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Diagnostic challenges in primary care: Identifying and avoiding cognitive bias

These 4 cases demonstrate how cognitive bias can impede the diagnostic process.

Medical errors in all settings contributed to as many as 250,000 deaths per year in the United States between 2000 and 2008, according to a 2016 study.[1] Diagnostic error, in particular, remains a leading cause of morbidity and mortality in the United States and worldwide. In 2017, 12 million patients (roughly 5% of all US adults) who sought outpatient care experienced missed, delayed, or incorrect diagnosis at least once.[2]

In his classic work, *How Doctors Think*, Jerome Groopman, MD, explored the diagnostic process with a focus on the role of cognitive bias in clinical decision-making. Groopman examined how physicians can become sidetracked in their thinking and "blinded" to potential alternative diagnoses.[3] Medical error is not necessarily because of a deficiency in medical knowledge; rather, physicians become susceptible to medical error when defective and faulty reasoning distort their diagnostic ability.[4]

Cognitive bias in the diagnostic process has been extensively studied, and a full review is beyond the scope of this article.[5] However, here we will examine pathways leading to diagnostic errors in the primary care setting, specifically the role of cognitive bias in the work-up of polymyalgia rheumatica (PMR), ovarian cancer (OC), Lewy body dementia (LBD), and fibromyalgia (FM). As these 4 disease states are seen with low-to-moderate frequency in primary care, cognitive bias can complicate accurate diagnosis. But first, a word about how to understand clinical reasoning.

There are 2 types of reasoning (and 1 is more prone to error)

Physician clinical reasoning can be divided into 2 different cognitive approaches.

Type 1 reasoning employs intuition and heuristics; this type is automatic, reflexive, and quick.[5] While the use of mental shortcuts in type 1 increases the speed with which decisions are made, it also makes this form of reasoning more prone to error.

Type 2 reasoning requires conscious effort. It is goal directed and rigorous and therefore slower than type 1 reasoning. Extrapolated to the clinical context, clinicians transition from type 2 to type 1 reasoning as they gain experience and training throughout their careers and develop their own conscious and subconscious heuristics. Deviations from accurate decision-making occur in a systematic manner due to cognitive biases and result in medical error.[6] **TABLE 1**[7] lists common types of cognitive bias.

An important question to ask. Physicians tend to fall into a pattern of quick, type 1 reasoning. However, it's important to strive to maintain a broad differential diagnosis and avoid premature closure of the diagnostic process. It's critical that we consider alternative diagnoses (ie, consciously move from type 1 to type 2 thinking) and continue to ask ourselves, "What else?" while working

TABLE 1

8 common cognitive errors and biases[7]

An assortment of cognitive biases has been compiled and described in the medical literature. Although there are at least 100 cognitive errors and biases identified, Morgenstern[7] has distilled them into the following 8.

1. Affective error: The tendency to convince oneself that what one *wants* to be true is true instead of a less appealing alternative. **Example**: A patient with a history of repeat migraines presents with a severe headache. The patient reports it is "the worst headache" she has had. Although the pain exceeds the patient's typical baseline for migraine, the doctor prescribes sumatriptan and sends the patient home. Later that night, the patient presents to the ED and is diagnosed with a subarachnoid hematoma.
2. Ambiguity effect: The tendency to select options or make a diagnosis for which the probability is known as opposed to selecting options for which the probability is unknown. **Example**: A patient presents with influenza-like symptoms that include fever, chills, and night sweats. The physician diagnoses the flu, while overlooking the patient's admission of recent high-risk sexual behavior, which may suggest that this is actually an initial presentation of acute HIV infection.
3. Ascertainment bias: When thinking is shaped by prior expectations. **Example**: A patient with a history of homelessness and alcohol dependence presents with stupor. The physician assumes that the stupor is due to alcohol use and neglects to check the patient's blood glucose level—even though the patient's chart indicates that he has type 2 diabetes. A diagnosis of severe hypoglycemia is overlooked.
4. Anchoring: Prematurely settling on a diagnosis based on a few important features of the initial presentation and failing to adjust the diagnosis as new information becomes available. **Example**: A patient with a history of osteoarthritis presents with new bilateral shoulder pain and immobility. The physician continues to treat the patient with NSAIDs, despite a lack of improvement and the presence of hip pain and immobility. The true diagnosis is polymyalgia rheumatica, which responds to corticosteroids, not NSAIDs.
5. Availability bias: The tendency to judge the likelihood of a disease by the ease with which relevant examples come to mind based on recent exposure. **Example**: After seeing 6 cases of influenza in the ED, the PCP considers the same diagnosis in another patient presenting with similar symptoms (plus shortness of breath), when in fact the patient has a pulmonary embolism. Despite the patient's shortness of breath, the PCP assumes the patient is yet another flu case.
6. Base-rate neglect: The failure to incorporate the true prevalence of a disease into diagnostic reasoning. **Example**: A PCP performs a full work-up for pulmonary embolism in all young low-risk patients with new-onset shortness of breath rather than first considering more common conditions, such as asthma or seasonal allergies.
7. Confirmation bias: After forming an opinion, the tendency to notice only confirmatory data and ignore data that fail to confirm the diagnosis. In some ways, this is similar to anchoring, but is different in that all new data that do not support the working diagnosis are ignored. **Example**: A patient presents to urgent care with shortness of breath and cough; lab results are positive for COVID-19 infection. The patient also has decreased breath sounds on the left side compared to the right. However, no chest X-ray is ordered, and the patient is sent home and told to come back to the ED if her symptoms worsen. She re-presents hours later to the ED and is diagnosed with left-side pneumothorax.
8. Premature closure: The tendency to stop too early in the diagnostic process before exploring the complete differential and all significant alternative diagnoses. **Example**: A PCP diagnoses and treats a 55-year-old woman with nausea, bloating, nonspecific abdominal pain, and early satiety for gastroesophageal reflux. The physician doesn't key in on the fact that the patient has had no success in treating her symptoms with a proton pump inhibitor or H_2 blocker and ignores mention of another physician who gave her a similar diagnosis. A diagnosis of ovarian cancer is missed.

NSAIDs, nonsteroidal anti-inflammatory drugs; PCP, primary care physician.

through differential diagnoses. This can be a powerful debiasing technique.

The discussion of the following 4 disease states demonstrates how cognitive bias can lead to diagnostic error.

CASE 1 ▶

An 82-year-old woman with a history of hypertension; wide-angle glaucoma; stage 2 chronic kidney disease; osteopenia; severe osteoarthritis (OA) affecting the hips, shoulders,

and knees; insomnia; and depression is transferred to a new family medicine practice for evaluation. She has been taking nonsteroidal anti-inflammatory drugs (NSAIDs) for chronic pain secondary to OA for 6 months, with no improvement in symptoms.

The patient is barely able to ambulate and appears to be in considerable pain. She is relying heavily on her walker and is assisted by her granddaughter. The primary care physician (PCP) obtains a detailed history that includes chronic shoulder and hip pain. Given that the patient has not responded to NSAID treatment over the previous 6 months, the PCP takes a moment to reconsider the diagnosis of OA and considers other options.

In light of the high prevalence of PMR in older women, the physician pursues a more specific physical examination tailored to ferret out PMR. He had learned this diagnostic shortcut as a resident, remembered it, and adeptly applied it whenever circumstances warranted. He asks the patient to raise her arms above her head (goalpost sign). She is unable to perform this task and experiences severe bilateral shoulder pain on trial. The PCP then places the patient on the examining table and attempts to assist her in rolling toward him. The patient is also unable to perform this maneuver and experiences significant bilateral hip pain on trial.

Based primarily on the patient's history and physical exam findings, the PCP makes a presumptive diagnosis of PMR vs OA vs combined PMR with OA, orders an erythrocyte sedimentation rate (ESR) and basic rheumatologic marker panel, and starts the patient on prednisone 10 mg/d. Lab work comes back and reveals mildly elevated ESR with all other findings within normal limits. Two weeks later, the patient returns for her follow-up visit, walking without a walker for the first time in years.

It's critical that we consider alternative diagnoses and continue to ask ourselves, "What else?" while working through differential diagnoses.

PMR can be mistaken for OA

PMR is the most common inflammatory rheumatic disease in older patients.[8] It is a debilitating illness with simple, effective treatment but has devastating consequences if missed or left untreated.[9] PMR typically manifests in patients older than age 50, with a peak incidence at 80 years of age. It is also far more common in women.[10]

Approximately 80% of patients with PMR initially present to their PCP, often posing a diagnostic challenge to many clinicians.[11] Due to overlap in symptoms, the condition is often misdiagnosed as OA, a more common condition seen by PCPs. Also, there are no specific diagnostic tests for PMR. An elevated ESR can help confirm the diagnosis, but one-third of patients with PMR have a normal ESR.[12] Therefore, the diagnostic conundrum the physician faces is OA vs rheumatoid arthritis (RA), PMR, or another condition.

The consequences of a missed and delayed PMR diagnosis range from seriously impaired quality of life to significantly increased risk of vascular events (eg, blindness, stroke) due to temporal arteritis.[13] Early diagnosis is even more critical as the risk of a vascular event and death is highest during initial phases of the disease course.[14]

FPs often miss this Dx. A timely diagnosis relies almost exclusively on an accurate, thorough history and physical exam. However, PCPs often struggle to correctly diagnose PMR. According to a study by Bahlas and colleagues,[15] the accuracy rate for correctly diagnosing PMR was 24% among a cohort of family physicians.

The differential diagnosis for PMR is broad and includes seronegative spondyloarthropathies, malignancy, Lyme disease, hypothyroidism, and both RA and OA.[16]

PCPs are extremely adept at correctly diagnosing RA, but not PMR. A study by Blaauw and colleagues[17] comparing PCPs and rheumatologists found PCPs correctly identified 92% of RA cases but only 55% of PMR cases. When rheumatologists reviewed these same cases, they correctly identified PMR and RA almost 100% of the time.[17] The difference in diagnostic accuracy between rheumatologists and PCPs suggests limited experience and gaps in fund of knowledge.

Making the diagnosis. The diagnosis of PMR is often made on empiric response to corticosteroid treatment, but doing so based solely on a patient's response is controversial.[18] There are rare instances in which patients with PMR fail to respond to treatment. On the other hand, some inflammatory conditions that mimic or share symptoms with

PMR also respond to corticosteroids, potentially resulting in erroneous confirmation bias.

Some classification criteria use rapid response to low-dose prednisone/prednisolone (≤ 20 mg) to confirm the diagnosis,[19] while other more recent guidelines no longer include this approach.[20] If PMR continues to be suspected after a trial of steroids is unsuccessful, the PCP can try another course of higher dose steroids or consult with Rheumatology.

CASE 2 ▶

A woman in her mid-40s presented to a PCP's office with a chief complaint of dyspepsia and bloating.[a] The patient was attending a meeting in New York City, and this was her first visit to this physician. The patient previously had been treated for these symptoms by her hometown PCP and gastroenterologist.

A full history and physical exam revealed a myriad of gastrointestinal (GI) complaints, such as diarrhea. But the PCP recalled a recent roundtable discussion on debiasing techniques specifically related to gynecologic disorders, including OC. Therefore, he decided to include OC in the differential diagnosis—something he would not routinely have done given the preponderance of GI symptoms. Despite the patient's reluctance and time constraints, the PCP ordered a transvaginal ultrasound. Findings from the ultrasound study revealed stage II OC, which carries a good prognosis. The patient is currently undergoing treatment and was last reported as doing well.

Early signs of ovarian cancer can be chalked up to a "GI issue"

OC is the second most common gynecologic cancer[21] and the fifth leading cause of cancer-related death[22] in US women. Compared to other cancers, the prognosis for localized early-stage OC is surprisingly good, with a 5-year survival rate approaching 93%.[23] However, most disease is detected in later stages, and the 5-year survival rate drops to a low of 29%.[24]

There remains no established screening protocol for OC. Fewer than a quarter of all cases are diagnosed in stage I, and detection of OC relies heavily on the physician's ability to decipher vague symptomatology that overlaps with other, more common maladies. This poses an obvious diagnostic challenge and, not surprisingly, a high level of susceptibility to cognitive bias.

More than 90% of patients with OC present with some combination of the following symptoms prior to diagnosis: abdominal (77%), GI (70%), pain (58%), constitutional (50%), urinary (34%), and pelvic (26%).[25] The 3 most common isolated symptoms in patients with OC are abdominal bloating, decrease in appetite, and frank abdominal pain.[26] Patients with biopsy-confirmed OC experience these symptoms an average of 6 months prior to actual diagnosis.[27]

Knowledge gaps play a role. Studies assessing the ability of health care providers to identify presenting symptoms of OC reveal specific knowledge gaps. For instance, in a survey by Gajjar and colleagues,[28] most PCPs correctly identified bloating as a key symptom of OC; however, they weren't as good at identifying less common symptoms, such as inability to finish a meal and early satiety. Moreover, survey participants misinterpreted or missed GI symptoms as an important manifestation of early OC disease.[28] These specific knowledge gaps combine with physician errors in thinking, further obscuring and extending the diagnostic process. The point prevalence for OC is relatively low, and many PCPs only encounter a few cases during their entire career.[29] This low pre-test probability may also fuel the delay in diagnosis.

Watch for these forms of bias. Since nonspecific symptoms of early-stage OC resemble those of other more benign conditions, a form of anchoring error known as *multiple alternatives bias* can arise. In this scenario, clinicians investigate only 1 potential plausible diagnosis and remain focused on that single, often faulty, conclusion. This persists despite other equally plausible alternatives that arise as the investigation proceeds.[28]

Affective error may also play a role in missed or delayed diagnosis. For example, a physician would prefer to diagnose and treat a common GI illness than consider OC. Another distortion involves *outcome bias*

Most PCPs correctly identified bloating as a key symptom of ovarian cancer; however, they weren't as good at identifying less common symptoms, such as inability to finish a meal.

[a] This was an actual case reported to the lead author (PDR) during a conference.

TABLE 2

Examples of cognitive biases that affect ovarian cancer diagnosis[7]

Affective error: Since ovarian cancer is a devastating condition, the physician would prefer to diagnose and treat a common GI illness and may not even consider ovarian cancer in the differential diagnosis.
Availability bias: Since more benign conditions such as irritable bowel syndrome are seen more frequently than ovarian cancer in the clinical setting, they are favored. This is also a form of outcome bias.
Anchoring: The patient presents with nonspecific symptoms of early ovarian cancer, including bloating and early satiety. The physician focuses on dietary choices and fails to explore GI or genitourinary conditions that may explain early satiety.
Premature closure: The physician orders unhelpful and expensive tests in an attempt to characterize the incorrect suspected GI pathology, then prematurely terminates the diagnostic process and accepts the most favored diagnosis. In addition, sunk-cost fallacy taints the decision process, making it difficult to abandon the presumed diagnosis, as the physician invests more and more time and effort in the incorrect diagnostic path.

GI, gastrointestinal.

wherein the physician gives more significance to benign conditions such as irritable bowel syndrome because they have a more favorable outcome and clear treatment path. Physicians also favor these benign conditions because they encounter them more frequently than OC in the clinic setting. (This is known as *availability bias*.) Outcome bias and multiple alternatives bias can result in noninvestigation of symptoms and inefficient or improper management, leading to a delay in arriving at the correct diagnosis or anchoring on a plausible but incorrect diagnosis.

An incorrect initial diagnostic path often triggers a cascade of subsequent errors. The physician orders additional unhelpful and expensive tests in an effort to characterize the suspected GI pathology. This then leads the physician to prematurely terminate the work-up and accept the most favored diagnosis. Lastly, *sunk-cost fallacy* comes into play: The physician has "invested" time and energy investigating a particular diagnosis and rather than abandon the presumed diagnosis, continues to put more time and effort in going down an incorrect diagnostic path.

A series of failures. These biases and miscues have been observed in several studies. For example, a survey of 1725 women by Goff and colleagues[30] sought to identify factors related to delayed OC diagnosis. The authors found that the following factors were significantly associated with a delayed diagnosis: omission of a pelvic exam at initial presentation, a separate exploration of a multitude of collateral symptoms, a failure to order ultrasound/computed tomography/CA-125 test, and a failure to consider age as a factor (especially if the patient was outside the norm).

Responses from the survey also revealed that physicians initially ordered work-ups related to GI etiology and only later considered a pelvic work-up. This suggests that well-known presenting signs and symptoms or a constellation of typical and atypical symptoms of OC often failed to trigger physician recognition. Understandably, patients presenting with menorrhagia or gynecologic complaints are more likely to have OC detected at an earlier stage than patients who present with GI or abdominal signs alone.[31] **TABLE 2**[7] summarizes some of the cognitive biases seen in the diagnostic path of OC.

CASE 3 ▶

A 56-year-old man is brought to the ED by his wife and children for evaluation of odd behavior and episodes of confusion. The patient recently had a negative neurologic work-up for transient ischemic attack and cerebrovascular accident and is admitted for further work-up. He reports visual hallucinations to nursing staff. Screening for memory problems shows no significant deficits. The patient in fact scored a 27 on the Mini–Mental State Examination, well within the normal range. The family notes that the patient has had difficulty with planning over the previous year and has

not seemed like his "old self." The patient has no history of psychosis, schizophrenia, bipolar disorder, or any other psychiatric illness.

While in the hospital, he becomes acutely upset by the hallucinations and is given haloperidol and lorazepam by house staff. In the morning, the patient exhibits severe signs of Parkinson disease that include rigidity and masked facies.

Given the patient's poor response to haloperidol and continued confusion, the team consulted Neurology and Psychiatry. Gathering a more detailed history from the patient and family, the patient is given a diagnosis of classic LBD. The antipsychotic medications are stopped. The patient and his family receive education about LBD treatment and management, and the patient is discharged to outpatient care.

Psychiatric symptoms can be an early "misdirect" in cases of Lewy body disease

LBD, the second leading neurodegenerative dementia after Alzheimer disease (AD), affects 1.5 million Americans,[32] representing about 10% of all dementia cases. LBD and AD overlap in 25% of dementia cases.[33] In patients older than 85 years, the prevalence jumps to 5% of the general population and 22% of all cases of dementia.[33] Despite its prevalence, a recent study showed that only 6% of PCPs correctly identified LBD as the primary diagnosis when presented with typical case examples.[32]

3 stages of presentation. Unlike other forms of dementia, LBD typically presents *first* with psychiatric symptoms, *then* with cognitive impairment, and *last* with parkinsonian symptoms. Additionally, rapid eye movement sleep behavior disorder and often subtle elements of nonmemory cognitive impairment distinguish LBD from both AD and vascular dementia.[32] The primary cognitive deficit in LBD is not in memory but in attention, executive functioning, and visuospatial ability.[34] Only in the later stages of the disease do patients exhibit gradual and progressive memory loss.

Mistaken for many things. When evaluating patients exhibiting signs of dementia, it's important to include LBD in the differential, with increased suspicion for patients experiencing episodes of psychosis or delirium. The uniqueness of LBD lies in its psychotic symptomatology, particularly during earlier stages of the disease. This feature helps distinguish LBD from both AD and vascular dementia. As seen in the case, LBD can also be confused with acute delirium.

Older adult patients presenting to the ED or clinic with visual hallucinations, delirium, and mental confusion may receive a false diagnosis of schizophrenia, medication- or substance-induced psychosis, Parkinson disease, or delirium of unknown etiology.[35] Unfortunately, LBD is often overlooked and not considered in the differential diagnosis. Due to underrecognition, patients may receive treatment with typical antipsychotics. The addition of a neuroleptic to help control the psychotic symptoms causes patients with LBD to develop severe extrapyramidal symptoms and worsening mental status,[36] leading to severe parkinsonian signs, which further muddies the diagnostic process. In addition, treatment for suspected Parkinson disease, including carbidopa-levodopa, has no benefit for patients with LBD and may increase psychotic symptoms.[37]

First-line treatment for LBD includes psychoeducation for the patient and family, cholinesterase inhibitors (eg, rivastigmine), and avoidance of high-potency antipsychotics, such as haloperidol. Although persistent hallucinations and psychosis remain difficult to treat in LBD, low-dose quetiapine is 1 option. Incorrectly diagnosing and prescribing treatment for another condition exacerbates symptoms in this patient population.

CASE 4 ▶

A 36-year-old Hispanic woman presents to the PCP for her annual physical exam. The patient's medical record shows 2 previous office visits over the past 2 years—an annual physical exam and an office visit for the flu vaccine. The patient is highly accomplished in her profession, working as a certified public accountant for a major corporation. She is a nonsmoker and reports only casual social drinking and no recreational drug use. The patient is slightly overweight for her height but is otherwise healthy. Previous lab studies are within normal limits.

CONTINUED

The uniqueness of Lewy body dementia lies in its psychotic symptomatology, particularly during earlier stages of the disease.

The patient has been experiencing chronic pain for the past few years after a motor vehicle accident. She has seen a physiatrist and another provider, both of whom found no "objective" causes of her chronic pain. They started the patient on sertraline for depression and an analgesic, both of which were ineffective.

The patient likes to exercise at a gym twice a week by doing light cardio (treadmill) exercise and light weightlifting. Lately, however, she has been unable to exercise due to the pain. At this visit, she mentions having low energy, poor sleep, frequent fatigue, and generalized soreness and pain in multiple areas of her body. The PCP recognizes the patient's presenting symptoms as significant for FM and starts her on pregabalin and hydrotherapy, with positive results.

Patients with fibromyalgia, who are often otherwise healthy, often present multiple times to the same PCP with a chief complaint of chronic pain.

Fibromyalgia skepticism may lead to a Dx of depression

FM, the second most common disorder seen in rheumatologic practice after OA, is estimated to affect approximately 1 in 20 patients (approximately 5 million Americans) in the primary care setting.[38,39] The condition has a high female-to-male preponderance (3.4% vs 0.5%).[40] While the primary symptom of FM is chronic pain, patients commonly present with fatigue and sleep disturbance.[41] Comorbid conditions include headaches, irritable bowel syndrome, and mood disturbances (most commonly anxiety and depression).

Several studies have explored reasons for the misdiagnosis and underdiagnosis of FM. One important factor is ongoing skepticism among some physicians and the public, in general, as to whether FM is a real disease. This issue was addressed by a study by White and colleagues,[42] who estimated and compared the point prevalence of FM and related disorders in Amish vs non-Amish adults. The authors hypothesized that if litigation and/or compensation availability have a major impact on FM prevalence, then there would be a near zero prevalence of FM in the Amish community. And yet, researchers found an overall age- and sex-adjusted FM prevalence of 7.3% (95% CI; 5.3%-9.7%); this was both statistically greater than zero ($P < .0001$) and greater than 2 control populations of non-Amish adults (both $P < .05$).

Many physicians consider FM fundamentally an emotional disturbance**,** and the high preponderance of FM in female patients may contribute to this misconception as reports of pain and emotional distress by women are often dismissed as hysteria.[43] Physicians often explore the emotional aspects of FM, incorrectly diagnosing patients with depression and subsequently treating them with a psychotropic drug.[39] Alternatively, they may focus on the musculoskeletal presentations of FM and prescribe analgesics or physical therapy, both of which do little to alleviate FM.

To make the correct diagnosis of FM, the American College of Rheumatology created a specific set of criteria in 1990, which was updated in 2010.[44] For a diagnosis of FM, a patient must have at least a 3-month history of bilateral pain above and below the waist and along the axial skeletal spine. Although not included in the updated 2010 criteria, many clinicians continue to check for tender points, following the 1990 criteria requiring the presence of 11 of 18 points to make the diagnosis.

At least 3 cognitive biases relating to FM apply: anchoring, availability, and *fundamental attribution error* (see **TABLE 3**).[7] Anchoring occurs when the PCP settles on a psychiatric diagnosis of exaggerated pain syndrome, muscle overuse, or OA and fails to explore alternative etiology. Availability bias may obscure the true diagnosis of FM. Since PCPs see many patients with RA or OA, they may overlook or dismiss the possibility of FM. Attribution error happens when physicians dismiss the complaints of patients with FM as merely due to psychological distress, hysteria, or acting out.[43]

Patients with FM, who are often otherwise healthy, often present multiple times to the same PCP with a chief complaint of chronic pain. These repeat presentations can result in compassion fatigue and impact care. As Aloush and colleagues[40] noted in their study, "FM patients were perceived as more difficult than RA patients, with a high level of concern and emotional response. A high proportion of physicians were reluctant to accept them because they feel emotional/psychological difficulties meeting and coping with these patients." In response, pa-

TABLE 3

Examples of cognitive biases that affect fibromyalgia diagnosis[7]

Ambiguity effect: The physician is more familiar with the presentation of depression than with the uncertainty of fibromyalgia. Thus, the physician gives the patient a diagnosis of depression for her complaints of poor sleep, anxiety, and chronic pain rather than spending more time exploring the possibility of fibromyalgia.
Ascertainment bias: A physician who sees many patients with somatic manifestations of depression is convinced that he or she is quite adept at treating depression in young professional women and overlooks the possibility that this patient's complaints of fatigue, widespread pain, poor sleep, and poor concentration are actually consistent with (and suggestive of) fibromyalgia.
Anchoring: The physician refuses to adjust a diagnosis of depression even though the patient continues to experience pain after receiving physical therapy and taking the antidepressants prescribed for her.
Availability bias: A physician diagnoses and treats a patient for depression but misses the chronic pain component of the presentation.
Base-rate neglect: The high prevalence of fibromyalgia is relatively unknown to the physician and thus, the physician diagnoses depression in that patient because he or she *is* aware of the prevalence of depression.

tients with undiagnosed FM or inadequately treated FM may visit other PCPs, which may or may not result in a correct diagnosis and treatment.

We can do better

Primary care physicians face the daunting task of diagnosing and treating a wide range of common conditions while also trying to recognize less-common conditions with atypical presentations—all during a busy clinic workday. Nonetheless, we should strive to overcome internal (eg, cognitive bias and fund-of-knowledge deficits) and external (eg, time constraints, limited resources) pressures to improve diagnostic accuracy and care.

Each of the 4 disease states we've discussed have high rates of missed and/or delayed diagnosis. Each presents a unique set of confounders: PMR with its overlapping symptoms of many other rheumatologic diseases; OC with its often vague and misleading GI symptomatology; LBD with overlapping features of AD and Parkinson disease; and FM with skepticism. As gatekeepers to health care, it falls on PCPs to sort out these diagnostic dilemmas to avoid medical errors. Fundamental knowledge of each disease, its unique pathophysiology and symptoms, and varying presentations can be learned, internalized, and subsequently put into clinical practice to improve patient outcomes. JFP

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