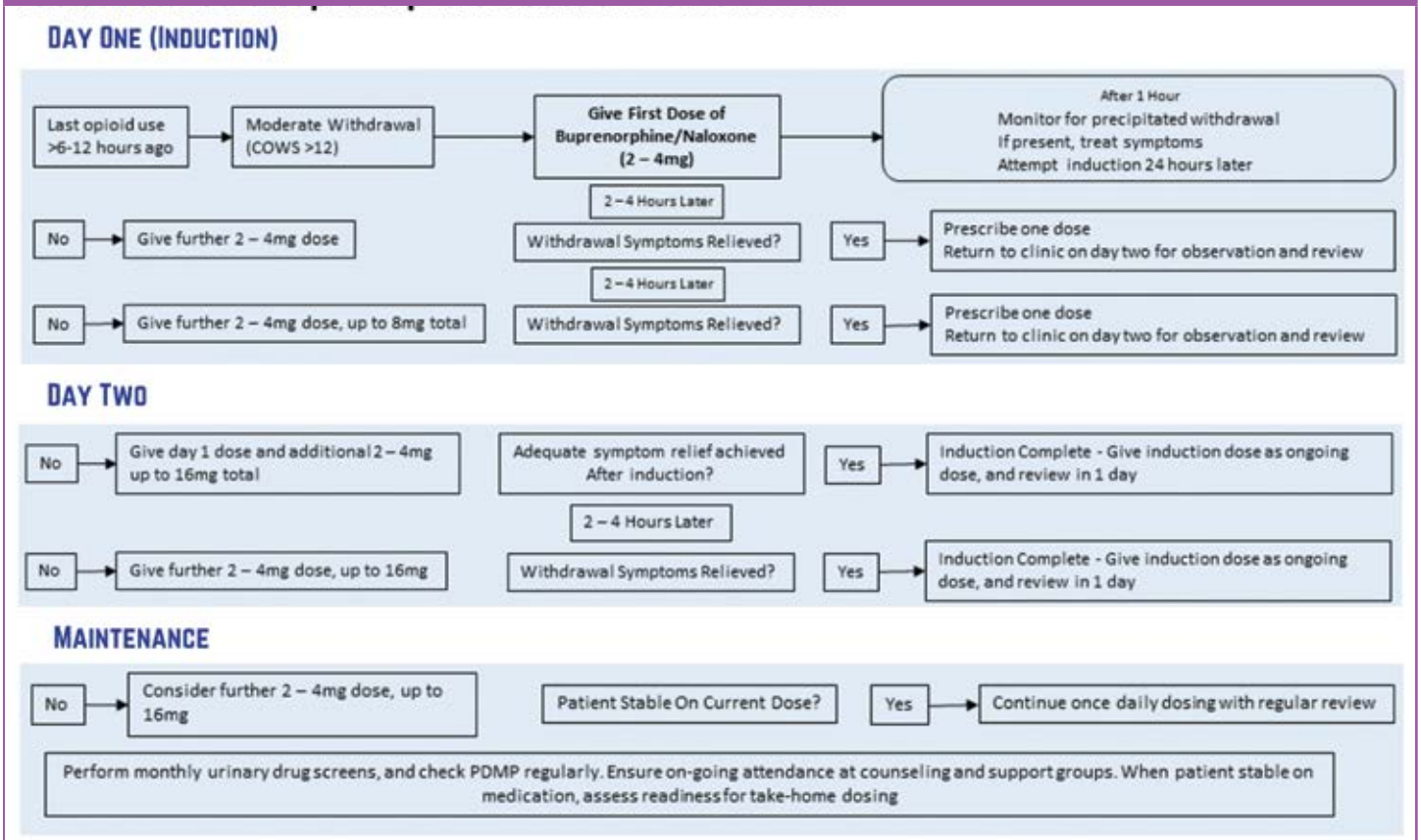
Table 1: Menu of medication options for pre-treatment before induction or for symptomatic management of opioid withdrawal

Medication	Dose & Interval	Indications	Notes
Clonidine	0.1mg PO q6h PRN	Anxiety, agitation	
Hydroxyzine or Diphenhydramine	25 – 50mg PO q6h PRN (or IV/IM) Can also give a large bedtime dose of hydroxyzine 100mg.	Pruritus, restlessness, insomnia	Hydroxyzine slightly more sedating; diphenhydramine slightly better for pruritus.
Ondansetron	4 – 8mg PO, SL or IV q8h PRN	Nausea	More effective before vomiting occurs than after.
Loperamide	4mg PO, then 2 additional mg PRN loose stool up to 16mg	Loose stools	
Gabapentin	300mg PO, at night or up to q8h PRN, and repeat or titrate as needed	Pain, insomnia, agitation	Dose more generously for withdrawal than for daily outpatient use – mild sedation is not unwelcome in this context.
Trazodone	50 – 200mg PO PRN at night	Insomnia	
Tizanidine	4mg PO up to q8h PRN	Anxiety, insomnia, agitation	Of all these PRN options, a dose of tizanidine may be the most helpful in the event of mild precipitated withdrawal.
Nonpharmacologic measures: Dim lights, provide warm blankets, minimize noise, and ensure access to PO liquids and toilet / emesis basin			

Table 2: Standard buprenorphine induction from SAMHSA[8]



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High dose induction

High-dose induction or "macrodosing" takes advantage of the safety of high doses of buprenorphine. This method rapidly introduces a large dose of buprenorphine which displaces the unwanted opioid quickly but floods enough receptors to exert a therapeutic effect, basically zooming right past the risk of BPOW. Because high-dose induction rapidly achieves a therapeutic dose, patients spend less time painfully titrating to full dose. BPOW is still possible, though the risk is lowest once the patient is in mild-moderate withdrawal. Expert recommendations vary about exactly when to start; we recommend waiting until COWS >8 or when the patient shows two or more observable signs of withdrawal (tachycardia, mydriasis, yawning, rhinorrhea, vomiting, diarrhea, or piloerection). Some prefer to time the first dose based on last use, waiting > 12 hours from short acting opioids or as long as > 72 hours from fentanyl. Although clinical trials have yet to be published, addiction medicine experts have also reported success with giving one large, receptor-flooding dose of 24 or 32mg sublingually all at once when withdrawal is just beginning. There is also evidence of the safety of a very high first-day dose, up to 64mg in total, although this is not an approved or necessary maintenance dose.[9] Clinicians can also use high doses to "rescue" a patient who has been thrown into precipitated withdrawal by a smaller buprenorphine dose (e.g., a failed standard induction), potentially salvaging that patient's relationship with buprenorphine; or to rapidly relieve the symptoms of precipitated withdrawal after receiving Naloxone for an overdose. High-dose induction requires hours rather than days - rapid enough to be undertaken in the office, the emergency department, or within a single shift on the hospital floor.

Table 3 shows a recommended high-dose induction schedule for 8mg sublingual buprenorphine films, starting when the patient is in moderate withdrawal. Remember that pretreatment with comfort medications is encouraged.

Table 3. High dose induction protocol taught at Institute for Family Health (FH) adapted[9,10,11]

Day 1- Time	Buprenorphine Dose	
Hour 0	16mg	
30 minutes, if patient endorses withdrawal symptoms	16mg	
1 hour later, if patient still endorses withdrawal symptoms	8mg	
Repeat hourly as needed for persistent withdrawal symptoms, up to eighth film	8mg	Total first-day dose up to 64mg or (8) 8mg films

Day	Buprenorphine Dose	Total Buprenorphine Dose
2	8mg BID, additional 8-16mg if needed	16-32mg
3	Repeat day 2 dose	16-32mg
4	Continue on 16-24mg as daily maintenance dose. Divided BID or TID doses may be most effective for pain. May take 4-6 weeks for maximum benefit from dose to be noted.	16-24mg

Microdosing induction

Microdosing, also known as "low-dose buprenorphine with opioid continuation," represents a different style of introducing buprenorphine. Rather than entirely displacing other opioids, microdosing involves giving tiny doses of buprenorphine and escalating very gradually, to gently cross-taper from other opioids to buprenorphine while trying to avoid any withdrawal, precipitated or otherwise. By the time the patient works up to full-dose buprenorphine, their other opioids (e.g., hydromorphone) can be discontinued or converted to as-needed dosing. Microdosing takes several days, and the small doses can be difficult to measure. (In the outpatient setting, it can involve splitting 8mg Suboxone films into maddeningly small slivers). However, there is an obvious advantage to transitioning to buprenorphine without requiring an interval of withdrawal symptoms and without interrupting pain management. For patients at risk of withdrawal from illicit drug use, it is appropriate to use short-acting opioids such as hydromorphone as a bridge, preventing withdrawal while transitioning to buprenorphine inpatient.[12] Microdosing also allows a smooth transition from methadone to buprenorphine, which is sometimes necessary to facilitate discharge to another facility (and sometimes simply a patient's preference).

Precipitated withdrawal is still possible with microdosing, especially mid-protocol with the subtherapeutic-but-not-tiny doses (or higher). If BPOW occurs, most experts recommend continuing the non-buprenorphine opioid without alteration, treating the symptoms, and postponing the next dose escalation by one day (i.e., if withdrawal symptoms occur on Day 4, that same dose should be repeated on Day 5 before escalating).[13] "Rescue" by converting to high-dose buprenorphine induction is another option in cases of severe BPOW.

There are many different microdosing protocols in the literature, ranging from "rapid" four-day sublingual inductions to ten-day transitions from methadone. In order to make tiny doses easier to administer, some protocols begin with buccal, transdermal or IV buprenorphine and transition to the preferred discharge regimen, sublingual films, for the larger doses. Several trusted protocols are quoted in Tables 4 and 5, but this is not an exhaustive menu of options. The hospital formulary is often the limiting factor in inpatient buprenorphine prescribing. Having the inpatient pharmacy verify and dispense a Suboxone (sublingual buprenorphone/naloxone) prescription from a community

Table 4. Standard inpatient or outpatient sublingual microdose protocol. This is one protocol used at the Institute for Family Health, adapted by the authors.[14] A similar protocol was recommended for outpatient and even telehealth use in JABFM[15]

Day	Buprenorphine sublingual dose (in units of 2mg films)	Total daily buprenorphine dose	Full agonist (eg hydromorphone)
1	¼ film in the AM	0.5mg	Continue
2	¼ film twice daily	1mg	Continue
3	½ film twice daily	2mg	Continue
4	1 film twice daily	4mg	Continue
5	1½ films twice daily	6mg	Continue
6	2 films twice daily	8mg	Continue
7	2 films three times daily, or ½ of an 8mg film three times daily	12mg	Considering stopping full agonist if patient is comfortable
8 and onward	8mg film twice or three times daily	16-24mg maintenance dose	Stop full agonist (or deescalate to PRN dosing for pain, if applicable)

pharmacy is another strategy sometimes available to family medicine teams. We encourage hospitalists to take inspiration from the options in Tables 4 and 5 and to work with their pharmacy and therapeutics committees to find the protocol that is most acceptable to their pharmacy and floor nurses.

Discharge considerations

Unlike inpatient methadone initiation, which requires coordination at discharge to secure a next-day appointment with a methadone clinic, discharge planning with buprenorphine is somewhat more flexible. The DEA no longer requires prescribers to hold a 2000-DATA (or "X") waiver to prescribe buprenorphine, although providers who have not previously completed an 8-hour opioid training must complete the CAA 2023 training when registering for or renewing their DEA.[19] Prescribers may also prescribe buprenorphine using their hospital's DEA number, with the permission of the institution. Buprenorphine prescriptions must be sent electronically in New York State, and should be sent in advance to avoid delays due to prior authorization (especially if using tablets or patches, which are more expensive than films). Ideally, patients should have their hospital follow-up visit within a week, with a primary care provider who can both review the hospitalization and continue the buprenorphine prescribing. However, since consistent access to buprenorphine reduces overdose risk, we feel it is appropriate to prescribe up to two or even four weeks' worth, if necessary, in order to bridge to the soonest available OBAT appointment (in addition to a closer discharge follow-up visit with the PCP). If the hospitalist team is not affiliated with a family medicine clinic, they or the hospital's care coordinator may wish to identify a primary care provider with OBAT experience (or an addiction psychiatrist) and establish an expedited intake protocol with them.

For patients seeking placement in acute rehab or long-term care facilities, it can be helpful to document when buprenorphine is being used for pain management, since there have been cases of discriminatory rejection of patients who require addiction treatment. For patients of long-term care facilities, a written prescription for buprenorphine or other controlled substances is permitted and can be faxed to the facility.[19]

To review the potential applications of each dosing protocol, consider the following clinical examples:

Example candidates for standard induction-

- Convenient for outpatient induction, but can be offered to any patient in moderate withdrawal.

Example candidates for low dose induction (microdosing)-

- Hospitalized patient, previously on methadone, requiring breakthrough opioids for episodic pain but also experiencing over-sedation.
- Patient with high opioid tolerance due to IVDU, now requiring high dose hydromorphone for postoperative pain, facing >1- week hospitalization and interested in eventually discharging on buprenorphine-naloxone films.
- Any patient interested in establishing buprenorphine treatment, but not currently in withdrawal. Excellent option for patients fearful of withdrawal due to prior bad experience with BPOW.
- Pregnant patients, as any withdrawal can precipitate preterm labor.

Example candidates for high dose induction-

- Patient who injects drugs, s/p Narcan in the emergency department and facing a brief unrelated medical admission, now in withdrawal and asking to leave against medical advice.
- Patient with opioid use disorder and high opioid tolerance who faces a painful surgery 2 days in the future. If the patient is already withdrawing or can hold out until withdrawal begins, rapid high-dose induction can be done so that the patient is already on buprenorphine before surgery, and additional opioids can be given as needed with less risk of respiratory suppression. It may be necessary to reassure the surgical team that patients on buprenorphine require generous doses of breakthrough opioids.

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Table 5: Comparison of our current sublingual microdose induction with other protocols adapted[7, 14-18]
Each begins with a full opioid agonist (eg hydromorphone) and some form of buprenorphine, and transitions to sublingual buprenorphine films. Most protocols do not taper the full agonist prior to discontinuation, but the full opioid agonist doses should be at least partly "PRN," to ensure that the smallest effective daily dose is used. BID – twice daily; TID – three times daily; TDD – total daily dose; SL – sublingual; IV - intravenous
‡In each protocol, if full dose buprenorphine is inadequate for pain control, the full agonist instead of being discontinued can be offered in "PRN" doses for breakthrough pain.

	Sublingual (SL) 2mg to 8mg films (also convenient for outpatient use)	**Buccal films to SL films**	**"Rapid" induction with SL 2mg films**	**Transdermal (Butrans) to SL**	**Intravenous to SL**
Day 1	¼ film in the AM, TDD 0.5mg Continue full agonist	225mcg buccal once (75mcg film + 150mcg film) Continue full agonist	0.5mg q3h, TDD 4mg Continue full agonist	Apply patch (dose based on tolerance; see notes†) Continue full agonist	0.15mg IV q6h Continue full agonist
Day 2	¼ film BID, TDD 1mg Continue full agonist	225mcg buccal BID Continue full agonist	1mg SL q3, TDD 8mg Continue full agonist	Leave same patch Reduce full agonist by half	0.3mg IV q6h Continue full agonist
Day 3	½ film BID, TDD 2mg Continue full agonist	450mcg buccal BID Continue full agonist	8-16mg once plus 1-4mg q3h PRN withdrawal or pain, maximum TDD 32mg Continue full agonist	Leave patch and give 2mg buprenorphine SL film BID Discontinue full agonist or give breakthrough PRN if needed	2mg SL film BID Continue full agonist
Day 4	1 film BID, TDD 4mg Continue full agonist	2mg SL film BID Continue full agonist	Consolidate Day 3 dose into once daily maintenance dose Discontinue full agonist	Leave patch and give 4mg SL buprenorphine film TID Discontinue full agonist	4mg SL film TID Discontinue full agonist or give small PRN if needed
Day 5	1½ films BID, TDD 6mg Continue full agonist	4mg SL film BID Continue full agonist		Give 8mg buprenorphine film BID and remove patch at night	Continue full dose SL buprenorphine (8mg BID or TID) Discontinue full agonist
Day 6	2 films BID, TDD 8mg Continue full agonist	4mg SL film TID Continue full agonist		Continue full dose SL buprenorphine film (8mg BID or TID)	
Day 7	2 films TID, or ½ of an 8mg film TID; TDD 12mg Discontinue full agonist or give breakthrough PRN if needed	8mg SL BID Discontinue full agonist			
Day 8+	8mg film BID or TID; TDD 16-24mg Discontinue full agonist‡				

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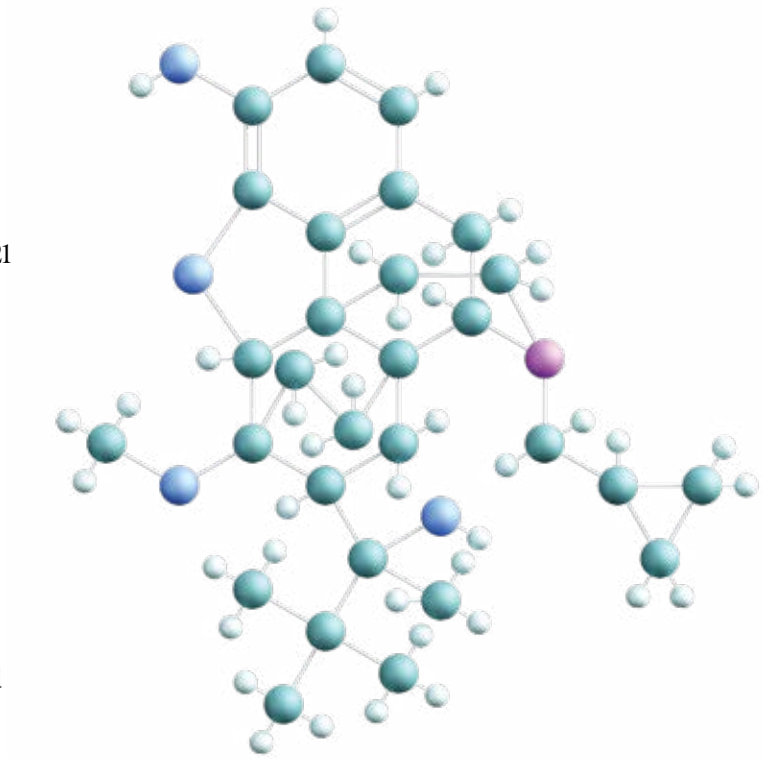
Julia Cooper, MD, AAHIVS *completed her residency and HIV specialist training at the Lawrence Family Medicine Residency and then served as core faculty at the Harlem Residency in Family Medicine, where she practiced full spectrum outpatient care and rounded at Mount Sinai Hospital. She recently resigned from Harlem and will soon be starting at Trillium Health in Rochester. Dr. Cooper enjoys teaching, especially about HIV and other infectious diseases, addiction medicine, liver disease, and gender-affirming care, and is a member of NYSAFP.*

Talia Urdanigo, MD *is a graduate of the Mount Sinai Downtown Residency in Urban Family Medicine. She is currently an addiction medicine fellow at the Institute for Family Health. Her interests include working with marginalized communities, particularly at the intersection of addiction, homelessness and LGBTQ identities, and promoting the continued education of clinicians.*



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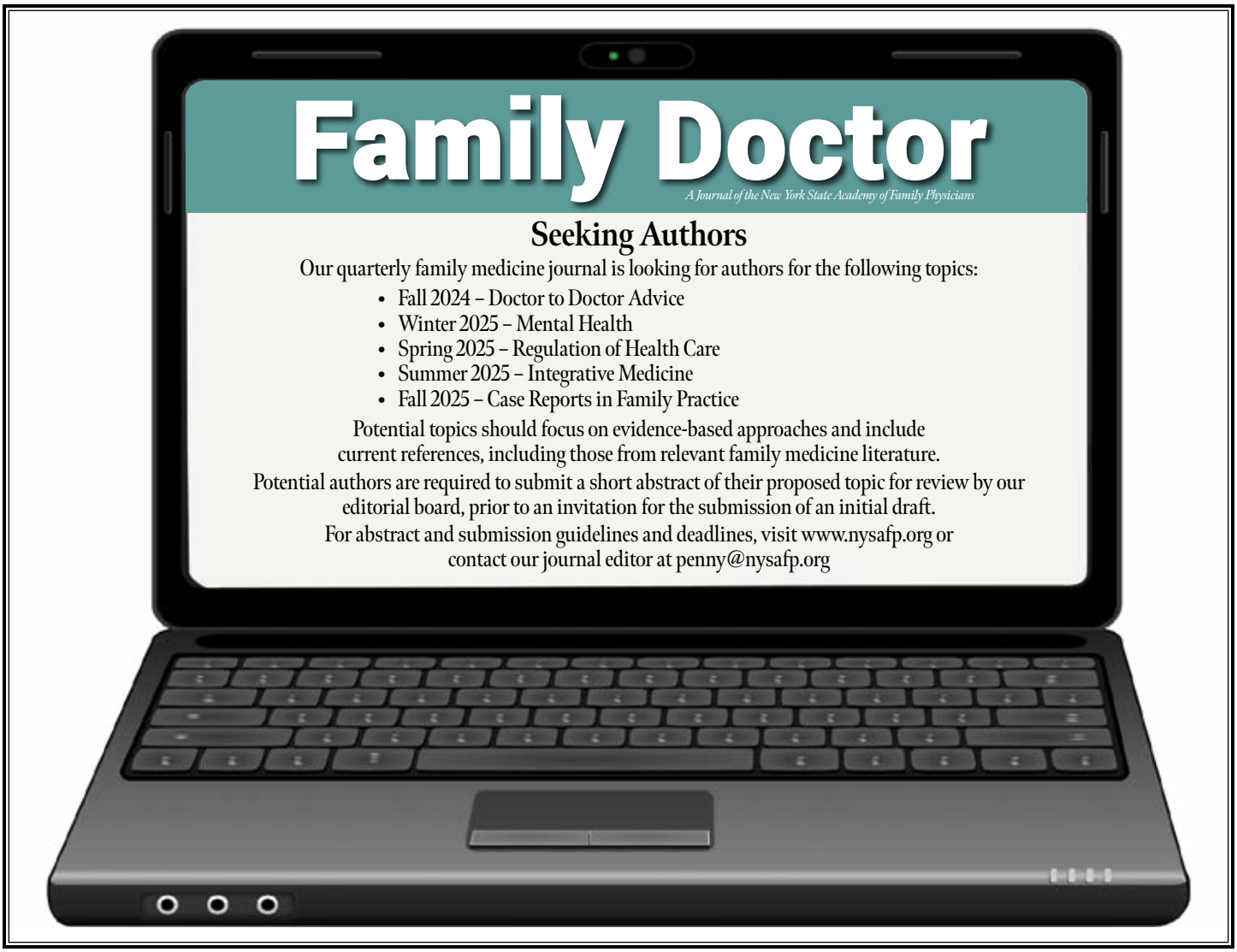

2024 Congress of Delegates

All documents available on our website:
https://www.nysafp.org/nysafp-governance/congress-of-delegates/

May 11 – 9:00am Virtual opening
May 18-19 – Reconvene at the Desmond Hotel Albany

The 76th Congress of Delegates (COD) of the New York State Academy of Family Physicians will convene on Saturday, May 11th at 9:00 AM as a virtual meeting. Congress will open and proceed with the business portion of the meeting.

Resolutions will be available for virtual testimony from the conclusion of the May 11 opening session until May 16 at 11:59pm

The Congress will reconvene in person on Saturday, May 18th and 19th at the Desmond Hotel in Albany to conclude business. Join us for lunch when we will have Commissioner McDonald join us for a Town Hall session.

AAFP President, Steven P. Furr, MD, FAAFP and Shawn Martin, Executive Vice President and Chief Executive Officer will also be on hand for the weekend.

Join us and make a difference!

Become a delegate or alternate from your county to have a vote and shape the future of family medicine in New York State.

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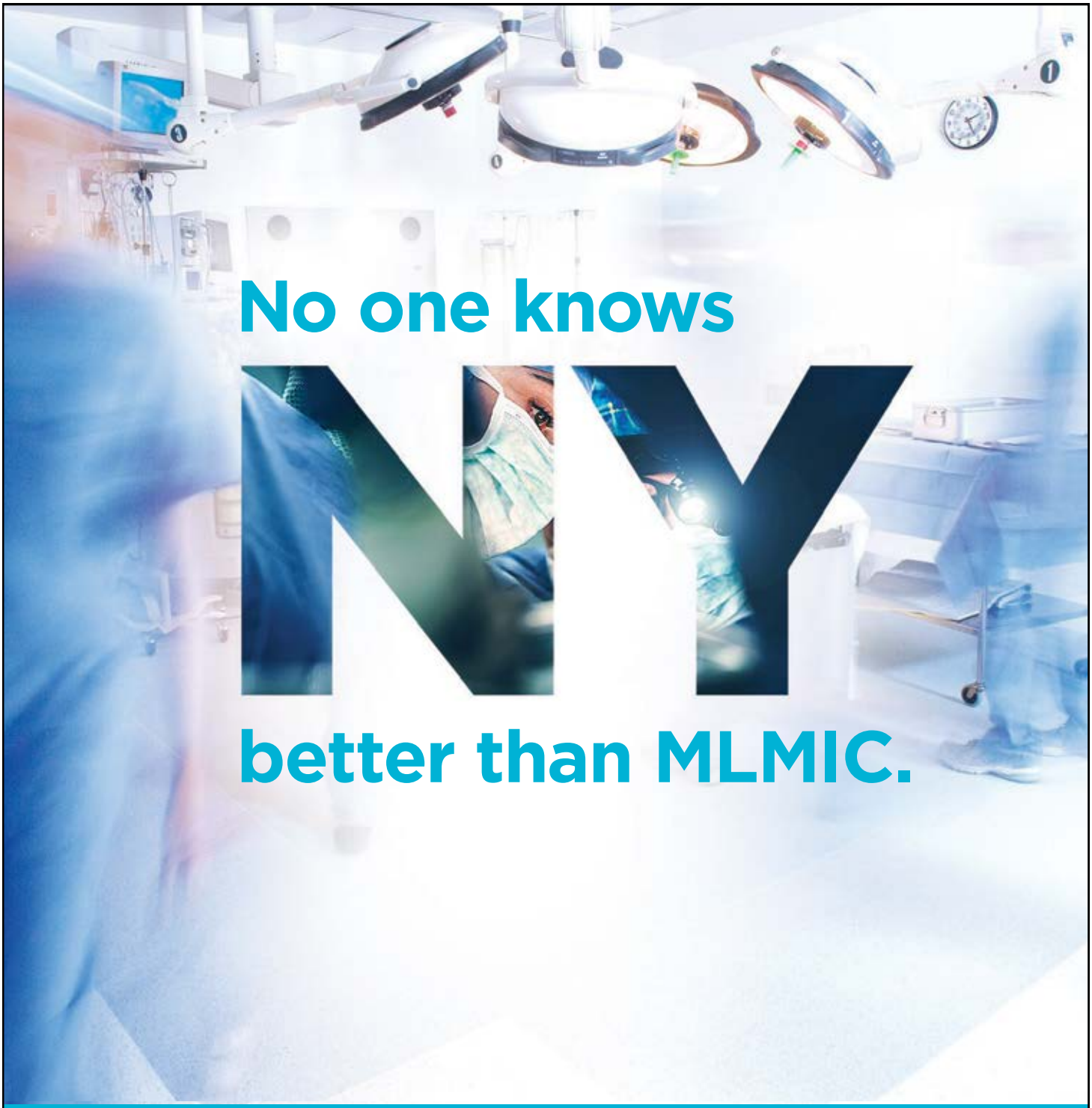

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- NYSAFP Membership Provides:
- Advancing our Specialty, Saving Members Time, Maximizing Values of our Dues
- Representation at the AAFP
- Representation of the local county chapters at the NYSAFP Congress of Delegates
- Promotion of family medicine in the medical schools and support of student programs
- Support of family medicine residency & fellowship training programs
- Representation of family medicine in the federal & state legislatures and policy makers through the PAC
- Saving Members Time
- Hosting of relevant and interactive CME workshops
- Hosting of ALSO instructor and provider courses
- Opportunity to interact with fellow family physicians throughout the state
- Reliable source of relevant and current events
- Weekly e-NewsBrief
- Quarterly peer reviewed journal – Family Doctor
- Timely access to current events of Academy via social media (NYSAFP Facebook | NYSAFP Twitter)
- Maximizing the Values of our Dues
- Sponsorship of students and residents to Academy meetings (Winter Weekend, Regional Family Medicine) and the Congress of Delegates
- Cultivation of the next generation of family physicians by offering scholarships and awards to pre-medical students, medical students, and residents to participate in family medicine conferences and programs
- Support of residents and new physicians in development of leadership skills and practice opportunities
- AAFP Member Services: http://www.aafp.org/online/en/home/membership/resources.html
- A list of the AAFP professional resources
- A list of the AAFP "Member Advantage"
- Additional Partnerships: http://www.nysafp.org/index/resources-6/partner-programs-106.html
- Jobs Board