



FOR PERPLEXITY HEALTH

Clinical Reasoning Case Study.

A reusable patient profile and five multi-turn physician reasoning chains, exported as structured JSON for evaluation and training.

KNOWLEDGE BOUNDARY · SHARED ACROSS ALL FIVE CHAINS

The patient knows his past medical history, past surgical history, family history, medications, and allergies. The patient knows he had a CT chest in Florida during his hospital admission but thinks that the results only showed pneumonia. He is not aware that there are calcifications in his aorta, which would be an additional indication to stay on apixaban in addition to having atrial fibrillation. In addition, there is medication nonadherence—the patient only takes his statin a few times a month rather than daily as instructed.

SYNTHETIC

NO PHI

PHYSICIAN-AUTHORED

1 PATIENT

5 CHAINS

36 TURNS

What Perplexity gets from this format.

CLINICAL REASONING SANDBOX

One synthetic patient profile branches into five multi-turn clinical reasoning traces. Each trace shows the user-facing encounter, the physician's reasoning at each turn, and the relevant chart context, exported as structured JSON for evaluation and training.

- 01 Internal monologue per turn.**
Every physician response ships with the reasoning that produced it. The thinking that would otherwise stay in the doctor's head, recorded as inspectable bullets next to the encounter.
- 02 Physician-authored quality.**
Every chain is reviewed and signed off by a practicing physician. Case writeup, conversation arc, and reasoning bullets included.
- 03 Branching diversity from one patient.**
Five different conversations from the same chart. The reasoning forks based on the question itself, capturing the variation real users generate.
- 04 Multi-turn by default.**
Eight turns per chain on average. Captures how reasoning evolves through the back-and-forth as new information surfaces.
- 05 Safety boundaries per case.**
Each case has explicit non-negotiables and failure modes calibrated to the clinical scenario. Case-specific guardrails the model can be scored against.
- 06 Inspectable trace records.**
Every chain exports to structured JSON: patient profile, initial question, knowledge boundary, turn-by-turn encounter, reasoning bullets, and safety boundaries. Filter, export, route into evals or training workflows.

The same chart underlies every chain.

(1/3) · Identity, history & medications

SEX	AGE	RACE	MARITAL	WORK	ALLERGIES
Male	50	White	Married	Plumber	None known

History

Past medical Asthma · Hypertension · Hyperlipidemia · Paroxysmal atrial fibrillation · Pneumonia 1 year ago

Past surgical None

Family Mother – Hypertension · Father – Hypertension, Type 2 diabetes · Maternal grandfather – Alzheimer’s disease

Allergies No known drug allergies

Medications

Amlodipine
5 mg daily

Apixaban
5 mg twice daily

Metoprolol
25 mg daily

Valsartan
80 mg daily

Atorvastatin
40 mg daily

The same chart underlies every chain. (2/3) · Vitals & chemistry trend

Vitals

5/4/26	Blood pressure 135/84 · Heart rate 78 · Height 170.2 cm · Weight 95 kg
11/1/25	Blood pressure 148/79 · Heart rate 70
5/15/25	Blood pressure 159/90 · Heart rate 80 · Height 171 cm · Weight 85 kg

Chemistry

5/4/26	Sodium 141 · Potassium 4.8 · Chloride 107 · BUN 20 · CO ₂ 24 · Creatinine 1.23 · eGFR 72 · Glucose 100 · Anion gap 10
11/1/25	A1c 6.2
5/15/25	Sodium 142 · Potassium 5.3 · Chloride 107 · BUN 16 · CO ₂ 26 · Creatinine 1.16 · eGFR 77 · Glucose 107 · Anion gap 9
4/12/24	Sodium 141 · Potassium 4.0 · Chloride 105 · BUN 17 · CO ₂ 25 · Creatinine 1.23 · eGFR 72 · Glucose 108 · Anion gap 11 · A1c 5.6

The same chart underlies every chain. (3/3) • Snapshot Labs & records

Lipid panel • 5/4/26

Total cholesterol	245
HDL	42
LDL	172
non-HDL	203
Triglycerides	166

Complete blood count • 4/12/24

White cells	5.93	MCH	27.1
Red cells	5.8	MCHC	34.4
Hemoglobin	15.7	Platelets	264
Hematocrit	45.7	MPV	10.6
MCV	78.8	RDW	13.1

Records

Last clinic note • 5/4/26	Routine cardiology visit for hypertension follow-up after amlodipine was started for elevated blood pressures. Patient reports taking apixaban for one month after being hospitalized in Florida for pneumonia, where atrial fibrillation was incidentally found.
Prior cardiology	Two prior cardiology visits for hypertension workup and initiation of antihypertensive therapy.
Florida hospitalization • 1 month ago	Admitted for pneumonia while on vacation. CT chest diagnosed pneumonia and incidentally revealed aortic calcifications . Atrial fibrillation incidentally identified. Discharged on apixaban. <i>The patient has not been told about the aortic calcifications.</i>
Asthma history	Patient has history of asthma that started since childhood but flares when he gets sick or exercises.

Every turn, six buckets of reasoning.

SCHEMA

Each physician reasoning entry is tagged with one of six buckets. Empty buckets are omitted on a turn; a repeated prefix means a continuation within the same bucket.

NI

New information

What I just observed or heard this turn — the factual update from the patient or chart.

EXAMPLE • CHAIN 1 • TURN 5

"Medication nonadherence confirmed. Reason given: fear of muscle pain (patient is a runner)."

UT

Updated thinking

How that information shifts my picture — the synthesis between observation and stance.

EXAMPLE • CHAIN 1 • TURN 5

"Fear-driven, not symptom-driven. Different intervention than if he'd had real myalgia."

OU

Open uncertainty

Gaps that remain in my picture, whether or not I'm acting on them this turn.

EXAMPLE • CHAIN 1 • TURN 4

"Is the uncontrolled LDL an adherence problem or a pharmacology problem?"

NA

Next-action rationale

Why I'm asking this specific next question or making this specific next move.

EXAMPLE • CHAIN 1 • TURN 4

"'When did you start this medication?' is a neutral opener that surfaces adherence without front-loading judgment."

WH

Working hypothesis

Where I currently stand — my best stance on the clinical picture or treatment direction.

EXAMPLE • CHAIN 1 • TURN 1

"With CHA₂DS₂-VASc score of 2, that confers an annual stroke risk of ~2.2%, and meets class I indication for AC."

CM

Commentary

Pathophysiology, guidelines, scoring systems — background knowledge being brought to bear.

EXAMPLE • CHAIN 1 • TURN 3

"Atherosclerosis is the substrate that ultimately can lead to a myocardial infarction, or heart attack colloquially."

Bleeding fear about a newly started blood thinner. (1/4)

PATIENT ENCOUNTER

Turn 0

PATIENT I was in the hospital a few weeks ago and they told me I have atrial fibrillation and started me on blood thinners. I saw my cardiologist today and he told me to continue taking it, but I heard that this can cause bleeding. Should I continue taking these?

PHYSICIAN You are likely taking blood thinners due to the risk of stroke associated with atrial fibrillation. You are correct that blood thinners increase your risk of bleeding. Whether you should continue taking these is a nuanced question that balances the benefit of preventing stroke vs the risk of bleeding. Did your doctor explain to you why you should be on it?

Turn 1

PATIENT My cardiologist said that atrial fibrillation increases the risk of clots and that I need to take a blood thinner indefinitely.

PHYSICIAN Your doctor likely used a score called CHA₂DS₂-VASc to estimate your risk of a stroke. In your case, you have a diagnosis of HTN, giving you a score of 1. Looking through your chart, I noticed that from your CT chest done in Florida, it was noticed that you have calcifications in the large blood vessel in your body (aorta). This gives you a total score of 2. Based on clinical guidelines, there is a strong indication to be on blood thinners like apixaban in men for scores > 1, which applies to you. Have you noticed any issues with bleeding?

PHYSICIAN INTERNAL REASONING

OU: Whether the cardiologist explained the stroke-prevention indication for AC.

NA: Asking 'did your doctor explain why you should be on it?' separates a knowledge gap (need to teach the indication) from a fear-only concern (need to reinforce a known indication).

WH: An individual's risk of thrombosis is weighted against their risk of bleeding with taking anti-coagulation.

CM: AFib leads to blood stasis in the left atrium, which can eventually form a clot; if the clot travels elsewhere with systemic blood flow, that can lead to thromboembolic disease — for example, a clot traveling to the brain causes a stroke; a clot traveling to a leg can cause critical limb ischemia. Anticoagulation prevents clot formation and greatly reduces the risk of stroke associated with AFib.

NI: Cardiologist explained the stroke-prevention indication for AC and that it is indefinite.

UT: Not an indication-understanding gap — the concern is specifically the bleeding side of the tradeoff. Next moves: make the stroke-benefit side concrete with his actual CHA₂DS₂-VASc score, then assess the bleeding side.

NA: However, it is important to also ask about bleeding risk since that is the major adverse effect of taking an anti-coagulant.

WH: With CHA₂DS₂-VASc score of 2, that confers an annual stroke risk of ~2.2%, and meets class I indication for AC.

CM: CHA₂DS₂-VASc is the most popular way to estimate clotting risk that is also recommended by guidelines, which awards 1 point for heart failure, 1 point for HTN, 1-2 points depending on age, 1 point for diabetes, 2 points for stroke/systemic thromboembolism, 1 point for vascular disease, and 1 point for female sex. Patients with higher CHA₂DS₂-VASc scores have estimated higher risk of stroke, which is measured in terms of annual stroke risk.

For example, even a CHA₂DS₂-VASc score of 1 confers an estimated annual stroke risk of ~1.3% in patients who have AFib.

People sometimes have calcifications that can be seen or incidentally reported on prior CT scans which reflect vascular disease. However, unless that is explicitly investigated, it is usually not noticed and accidentally omitted from the scoring.

Bleeding fear about a newly started blood thinner. (2/4)

PATIENT ENCOUNTER

Turn 2

PATIENT They did a CT chest because I had pneumonia and told me everything else was normal. I've never had any abnormal bleeding in the past.

PHYSICIAN It sounds like the CT chest was done to answer a clinical question of whether you had pneumonia; the calcifications seen in the aorta were reported by the radiologist as an incidental finding. They may not have thought about the significance of that finding down the line for you. That is great you don't have bleeding! Do you have issues with balance or frequent falls?

Turn 3

PATIENT What do calcifications in the aorta mean for me? Is this dangerous? No balance issues or falls.

PHYSICIAN It shows that your blood vessels are going through a process of developing plaque, and some have developed in your arteries. They are not dangerous at the moment but means you are at increased risk of developing a heart attack. Regarding your blood thinner, if you have not had recent balance issues or falls, I don't see a contraindication to you taking it.

PHYSICIAN INTERNAL REASONING

- NI:** No history of abnormal bleeding.
Patient unaware of the aortic calcifications on the Florida CT.
- UT:** Most important historical bleeding screen is negative. The unknown-calcifications situation is exactly the case the score-undercounting principle describes.
- NA:** Even though the patient said "no bleeding," he could still be at risk for future disabling bleeding — most importantly, a fall with head strike.
- CM:** A patient's CHA₂DS₂-VASc is usually underestimated due to accidental omission of risk factors that contribute to the score. This could lead to critical branchpoints in decision making, such as whether to start anti-coagulation. Ensuring comprehensive review of medical history to obtain an accurate CHA₂DS₂-VASc score is essential.

- NI:** No balance issues or falls.
- UT:** Bleeding side of the tradeoff is now closed — AC decision is settled.
- WH:** Aortic atherosclerosis suggests the same process is likely developing in his coronary arteries, increasing his risk for MI.
Patient has no clear contraindications to anticoagulation for AFib.
- CM:** Atherosclerosis is the substrate that ultimately can lead to a myocardial infarction, or heart attack colloquially.

Bleeding fear about a newly started blood thinner. (3/4)

PATIENT ENCOUNTER

Turn 4

PATIENT Should I be taking anything for this? Are there any activities or foods, etc. I should avoid given that I have atrial fibrillation and also these calcifications?

PHYSICIAN It is important to be on a heart healthy diet, which means food rich in vegetables, non-red meat like fish or chicken, olive oil, fruit, and non-processed carbs like whole grains. Furthermore, exercising at a moderate level a few times a week that brings up your heart rate and makes you feel like you're working hard, but not too hard is recommended. Outside of this, lowering your cholesterol is the best strategy for the plaque in your arteries. I see in your profile that you are already taking a statin – atorvastatin 40 mg daily, which is the main treatment for this. However it is concerning that your “bad” cholesterol level (LDL-C) is still very high at 172. When did you start this medication?

Turn 5

PATIENT My cardiologist started me on it a year ago, but I have not been taking it regularly, probably a few times a month. I heard that it causes muscle pain, and I'm a runner, so I've been avoiding taking it daily.

PHYSICIAN Muscle pains can be a side effect of statins, but have you actually noticed muscle pains when you take it?

PHYSICIAN INTERNAL REASONING

NI: Chart: atorvastatin 40 mg daily already prescribed; LDL-C 172.

UT: LDL is way off target for a high-intensity statin (expected 30–50% drop). Most parsimonious explanation is nonadherence, not a wrong drug/dose.

OU: Is the uncontrolled LDL an adherence problem or a pharmacology problem?

NA: ‘When did you start this medication?’ is a neutral opener that surfaces adherence without front-loading judgment.

WH: Based on the PREVENT-ASCVD equation, the patient's estimated 10-year risk of ASCVD is 7% (intermediate risk). That would support lowering LDL to < 100. However, the presence of aortic atherosclerosis suggests that his atherosclerosis is more advanced than pure primary prevention, supporting an LDL goal of < 70.

Despite being on high dose statin, the patient's LDL-C is still uncontrolled, raising concern about medication non-adherence.

CM: Diet and lifestyle are first line modifications to reduce cardiovascular risk. Guidelines recommend a Mediterranean style diet composed of plant base foods, healthy fats, and lean protein, minimizing red meat, processed foods, and added sugars.

Reducing cholesterol provides best evidence for preventing future MI across numerous clinical trials.

In general, there are no true “adverse effects” to LDL being as low as possible whereas the benefit is maximal prevention of further development of atherosclerosis.

NI: Medication nonadherence confirmed.

Reason given: fear of muscle pain (patient is a runner); started a year ago, taken only a few times a month.

UT: Fear-driven, not symptom-driven. Different intervention than if he'd had real myalgia.

OU: Has he actually experienced muscle pain or only heard about it as a risk?

NA: Asking whether he has actually experienced muscle pain distinguishes fear of a side effect from a real one — the two lead to opposite recommendations (reassurance vs. switching agents).

Bleeding fear about a newly started blood thinner. (4/4)

PATIENT ENCOUNTER

Turn 6

PATIENT I have not had any muscle pain.

PHYSICIAN I see, I would recommend taking the atorvastatin 40 mg daily as prescribed. If you notice muscle pains, you can always let your doctor know and he can try a different medication.

Turn 7

PATIENT Is there anything else I should do?

PHYSICIAN yes, in summary, take your blood thinner (apixaban), take your cholesterol lowering medication (atorvastatin), and make an appointment with your cardiologist in 6 weeks where they can recheck your cholesterol level once you've been on the atorvastatin for a sufficient amount of time to see its full effect.

PHYSICIAN INTERNAL REASONING

NI: No muscle pains or other side effects.

UT: Confirms fear-driven nonadherence — no real myalgia ever. Recommendation can be unambiguous.

WH: Given no muscle pains or other side effects, this means no clear contraindications to statins, so guidelines recommend being on it.

The target LDL-C level would be < 100 or ideally < 70, which may be achievable given atorvastatin 40 mg is a high intensity statin that is estimated on average to reduce a patient's LDL-C by 30-50%.

WH: Three-part close: (1) continue apixaban (AC indication intact, no bleeding/fall contraindications); (2) take atorvastatin daily as prescribed (no real myalgia, LDL target < 70); (3) follow up with cardiology in 6 weeks for a repeat lipid panel.

After starting (or restarting at full adherence on) a statin, it usually takes up to 6 weeks to see its full effect on cholesterol lowering, so obtaining a repeat lipid panel in 6 weeks is recommended.

A1c went up. Should I be concerned? (1/3)

PATIENT ENCOUNTER

Turn 0

PATIENT I just noticed that the A1c number recently went up; the cardiologist didn't comment on it, but should I be concerned?

PHYSICIAN Your bloodwork from 11/1/25 does show an A1c of 6.2%, a rise from 5.6% in 4/12/24. This indicates that you now have a diagnosis of pre-diabetes. While it does not mean you have diabetes, it means that you are at increased risk of developing diabetes and your body is having more difficulty managing its sugar levels.

Turn 1

PATIENT Do you know why my cardiologist didn't mention it going up?

PHYSICIAN Your cardiologist probably did not recognize the number as high enough to have a diagnosis of diabetes. He/she may have given recommendations for dietary changes or lifestyle changes.

Turn 2

PATIENT Yes, in fact my cardiologist did refer me to a nutritionist and advised me to exercise more. What does the A1c lab mean?

PHYSICIAN The HbA1c test measures the average blood sugar levels over the past 2-3 months based on the amount of hemoglobin (protein found in red blood cells that transfers oxygen) with sugar attached to it. When the A1c number is higher, that means there's a higher percentage of hemoglobin with sugar attached. When A1c is between 5.7% to 6.4%, that gives a diagnosis of prediabetes, placing you at higher risk for diabetes. A1c of 6.5% and above gives a diagnosis of diabetes.

PHYSICIAN INTERNAL REASONING

NI: A1c rose from 5.6% (4/12/24) to 6.2% (11/1/25). Patient noticed; cardiologist did not comment.

UT: Crosses the prediabetes threshold (5.7–6.4%) — new diagnostic category for him.

WH: Given A1c of 6.2, the patient now has new diagnosis of prediabetes.

Given his risk profile of HTN, HLD, and obesity, this is not unexpected.

WH: The cardiologist was probably either focused on other aspects of the patient's care, had limited time to address everything, didn't notice the increase, or did so by giving the main guidance, which is dietary and lifestyle modification.

CM: The main treatment for prediabetes is dietary and lifestyle optimization and reducing associated risk factors for cardiometabolic disease.

NI: Patient confirms cardiologist already gave standard prediabetes advice (nutritionist referral, exercise).

UT: Cardiologist did the right thing — confirms the working hypothesis from prior turn.

CM: A1c reflects average blood glucose over the prior 2–3 months, measured as the percentage of hemoglobin with glucose attached. Diagnostic cutoffs: 5.7–6.4% prediabetes; $\geq 6.5\%$ diabetes.

A1c went up. Should I be concerned? (2/3)

PATIENT ENCOUNTER

Turn 3

PATIENT Why is diabetes (or high A1c) bad?

PHYSICIAN Diabetes is a chronic condition where the body is unable to properly use sugar (glucose) in the blood effectively. This leads to higher glucose levels in the blood, which the body is not accustomed to and leads to damage long term. Almost all organs are affected – high blood sugar damages the blood vessels, the kidneys, nerves, and eyes.

Turn 4

PATIENT That's alarming - do you know why I am now at greater risk of diabetes?

PHYSICIAN There are many reasons why your A1c went up. I notice that your weight has also increased, which can explain why your A1c has as well. Have you recently had changes in your diet or other factors going on in your life?

PHYSICIAN INTERNAL REASONING

CM: Diabetes is a chronic condition where the body cannot use glucose effectively. The resulting elevated blood glucose damages tissues over time — almost all organs are affected, with classic targets being blood vessels, kidneys, nerves, and eyes.
The long-term damage from high blood sugar is a fact that many in the lay public may not realize.

NI: Patient now alarmed by the diagnosis.
Chart on review: weight 85 kg (11/1/25) → 95 kg (5/4/26) — recent gain.

UT: Modest A1c rise is most parsimoniously explained by weight gain → insulin resistance — not by a new pancreatic process.

OU: What's actually driving the weight gain — diet change, life event, something else?

NA: Open-ended question about diet and life events — surfaces a modifiable driver of the weight gain without leading. Diet, work stress, sleep, medications are all on the table at this point.

WH: In this patient, the modest increase is not cause for alarm i.e. red flags. Based on this patient's profile of a 50-year-old male with many risk factors, becoming pre-diabetic is not surprising.

CM: Obesity is closely linked to prediabetes (and diabetes) and there is a likely causative role where excess fat triggers metabolic changes leading to insulin resistance, leading to increased risk of diabetes.

A1c went up. Should I be concerned? (3/3)

PATIENT ENCOUNTER

Turn 5

PATIENT Yes, recent work has been very stressful so I have been eating out a lot – could that explain it and if so, could this be reversed?

PHYSICIAN If you've been eating an unhealthier diet that can explain both the weight gain and increase in A1c. By discussing with a nutritionist as recommended by your cardiologist, they can help you find food options that keep you healthy. The A1c is definitely reversible if you make these changes.

Turn 6

PATIENT Could you summarize what we talked about for me?

PHYSICIAN Yes, your A1c was found to rise from 5.7% to 6.2%, putting you in the pre-diabetes range, which means you are at increased risk of developing diabetes. Diabetes is a chronic disease caused by high sugar levels in your blood, leading to damage to many different organs in your body. Starting with lifestyle interventions is the first step in treating pre-diabetes and preventing diabetes.

PHYSICIAN INTERNAL REASONING

NI: Cause confirmed: work stress → eating out a lot, driving the weight gain → A1c rise. Patient also asks whether the rise is reversible.

UT: Behavioral, reversible driver — not pancreatic disease. Frame for the patient: yes, this can be reversed.

WH: Since his cardiologist already recommended that he see a nutritionist, they are able to walk through all the food that the patient eats and identify those that they would recommend to replace with healthier alternatives.

CM: Close by emphasizing that diabetes is a chronic disease requiring long-term intervention, starting with diet and lifestyle as the first step in treating prediabetes and preventing progression.

Should I be on a GLP-1? (1/3)

PATIENT ENCOUNTER

Turn 0

PATIENT Should I be on a GLP1?

PHYSICIAN You are probably referring to GLP-1 agonists, which are a class of medications that was initially used as a treatment for diabetes, however recently has become popular after demonstrating tremendous efficacy for weight loss. Are you thinking about GLP1 from the diabetes or weight loss aspect?

Turn 1

PATIENT Weight loss

PHYSICIAN GLP1 agonists are effective at weight loss; I do see that you have gained weight recently. Have you identified reasons why you could have gained weight recently?

Turn 2

PATIENT Yes, I recently had a lot of stress at work, so I order out a lot and am overeating. I am supposed to see a nutritionist per my cardiologist, but it's not scheduled yet.

PHYSICIAN Yes, dietary and lifestyle changes are the first step to addressing weight gain. GLP1 may help if those changes are not sufficient.

PHYSICIAN INTERNAL REASONING

NI: The patient is asking about GLP-1 receptor agonists.

OU: Is the patient asking from the diabetes side or the weight-loss side?

NA: However, to start the conversation would want to get more information into the reason why the patient is asking this question.

WH: The patient is probably asking due to the publicity these medications have had at reducing weight.

NI: Patient confirms weight-loss framing.

UT: Question is aligned with chart context (recent weight gain) — coherent ask, but driver-of-weight-gain matters before committing to GLP-1.

OU: What's driving the weight gain — modifiable or not?

NA: Try to discern the reasons why the patient would be wondering about GLP1/weight loss at this time.

NI: Stress-driven overeating identified as the driver. Cardiologist referred to nutritionist but appointment not yet scheduled.

UT: Behavioral, reversible cause — same driver as the A1c rise. GLP-1 is not first-line in this picture.

CM: Diet and lifestyle are first-line for weight gain; GLP-1 receptor agonists are an adjunct, useful when behavioral change alone is insufficient.

Should I be on a GLP-1? (2/3)

PATIENT ENCOUNTER

Turn 3

PATIENT I worry diet isn't enough – who can prescribe a GLP1?

PHYSICIAN Many physicians are able to prescribe GLP-1 agonists. The first step would be discussing this with your primary care physician.

Turn 4

PATIENT What should I watch out for before starting GLP1?

PHYSICIAN GLP-1 agonists are contraindicated for patients who have a history of a specific type of thyroid cancer (medullary thyroid carcinoma) or a rare syndrome called MEN2, or prior allergies to GLP1 medications. In addition, people who previously had pancreatitis or prior gastrointestinal issues need to be careful and should speak to their doctor at length before starting.

Turn 5

PATIENT Would insurance coverage be an issue for me?

PHYSICIAN It highly depends on your insurance, however based on your history, the main indication is for obesity, and most insurances would not cover based on this indication alone. However, since you also have pre-diabetes, high blood pressure, and high cholesterol, those may be comorbidities with obesity that could be covered depending on the insurance. There could be additional qualifying criteria for you - for example, when you sleep at night, do you happen to notice significant snoring or waking up from sleep?

PHYSICIAN INTERNAL REASONING

NI: Patient worried diet won't be enough; asks who can prescribe.

UT: Give the practical answer without arguing against his ask — lifestyle stays the foundation.

CM: The PCP is usually the first line physician who can prescribe a GLP-1 RA. Alternative physicians include cardiologists, endocrinologists, or weight loss specialists.

CM: GLP-1 RA contraindications — absolute: history of medullary thyroid carcinoma, MEN2 syndrome, prior allergy to GLP-1 medications. Relative cautions: prior pancreatitis or prior gastrointestinal issues — those patients should speak to their doctor at length before starting.

UT: Patient has multiple comorbidities — better chance for coverage than obesity alone. OSA, if present, would be another lever and a separate diagnosis worth making.

OU: Does the patient have OSA? Pre-test probability is high given obesity.

NA: Obstructive sleep apnea (OSA) can be a strong co-morbidity leading to insurance coverage, which would also be important to diagnose given the adverse health implications from OSA.

CM: Obesity is usually not an insurance coverable indication for GLP1, a commonly asked question. However, obesity with associated co-morbidity may lead to eligibility with some insurances.

Should I be on a GLP-1? (3/3)

PATIENT ENCOUNTER

Turn 6

PATIENT I do actually, it's gotten worse recently

PHYSICIAN It is quite likely you have obstructive sleep apnea (OSA), which is commonly seen with obesity. This is caused by tissues around the throat relaxing and causing temporary obstruction of breathing during sleep. It is an important diagnosis to make because it can lead to many adverse health consequences including increased risk of high blood pressure, heart disease and stroke. It may also lead to insurance coverage for a GLP-1 agonist.

Turn 7

PATIENT what should be my next steps

PHYSICIAN Speak to your physician about these symptoms and start by inquiring about a sleep study, which would make the diagnosis of OSA. The main treatment for OSA would be a CPAP machine to wear at night, in addition to treating the underlying driver (obesity). You may also ask about this in the context of obesity and getting on a GLP-1.

Turn 8

PATIENT Thank you

PHYSICIAN INTERNAL REASONING

NI: Patient confirms worsening snoring/sleep disturbance.

UT: OSA now likely — high pre-test probability + confirmatory symptom. Diagnosis should be pursued.

WH: OSA is an important diagnosis to make in patients with obesity given its high pre-test probability.

CM: OSA leads to long-term adverse health consequences including HTN, heart disease, and stroke. It is also treatable with CPAP at night, but also important to address the underlying drivers (i.e. obesity).

CM: Practical OSA workup: discuss symptoms with the PCP and request a sleep study to confirm the diagnosis. If positive, CPAP at night is first-line treatment; addressing the underlying driver (obesity) runs in parallel. The GLP-1 question can be re-raised in that context.

Will pneumonia damage my lungs? (1/2)

PATIENT ENCOUNTER

Turn 0

PATIENT I had pneumonia last month – will this damage my lungs?

PHYSICIAN To answer this question, I need to know more about the pneumonia you had last month - can you tell me the circumstances of that?

Turn 1

PATIENT Yes, I developed fevers and chills while vacationing in Florida so went to the hospital emergency room. They gave me the diagnosis after a chest X-ray. I was given antibiotics for a few days and eventually got better.

PHYSICIAN I see, did your symptoms completely resolve and/or do you feel any residual symptoms right now?

Turn 2

PATIENT Yes, they resolved after a week. Right now I am just worried that the pneumonia caused irreversible damage to my lungs.

PHYSICIAN Assuming a straightforward case of pneumonia that is now fully treated, your lungs should make a full recovery. There may be some residual effects but if you don't feel any symptoms and this was a one-time episode, I would not be concerned.

PHYSICIAN INTERNAL REASONING

OU: Severity, treatment, and course of the pneumonia — uncomplicated CAP vs something more complex?

NA: There is incomplete information based on a simple patient question, so need to first obtain more details before giving a complete response.

NI: Typical acute pneumonia course described: fevers/chills, ER visit, CXR diagnosis, antibiotics, recovery.

UT: Picture is consistent with uncomplicated CAP — pending confirmation of full symptom resolution.

NA: Verify that the presumed pneumonia was effectively treated.

CM: If there are any residual symptoms, that would suggest that what the patient had was more complex than an uncomplicated pneumonia presentation 1 month ago.

NI: Symptoms resolved within a week.

UT: Clean, fast resolution — confirms uncomplicated CAP, treated. Patient's underlying worry is about long-term lung damage.

WH: Based on information thus far, the patient seems to have had a straightforward episode of pneumonia and is now asymptomatic. That is reassuring for a good prognosis.

Will pneumonia damage my lungs? (2/2)

PATIENT ENCOUNTER

Turn 3

PATIENT That's good to know – what are situations where I should be concerned?

PHYSICIAN If you keep having persistent symptoms, that may suggest that you did not have a simple pneumonia that has been completely treated, and instead there is something else going on or you have difficulty clearing the infection. In addition, if people have recurrent episodes of pneumonia, that also suggests another process going on and increases the likelihood of long-term lung damage.

Turn 4

PATIENT Since I got pneumonia, do you think I am more likely to get it again?

PHYSICIAN If this is your only lifetime episode of pneumonia, it would be unlikely that you have a pre-disposition for pneumonia. In contrast, people who develop prolonged episodes or multiple episodes may have a predisposition that requires further workup.

Turn 5

PATIENT Is there anything I can do to prevent getting pneumonia again?

PHYSICIAN Good hygiene practices like frequent hand-washing with soap and water, staying away from sick contacts, ensuring adequate sleep, eating a healthy diet and exercise, will keep you healthy and prevent acquiring an infection like pneumonia. However, there is nothing that can completely prevent you from contracting pneumonia.

PHYSICIAN INTERNAL REASONING

CM: Red flags include persistent symptoms or repeated episodes that could suggest an underlying immunodeficiency, either congenital or acquired (like HIV).

In addition, alternative diagnoses such as bronchitis or viral infections may be mistaken for pneumonia and follow a more prolonged course, but would not respond as effectively to a course of antibiotics.

WH: Given pneumonia is an infectious disease and in the absence of predisposing factors, it would be unlikely to have greater predisposition for pneumonia down the line if the patient already made a full recovery.

CM: In contrast, whenever someone has pneumonia, it is important to ensure the patient has not had repeated episodes, which could point towards an underlying “red flag” predisposition such as immunodeficiency (e.g. HIV or inherited) or structural abnormality (e.g. malignancy).

CM: Pneumonia prevention is general infection control: frequent hand-washing with soap and water, staying away from sick contacts, adequate sleep, a healthy diet, and exercise — these keep the patient healthy and prevent acquiring an infection like pneumonia. Nothing can completely prevent contracting pneumonia.

Trouble breathing with exercise. Asthma, or the heart? (1/3)

PATIENT ENCOUNTER

Turn 0

PATIENT I am trying to lose weight by exercising but my asthma keeps flaring up during exercise. What do I do?

PHYSICIAN I see you have a diagnosis of asthma in your medical history; however, it is important to verify that your symptoms are due to asthma and not a different medical reason. What symptoms do you actually experience when you exercise?

Turn 1

PATIENT I feel like I have trouble breathing when I try to exercise.

PHYSICIAN Has this always happened when you exercise or is this new? What do you do for exercise and how much can you do?

PHYSICIAN INTERNAL REASONING

OU: What is his actual symptom? 'Asthma' is a label patients apply broadly.

NA: However, it is important to verify the diagnosis before giving recommendations, since other can't-miss diagnoses can be attributed to "asthma".

CM: Exercise-induced asthma is a real entity.

NI: Symptom is 'trouble breathing' on exertion.

UT: Dyspnea (not specifically wheeze) — compatible with asthma but also cardiac/pulmonary causes.

OU: Onset (new vs lifelong) and severity (functional impact).

NA: Understanding the severity and temporal nature of his symptoms is important to make an accurate diagnosis.

WH: While exercise-induced asthma could explain his symptoms, it would be unexpected to be new onset at his age.

Trouble breathing with exercise. Asthma, or the heart? (2/3)

PATIENT ENCOUNTER

Turn 2

PATIENT This only started happening a few months ago. Before, when I exercised, I was fine. I usually walk on a treadmill, before I could walk a mile but now, I have trouble catching my breath after half a mile.

PHYSICIAN I see, have you tried using an inhaler and is it effective?

Turn 3

PATIENT I have, but for some reason it does not make much of a difference.

PHYSICIAN I see, there are many reasons for people to develop trouble breathing with exercise, and it may not be because of asthma. Have you noticed any recent chest pain or pressure or leg swelling?

PHYSICIAN INTERNAL REASONING

NI: New onset few months ago. Progressive functional decline: 1 mile walking → 0.5 mile.

UT: New-onset progressive dyspnea at 50 — pattern is more consistent with progressive cardiac/pulmonary disease than asthma.

OU: Empirical treatment-response: does his inhaler help?

NA: Asking additional further confirmatory questions is essential to distinguish between causes of shortness of breath — given his history of asthma, he may already have an inhaler which can be the treatment, so understanding whether the treatment is empirically effective is helpful for the diagnosis.

WH: This is starting to not sound like exercise-induced asthma.

NI: Inhaler doesn't help.

UT: Asthma is effectively off the table. Reframe to 'dyspnea on exertion' with a wide differential.

OU: Cardiac vs pulmonary cause — need targeted screening for can't-miss diagnoses.

NA: Chest pain/pressure targets angina; leg swelling targets right-sided/biventricular heart failure — one sentence, two diagnoses.

WH: Recent-onset difficulty breathing with exertion plus lack of inhaler response makes exercise-induced asthma unlikely.

However, there are many can't-miss diagnoses for "dyspnea on exertion" — which is a more accurate description of his symptom.

CM: Alternative diagnoses include both pulmonary and cardiac causes — the differential includes ILD, COPD, heart failure, angina, and deconditioning. Asking key questions can help differentiate between these diagnoses, although a more comprehensive assessment requires in-person evaluation.

Trouble breathing with exercise. Asthma, or the heart? (3/3)

PATIENT ENCOUNTER

Turn 4

PATIENT I do notice some chest pressure and my legs have been swollen recently. I always have bad veins thought so it's probably that.

PHYSICIAN Based on what you're describing, the trouble breathing with exercise may not be due to asthma, but rather other conditions related to your heart and lungs. Have you also noticed any trouble breathing lying flat?

Turn 5

PATIENT Yes, more so than usual. I thought it was my GERD.

PHYSICIAN I worry that this may reflect an issue with your heart and recommend you get evaluated in person as soon as possible. Please reach out to your cardiologist about these symptoms you are describing.

PHYSICIAN INTERNAL REASONING

NI: Chest pressure and recent leg swelling reported (patient attributes to 'bad veins').

UT: Dyspnea + chest pressure + bilateral edema — cardiac causes (HF or angina) become the leading differential.

OU: Heart failure vs angina vs both — orthopnea would weight HF.

NA: Orthopnea ('breathing lying flat') is highly specific for HF — one-sentence screen to distinguish HF from angina-only.

WH: The differential shifts to cardiac conditions like heart failure or angina as the cause of his dyspnea on exertion.

CM: In his demographic (50M) and risk factors (HTN, HLD, prediabetes, obesity), those are more common.

Attributing lower extremity edema to "bad veins" is common and patients may feel that it's normal.

NI: Orthopnea confirmed (trouble breathing lying flat, worse than usual).

UT: Syndromic presentation: dyspnea + chest pressure + bilateral edema + orthopnea. No longer a chat-level question — needs urgent in-person evaluation.

NA: No more history needed at this stage — next move is escalation to in-person evaluation, not another question.

WH: His constellation of symptoms — dyspnea on exertion, chest pressure, bilateral lower-extremity edema, orthopnea — is most concerning for recent-onset heart failure vs. angina, both can't-miss diagnoses that should be evaluated in person by a physician.