



WESTERN MONTANA ENDODONTICS
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The Endo Files: Summer 2026

Bayes and the Power of the “Prior”

To support our referral partners, I would like to continue our series of clinical updates on modern endodontics. Let's start this installation with a diagnostic mystery:

A 60-year-old male developed a radiolucency in the lower right quadrant (Figure 1). He was initially evaluated elsewhere with a suspected vertical root fracture (VRF). A subsequent surgical biopsy showed no visible fracture, and pathology confirmed an apical granuloma. The patient was then referred to our office to evaluate the underlying endodontic etiology.



Figure 1

When the patient presented to us, he was asymptomatic. #29 felt slightly different to percussion; the other teeth in the quadrant were not sensitive; #28 and 29 responded to cold testing. Here is a summary of the additional findings from our initial consultation:

There were four indications that #29 could be necrotic and was the culprit of the lucency:

- PA and CBCT imaging (Figure 1c and 1d) demonstrated an apical lucency that seemed *mostly* centered on #29, but it bordered the mesial root of #30 as well.
- CBCT imaging also suggested the presence of at least one lateral canal in #29 that communicated with the lucency.
- #29 felt slightly different to percussion (#28, 30, and 31 were not sensitive).
- The PDL was uniform *apically* on #30.

There were other indications that #29 was not necrotic and #30 might be the source of the lucency:

- #29 had no visible cracks, caries, or previous restorations. The probability of this pulp becoming necrotic was low.
- #28, 29, and 31 responded to cold testing predictably. It was possible that residual nerve tissue in #29 is still responding, giving us a “false” result.
- #30 had previous endodontic treatment, making it a higher-risk suspect for persistent pathology.

What would you recommend for this patient?

When teaching biology in high school, organic chemistry in college, or endodontic diagnosis in dental school, concepts are often taught in neat, clean categories. These categories are likely necessary during the learning process to provide a framework for applying the freshly learned concepts. However, clinical dentistry exists in a world of uncertainty and gray areas. This can be especially true for diagnosis, where we have to make decisions based on incomplete, incongruous, and/or conflicting information.

Shortly after dental school, a few of my mentors introduced me to **Bayes’ Theorem**—a decision-making framework that uses probabilities to calculate the likelihood of a diagnosis based on all available evidence. While it stems from a mathematical formula, you don’t need math to use it.

At its core, Bayesian thinking describes how to rationally update our diagnostic confidence as new information comes in:

- **Prior Probability:** How likely is the condition *before* testing, based on baseline prevalence, patient history, and the radiographic evidence?
- **Test Likelihood:** How accurate or decisive is the diagnostic test we just ran?
- **Posterior Probability:** Our updated, real-time likelihood of the diagnosis *after* combining the test result with the prior probability.

The key insight is that a test result never exists in a vacuum—its true meaning depends heavily on how common or rare the pathology was *before* the test was performed.

One comforting part of Bayesian theory is that all of us, as clinicians, are inherently good at determining the “prior probability” of an event; however, the key takeaway from this article is utilizing an awareness of this **prior**, weighing it correctly, and deploying it to avoid diagnostic traps.

Let’s see how these concepts work in clinical scenarios. A 44 year old female presented to our office with a complaint of a dark anterior tooth and a “bump on my gums” (Figure 2). She stated that someone elbowed her front teeth while playing basketball years ago. Before testing the tooth in this scenario, what are the **priors**? Previous trauma, a dark anterior tooth, and a sinus tract. These dramatically increase the **prior probability** that this patient has a necrotic tooth.

After taking a radiograph, you notice an apical lucency and a calcified canal associated with #25. In Bayesian terms, this new evidence is our **likelihood**, and factoring it in updates our baseline to a much higher **posterior probability** that the tooth is necrotic.

But we don't stop there. This **posterior probability** now becomes our **new prior** as we move to clinical testing. When we perform a cold test and get zero response, this clinical data acts as a secondary update, shifting our probability across the finish line to a definitive, mathematically sound diagnosis of pulp necrosis with chronic apical abscess. The tooth was treated endodontically and internally bleached (Figure 2).

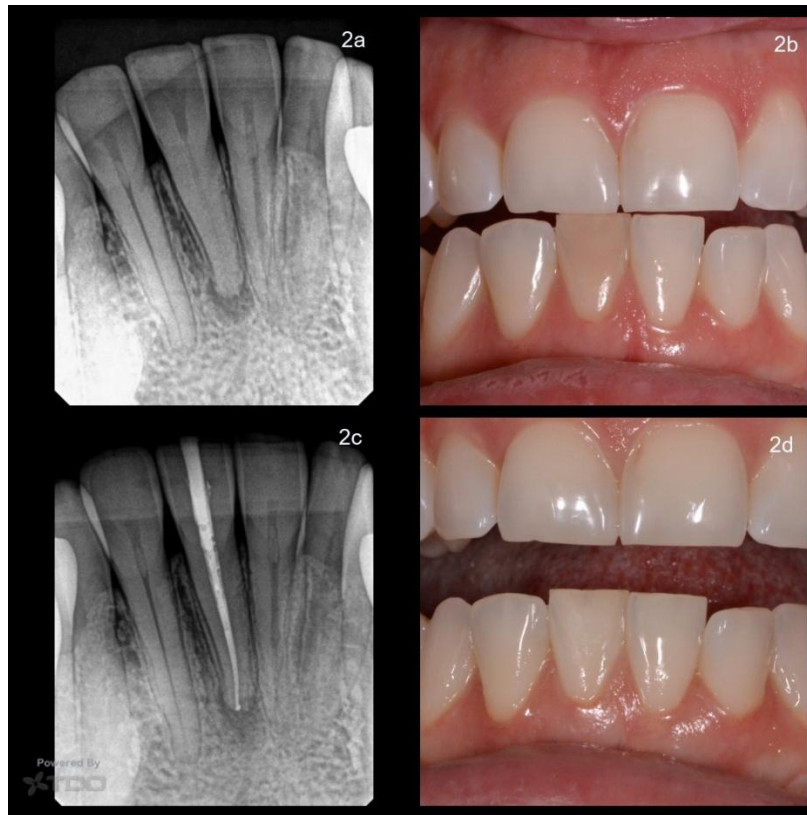


Figure 2

Let's look at another more nuanced scenario. A patient presented with aching pain on the upper right; she pointed to the right masseter area and right zygomatic process. She stated that she has a history of headaches, and her discomfort is worse in the morning.

Establishing the Priors:

Without testing information or radiographs, her **prior probability** of myofascial pain is high: a general aching pain, pain in the muscles of mastication, and pain in the morning (suggesting nighttime parafunction). Before taking a radiograph or picking up a cold spray, her baseline **prior probability for myofascial pain is very high**.

Updating with Radiographs:

Let's update our **prior** with more information: the radiographs demonstrate restorations that are not in close proximity to the pulps (Figure 3). The probability of an endodontic issue continues to drop, since there are no visible caries and no deep restorations—though a cracked tooth remains a remote possibility, and can sometimes occur in cases with shallow restorations.



Figure 3

Updating with Clinical Testing:

Let's update our **prior** again: a clinical exam did not reveal evidence of significant cracks. Endodontic testing yielded ambiguous results: moderate percussion tenderness on both #3 and #4, along with heightened, slightly lingering cold sensitivity across #2, #3, and #4 without a single focal culprit.

The Bayesian Takeaway:

Evaluated in a vacuum, those vague cold and percussion findings might tempt a clinician to access #3 or #4. But when we weigh those murky results against our strong baseline for myofascial pain, the **posterior probability** shifts decisively toward non-odontogenic origin.

Because the **prior probability** of muscle-driven pain was so high to begin with, ambiguous endodontic testing carries very little diagnostic weight. In fact, in a scenario like this—as in the previous example in this article (Figure 2)—running additional endo tests (like repeating cold testing or applying an EPT) won't meaningfully alter our **posterior probability** or cross a treatment threshold. Further EPT testing would just add noise to a picture where we need to lean on **priors**.

One of my mentors (John Khademi) likes to say “*you can't test your way out of a tough diagnosis.*” Before picking up a cold pellet or EPT, analyze the history, clinical context, and radiographs. Establish the baseline possibilities *first*.

Clinical context—patient history, symptom clusters, and baseline pathology risk—must always anchor the interpretation of any diagnostic test. Isolated findings separated from **prior probability** routinely lead to overdiagnosis or missed diagnoses.

Now, let's revisit our opening mystery. What is your diagnosis? Here is how updating our probabilities led us to the right answer:

1. Baseline Prior: Radiographically, #29 was unrestored and did not have caries (*Very Low Prior* for necrosis). #30 has prior endodontic treatment (*decent prior* for persistent infection).
2. Clinical Updates & Test Likelihood: Clinically, #29 had no visible cracks and responded predictably to cold. The posterior probability that #29 was necrotic became vanishingly small. Neither the potential lateral canal on CBCT nor the slight sensation to percussion on #29 was strong enough to push us over a treatment threshold.
3. Posterior Probability: Despite the subtle indications pointing toward #29, the Bayesian posterior probability overwhelmingly favored #30 as the true etiology.

We selectively retreated the mesial root of #30 over multiple visits with calcium hydroxide. The apical lucency resolved, leaving #29 untouched and vital (Figure 4).

Try to avoid making a diagnosis based on testing alone. Weigh the accuracy of the testing after taking into account the patient history and radiographs. We live and work in a world of uncertainty; I hope Bayes' theorem can assist you with future diagnoses and treatment planning.

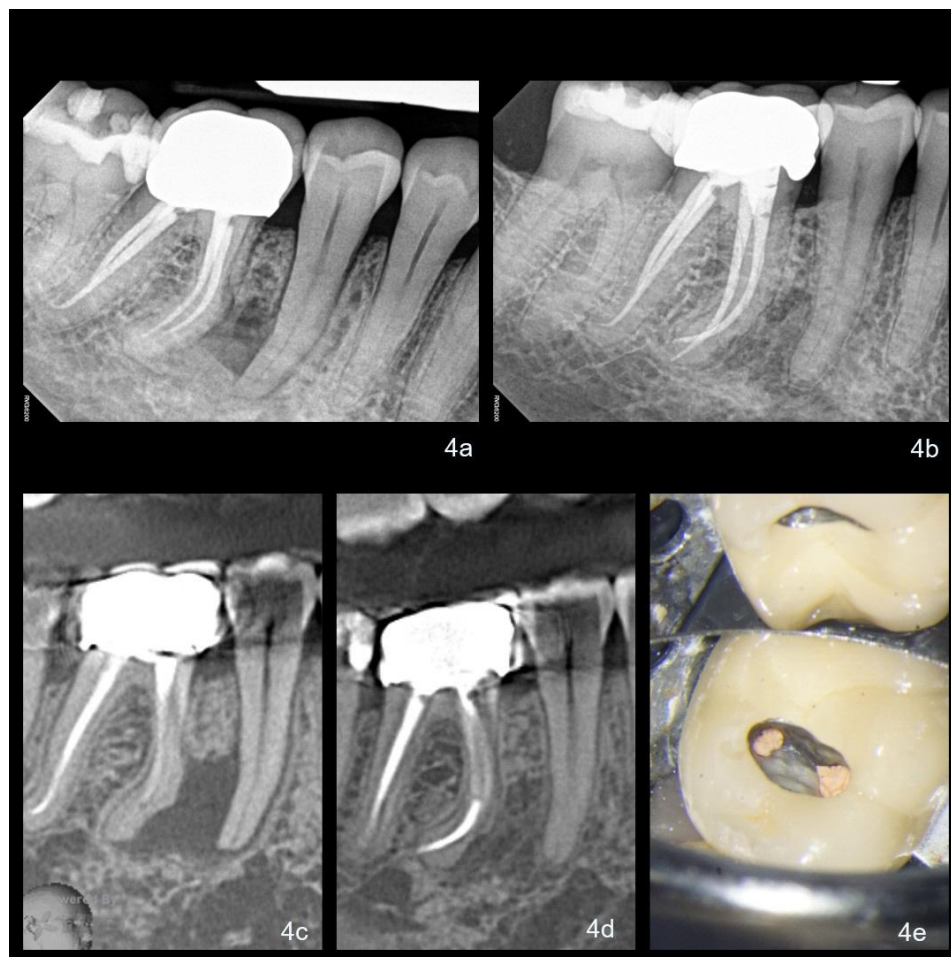


Figure 4. 4a: pre-op PA. 4b: post-op PA after selective retreatment of the mesial root of #30. 4c: pre-op CBCT. 4d: CBCT after 5 months of calcium hydroxide in the mesial root of #30. 4e: photo of obturation of mesial canals #30.