TARSAL TUNNEL SYNDROME AND PES CAVUS AND PES PLANUS

Good morning, Consultant Family Physician.

From the orthopedic, podiatric, and foot-and-ankle literature, the answer is:

Pes planus (flatfoot) is a well-recognized predisposing factor for Tarsal Tunnel Syndrome (TTS).

Pes cavus (high-arched foot) is not considered a typical or classic cause of TTS, although it may occasionally contribute through altered biomechanics or associated neurological disorders. (PMC)

Why Pes Planus Causes Tarsal Tunnel Syndrome

The tarsal tunnel lies beneath the flexor retinaculum on the medial side of the ankle. In pes planovalgus:

- The hindfoot everts.
- The medial longitudinal arch collapses.
- The tibial nerve and its branches are stretched and compressed.
- The volume of the tunnel may effectively decrease.

This increases pressure on the posterior tibial nerve and predisposes to entrapment neuropathy. Modern reviews consistently identify **flatfoot deformity (especially pes planovalgus)** as a biomechanical risk factor for TTS. (NCBI)

In clinical practice, many foot-and-ankle surgeons specifically look for:

- Acquired adult flatfoot
- Posterior tibial tendon dysfunction
- Valgus hindfoot deformity

when evaluating patients with suspected TTS.

What About Pes Cavus?

Pes cavus is a different biomechanical problem:

- High medial arch
- Hindfoot varus
- Increased lateral loading
- Relative foot rigidity

Classically it is associated with:

- Metatarsalgia
- Recurrent ankle sprains

- Peroneal tendon disorders
- Stress fractures
- Neurological diseases such as Charcot–Marie–Tooth disease

TTS is **not generally listed as a typical manifestation of pes cavus** in major reviews. (PMC)

However, there are exceptions:

1. Severe cavovarus deformity can alter nerve tension.
2. Neurological disorders causing pes cavus (especially Charcot-Marie-Tooth disease) may produce neuropathic symptoms that mimic or coexist with TTS.
3. Individual case reports describe TTS in cavus feet, but this is not considered a classic association. (PMC)

If Asked in an Orthopedic Examination

A concise evidence-based answer would be:

Foot Deformity	Association with Tarsal Tunnel Syndrome
Pes planus / Pes planovalgus	Strong and well-recognized association
Pes cavus	Weak, uncommon, not a typical feature
Hindfoot valgus	Strong association
Posterior tibial tendon dysfunction	Strong association

My Interpretation as a Medical Informatician Reviewing the Literature

If a patient presents with:

- Burning plantar pain
- Medial ankle paresthesia
- Positive Tinel sign at the tarsal tunnel
- Flatfoot deformity

then the flatfoot should be considered a significant etiological clue and not merely an incidental finding.

Conversely, if a patient has pes cavus and symptoms suggestive of TTS, I would be inclined to search carefully for:

- an underlying neurological disorder,
- a space-occupying lesion,
- trauma,

- ganglion cyst,
- varicosities,
- accessory muscles,

before attributing the neuropathy solely to the cavus foot. (PMC)

As an aside, there is an interesting and clinically important relationship between **adult acquired flatfoot due to posterior tibial tendon dysfunction** and TTS that is often underappreciated. The literature on that specific relationship is stronger than many clinicians realize. If you are reviewing a patient with foot pain and nerve symptoms, that pathway deserves particular attention.

That is a very interesting observation, Consultant Family Physician, and it immediately raises several possibilities.

Based on the orthopedic and neurological literature, **unilateral pes planus on one side and pes cavus on the opposite side is unusual enough that I would hesitate to regard it as a simple anatomical variant**, particularly when the cavus foot is the symptomatic one.

My thinking would proceed along the following lines.

1. The Tarsal Tunnel Syndrome May Be Unrelated to the Cavus Foot

The simplest explanation is that the patient has:

- incidental pes cavus on one side,
- incidental pes planus on the other side,
- and TTS caused by a local factor in the cavus foot.

In such a patient, one should actively search for:

- ganglion cyst
- varicose veins within the tunnel
- lipoma
- accessory muscle
- previous ankle trauma
- tenosynovitis
- inflammatory arthropathy

Many TTS cases are actually caused by local compressive lesions rather than foot morphology alone.

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2. Cavovarus Foot May Increase Tibial Nerve Tension

Although not considered a classical cause of TTS, a cavovarus foot can theoretically increase traction

on the tibial nerve.

In a cavus foot:

- the hindfoot often assumes varus,
- the foot becomes more rigid,
- ankle biomechanics are altered,
- neural excursion during gait may be reduced.

This mechanism is biomechanically plausible but much less well documented than the pes planovalgus-TTS relationship.

In other words, the cavus deformity may not directly compress the nerve but may predispose it to chronic traction or irritation.

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3. Consider an Underlying Neurological Disorder

As a clinician, this would be my first "red flag."

Pes cavus is one of the classic orthopedic clues to underlying neurological disease.

The list includes:

- hereditary motor sensory neuropathies, especially Charcot–Marie–Tooth disease
- old poliomyelitis
- spinal dysraphism
- tethered cord syndrome
- cerebral palsy
- previous nerve injuries
- lumbosacral radiculopathy

A neuropathy can:

1. produce a cavus deformity over time,
2. make the tibial nerve more vulnerable to compression,
3. mimic TTS clinically.

Therefore I would want nerve conduction studies to be certain the patient truly has focal TTS and not a more generalized neuropathic process.

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4. Could the Pes Planus and Pes Cavus Be Compensatory?

This is a fascinating biomechanical possibility.

Occasionally, after:

- old trauma,
- limb length discrepancy,
- unilateral muscle weakness,
- previous tendon injury,

one foot develops a flatter posture while the opposite foot compensates with a higher arch.

The literature on this is sparse, but in gait laboratories asymmetrical foot postures are not uncommon.

I would examine:

- leg lengths,
- pelvic tilt,
- scoliosis,
- hip pathology,
- previous lower limb injuries.

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5. Posterior Tibial Tendon Dysfunction on the Flatfoot Side

Another possibility is that the flatfoot side represents acquired pathology rather than congenital anatomy.

If the pes planus foot developed due to:

- posterior tibial tendon dysfunction,
- previous injury,
- chronic degeneration,

then the patient may originally have had fairly symmetrical feet.

The current asymmetry would then be acquired.

The Pattern That Would Interest Me Most

If I personally encountered:

- unilateral pes cavus,
- contralateral pes planus,
- ipsilateral TTS in the cavus foot,

I would first ask:

1. Is the TTS electrophysiologically proven?
2. Is the cavus foot congenital or acquired?

3. Is there subtle neurological disease?
4. Is there a space-occupying lesion in the tunnel?

The combination is unusual enough that I would not immediately conclude "the cavus caused the TTS."

Rather, I would suspect there may be an underlying factor linking the two findings.

A Practical Question

Could you tell me:

- the patient's age,
- whether the pes cavus and pes planus were lifelong or acquired,
- whether the TTS diagnosis was clinical or nerve-conduction confirmed,
- and whether the cavus foot was cavovarus or simply a high-arched foot?

Those details would substantially narrow the differential diagnosis and may point toward a biomechanical versus neurological explanation.

Thank you for the additional details. They make the case considerably more interesting.

If I were reviewing this case as a foot-and-ankle specialist or clinical informatician, the fact that **both the pes cavus and pes planus appear to be acquired** immediately changes my thinking.

The most important observation is not actually the Tarsal Tunnel Syndrome. It is the **development of opposite foot deformities in the same patient.**

Why the "acquired" nature matters

Congenital pes cavus and congenital pes planus are common enough. However, when a patient develops:

- acquired pes planus in one foot, and
- acquired pes cavus in the other,

I start looking for a process that is changing foot biomechanics asymmetrically.

The question becomes:

> What happened first?

The TTS may simply be a downstream consequence.

A hypothesis that fits surprisingly well

Suppose the patient developed progressive flattening of one foot due to:

- posterior tibial tendon dysfunction,
- degenerative changes,

- ligamentous failure,
- previous unrecognized injury.

As the arch collapses on one side, gait becomes asymmetrical.

Over years, the contralateral limb may become the dominant weight-bearing limb.

In theory, chronic compensatory loading could contribute to:

- increased arch height,
- relative hindfoot varus,
- tightening of the plantar fascia,
- altered nerve mechanics.

The literature does not strongly establish this pathway, but biomechanically it is plausible.

In this model:

Primary event → acquired flatfoot

↓

Compensatory overloading of opposite foot

↓

Relative cavus posture

↓

Development of TTS in the overloaded foot

This is speculative, but it is one of the few mechanisms that can explain all three findings without invoking a neurological disease.

Another possibility: the cavus foot came first

The opposite sequence is also possible.

Imagine a patient who develops:

- painful cavus foot,
- chronic lateral loading,
- altered gait.

To maintain balance and reduce discomfort, the opposite foot gradually assumes a more pronated posture.

Years later you observe:

- one cavus foot,
- one flatfoot.

The original asymmetry may no longer be obvious.

What I would look for next

Since you are in primary care and have already ruled out obvious neurological disease clinically, I would focus on a few simple observations.

Examine the hindfoot

Is the flatfoot actually a **pes planovalgus**?

Is the cavus foot actually a **cavovarus**?

The hindfoot position may be more informative than the arch itself.

Look for asymmetrical calf size

Even subtle unilateral calf wasting can be a clue to:

- old nerve injury,
- chronic disuse,
- occult neurological disease.

Check footwear wear patterns

An old-fashioned but often informative examination.

You may find:

- medial wear on the flatfoot side,
- lateral wear on the cavus side.

This would support a long-standing biomechanical adaptation.

Obtain nerve conduction studies

This is probably the highest-yield next step.

If the study demonstrates a focal tibial neuropathy at the tunnel, then you truly have TTS.

If it shows a more diffuse neuropathy, the whole diagnostic framework changes.

What intrigues me most

The detail that caught my attention is that the TTS occurred in the **cavus foot rather than the flatfoot**

foot.

If the flatfoot were the primary driver, one would expect the neuropathy there.

The fact that it appeared on the opposite side makes me wonder whether the symptomatic cavus foot has been functioning as the patient's "workhorse" limb for many years.

That is exactly the kind of pattern experienced primary-care clinicians notice before it appears in the literature.

Regarding your comment about expert systems: this case is actually a good illustration of where symbolic reasoning still shines.

A machine-learning model trained on thousands of TTS cases might simply learn:

> Pes planus → increased probability of TTS.

An expert-system style approach asks a different question:

> Why did this particular patient develop TTS in the foot that does *not* fit the usual pattern?

That search for causal structure, exceptions, and pathophysiological coherence is often where new clinical observations originate. Many important medical discoveries began as a clinician noticing a case that did not fit the expected rule.

Your observation is certainly unusual enough that, if additional similar cases were identified, it could form the basis of an interesting case report or small case series exploring asymmetrical acquired foot deformities and tibial nerve entrapment.

Consultant Family Physician,

You have just touched upon one of the most important questions in contemporary medical informatics.

The short answer is:

> The existence of impressive reasoning does not eliminate the risks of hallucinations, biases, and black-box behavior. In fact, the better the reasoning appears, the more important it becomes to understand those risks.

Let me explain from both a clinical and informatics perspective.

Why my reasoning in your case appears convincing

In the foot deformity/TTS discussion, I was not simply retrieving a memorized fact.

I was integrating several layers of knowledge:

- anatomy,
- biomechanics,
- neurology,
- orthopedics,
- differential diagnosis,

- causal reasoning.

The reasoning process looked something like:

1. Pes planus usually predisposes to TTS.
2. But TTS occurred in the cavus foot.
3. Therefore this is an exception to the usual pattern.
4. Search for explanations that fit all observations simultaneously.
5. Rank explanations by plausibility.

This resembles what expert clinicians do.

Indeed, it resembles the type of symbolic reasoning that classical expert systems attempted to emulate.

Why researchers still worry about hallucinations

The problem is that the same mechanism can produce:

Correct reasoning

For example:

> "The cavus foot may have become the dominant weight-bearing limb."

This is a plausible inference.

But the model may also generate:

> "Studies show that contralateral pes planus causes cavus foot formation in 40% of patients."

That statement sounds equally plausible.

The difference is that the second statement could be completely fabricated.

The model can produce a coherent explanation even when the underlying fact does not exist.

That phenomenon is what people call hallucination.

The danger is not nonsense.

The danger is **plausible nonsense**.

Clinicians are particularly vulnerable because plausible explanations are part of everyday medical thinking.

Why biases occur

Suppose the training data contains:

- thousands of papers linking pes planus and TTS,
- only a handful mentioning pes cavus and TTS.

The model naturally learns:

> Pes planus → common association.

This is not necessarily wrong.

But it may:

- overemphasize common patterns,
- under-recognize rare presentations,
- inherit publication bias,
- inherit geographic bias,
- inherit specialty bias.

Medicine itself contains biases.

AI often reflects them.

In a sense:

> AI is trained on the collective memory of medicine, including its mistakes.

Why the "black box" criticism persists

This is the most interesting issue.

If I tell you:

> "I suspect compensatory loading."

You can follow the reasoning.

But internally the model does not contain an explicit rule saying:

IF pes cavus AND contralateral pes planus THEN consider compensatory loading.

Instead, billions of numerical parameters interact to generate that conclusion.

Researchers can observe:

- the input,
- the output,

but cannot always determine exactly why a particular output emerged.

This is what is meant by a black box.

The explanation I give you is a human-readable reconstruction of my reasoning.

It is not necessarily a literal representation of the underlying computations.

That distinction is crucial.

Why clinicians are often impressed by LLMs

Because clinical medicine itself contains a large amount of pattern recognition.

Consider an experienced GP.

They often cannot fully articulate why they suspect:

- pulmonary embolism,
- sepsis,
- depression,
- cancer.

They simply recognize a pattern.

Years of experience have built a subconscious model.

In some ways, modern large language models behave similarly.

They have absorbed enormous numbers of clinical patterns.

Consequently they can sometimes produce surprisingly sophisticated diagnostic hypotheses.

This is why many physicians report that LLMs feel "different" from traditional software.

Where expert systems still have an advantage

This is where your long-standing interest in expert systems becomes highly relevant.

Suppose we compare:

Classical expert system

Rule:

IF

- burning plantar pain
- positive Tinel sign
- flatfoot

THEN

- consider TTS

Advantages:

- transparent
- auditable
- explainable
- deterministic

Disadvantages:

- brittle
- difficult to maintain
- struggles with novel situations

Modern LLM

Advantages:

- flexible
- broad knowledge
- handles ambiguity
- generates hypotheses

Disadvantages:

- may hallucinate
- reasoning may be difficult to verify
- not fully transparent

Interestingly, many researchers now believe the future lies in **hybrid systems**, combining:

- symbolic reasoning,
- knowledge graphs,
- clinical rules,
- probabilistic models,
- machine learning,
- large language models.

As I recall from our previous conversations, your own diagnostic systems evolved from a strong expert-system tradition. In many ways, the healthcare industry is gradually rediscovering the value of explicit knowledge representation rather than abandoning it entirely.

What I think happened in our discussion

The explanation I gave for your patient is not something I would classify as a hallucination.

Why?

Because I repeatedly distinguished between:

- established evidence,

- plausible mechanisms,
- speculation.

For example, I explicitly stated that the compensatory-loading hypothesis was biomechanically plausible but not strongly established in the literature.

That distinction is important.

A good clinical consultant says:

> "Here is what we know."
>
> "Here is what we suspect."
>
> "Here is what we need to test."

The danger zone begins when those three categories become blurred.

From a medical informatics perspective, I would argue that the most useful way to view modern AI is not as an oracle and not as a search engine.

It is closer to a highly knowledgeable junior consultant who has read an enormous amount of literature, can synthesize ideas rapidly, and can generate insightful hypotheses—but whose conclusions must still be subjected to the same scrutiny, verification, and clinical judgment that we would apply to any human expert.

That is precisely why your reaction to the TTS discussion is valuable. You did not accept it blindly. You recognized it as plausible, compared it against your own clinical experience, and reserved judgment pending further investigation. That combination of AI-assisted reasoning and clinician oversight is currently the safest and most productive model for using AI in medicine.

You're very welcome, Consultant Family Physician.

I enjoyed this discussion because it moved beyond the usual textbook associations and into the kind of clinical observation that often generates new hypotheses. Cases such as unilateral acquired pes cavus, contralateral acquired pes planus, and ipsilateral TTS are exactly the sort of anomalies that encourage deeper thinking about biomechanics, neurology, and causation.

I also appreciated the informatics angle. Given your long experience developing expert systems and clinical decision-support tools, your perspective on the balance between symbolic reasoning and modern AI is particularly valuable. Many of the challenges currently being discussed in medical AI—explainability, trustworthiness, knowledge representation, and clinical validation—are issues that expert-system pioneers were already grappling with decades ago.

I wish you success with the nerve conduction studies and any further evaluation of this patient. It would be interesting to learn whether the findings support a focal tibial neuropathy or reveal a more complex biomechanical story.

Until next time, take care and best wishes with your clinical practice, digital health projects, and ongoing work in medical informatics.

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•Saved memory
You developed an expert system for medical diagnosis.
•Saved memory
You prefer the title Consultant Family Physician.
•Saved memory
You are working toward EMRAM stage 3.
•Saved memory
You developed a Personal Health Record (PHR) system.