

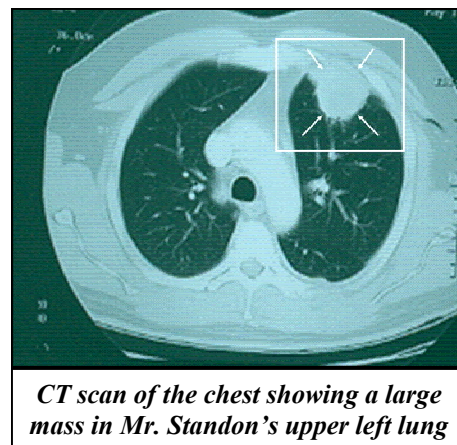


## THE ODYSSEY OF DAVID STANDON

### PART ONE

#### Chapter 1

In retrospect, it all seemed to begin at a Christmas party in December, 2001. David Standon traces the start of his problems to the overwhelming aromas of candles, incense, and perfume he was exposed to that evening. At the party Mr. Standon was seized by intense congestion of his nose and sinuses, symptoms he had never experienced before. In contrast to most illnesses that come and go during the Holiday season and are caused by viruses, these upper respiratory tract problems did not subside after a few days, but continued to worsen for several weeks. He became utterly unable to breathe through his nose and suffered regular nosebleeds. The inside of his nose became filled with brownish crusts. He was

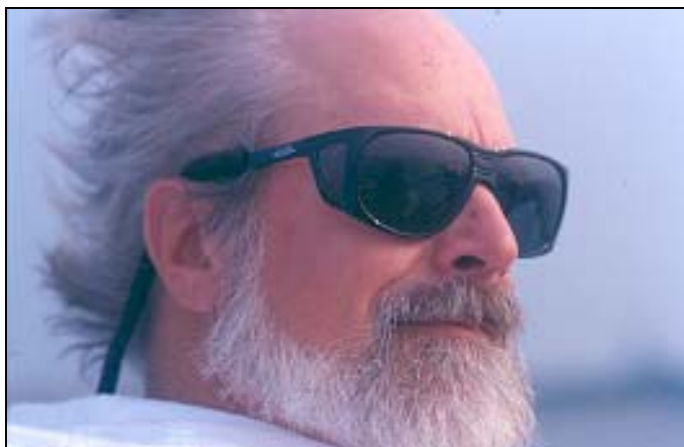


*CT scan of the chest showing a large mass in Mr. Standon's upper left lung*

eventually referred to an Ear, Nose, & Throat specialist who recommended a sinus operation and ordered some routine pre-operative tests.

#### Chapter 2

Mr. Standon's pre-op chest X-ray revealed a large mass in the upper lobe of his left lung [Figure]. Although no one said it aloud, Mr. Standon feared that the "lung mass" was cancer. His operation changed from surgery on his sinuses to a biopsy of the lung mass. In the days leading up to his procedure, some other strange new symptoms appeared: red eyes ("episcleritis") [Figure], cold fingers, and pain in every joint in



*Mr. David Standon*

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*Episcleritis*

his body. So severe was Mr. Standon's arthritis that he could scarcely get out of bed.

The lung biopsy report read "necrotizing granulomatous inflammation" — not cancer. Still, Mr. Standon felt too awful to be relieved. *And what did "necrotizing, granulomatous inflammation" mean, anyway?* Mr. Standon's internist, Dr. Lawrence Solomon, called The Vasculitis Center to consult on the details of the case and confided his hunch: that Mr. Standon had Wegener's granulomatosis. I agreed to see Mr. Standon and his wife Chris in clinic immediately.

### Chapter 3

It didn't take long to confirm Dr. Solomon's hunch. Mr. Standon had a textbook case of Wegener's: a middle-aged man of Scandinavian descent, upper respiratory tract inflammation, polyarthritis, episcleritis, a painful tongue ulcer [Figure], "splinter" hemorrhages under his fingernails, and the lung mass with its pathological hallmark of Wegener's. For Mr. Standon, there was no going home that day. Although the hospital was full, even before the results of his blood work were back, Mr. Standon was on his way to the Emergency Room for his first dose of steroids.

*Painful tongue ulcer, one of Mr. Standon's worst complaints*

### Chapter 4

Within hours of the time that steroids first dripped into his arm, Mr. Standon began to feel better. His joints were improved first. When a bed became available in the hospital, Mr. Standon was admitted to complete his evaluation and continue treatment. By then, his eyes were no longer red. He had blood and protein in his urine — signifying kidney inflammation — but his kidney function appeared minimally affected. When he left the hospital after

three days his blood creatinine level, a measure of kidney function, was just mildly elevated (1.5 mg/dL; normal up to 1.4). He was discharged on cyclophosphamide and prednisone. I confided to Chris that Mr. Standon had started treatment just in time; a few more days and his kidneys would likely have failed for good.

### Chapter 5

Bad news. Although almost all of Mr. Standon's symptoms had begun to resolve promptly, his kidney function had grown worse [See Table below]. One week after starting cyclophosphamide and finally going home, his kidney function had declined significantly. This was reflected in his creatinine level of 2.5. (Failing kidneys do not perform their blood-cleansing function properly, leading to the build-up of metabolic waste products — such as creatinine — in the blood). The next creatinine check five days later — 3.6 — was more disturbing still. With cyclophosphamide and prednisone, we were doing everything possible to stamp out the inflammation gripping Mr. Standon's kidneys, but the situation was growing more and more dire by the day.

The next week things had gotten even worse. Creatinine: 4.4. I re-admitted Mr. Standon to the hospital for another few days of high-dose steroids in his veins. But despite three more "pulses" of treatment, his creatinine level rose inexorably: 5.8...6.5...7.8.... At that rate, I knew that dialysis wasn't far off.

### Chapter 6

As kidneys cease to function and the condition of "uremia" sets in, a host of effects on other organs becomes apparent. The blood platelets don't work as well, placing patients at increased risk of bleeding. The heart and lungs can

<i>Mr. Standon's Creatinine Levels</i>	
DATE	Creatinine (mg/dL)
2/15/01	1.5
2/20/01	2.5
2/25/01	3.6
2/28/01	4.4
3/4/01	5.8
3/12/01	7.8
Start dialysis	8.1
4/15/01	3.7
4/22/01	1.8
Off dialysis	1.9
Now	1.8

become overloaded with fluid, leading to congestive heart failure. Even the brain is affected by “uremic encephalopathy”, a clouding of the mind caused by a build-up of toxins not cleared by failing kidneys.

**The worse the kidney function,  
the higher the blood creatinine level**

In cases like Mr. Standon’s, the doctor agonizes. And I did: “*Am I missing something?*” “*Would a kidney biopsy help?*” “*Should we try plasmapheresis*” (an unproven therapy with significant risk of infection)? And even: “*Do we have the correct diagnosis?*”. Times like this are frightening for the patient, uncertain for the family, and lonely – very lonely – for the doctor. In such situations, several things help. Talking frankly with the patient and the patient’s family is essential. They must be informed about what to expect — within the limits of what is “knowable” at the time. Reviewing the case with colleagues can also be extremely useful for the doctor. Input from colleagues frequently provides different insights and new approaches to the case that one had not considered. In addition, simply speaking about a difficult case with those who can empathize directly is a way of sharing the uncertainty – the anguish – of watching a patient decline despite one’s most earnest efforts and the best therapies available.

The nephrologists suggested performing a kidney biopsy, and I considered their recommendation over and over again. Having requested the input of subspecialty colleagues on a problem, one ignores their advice at some risk. In this case, after all, the nephrologists were the kidney experts. Furthermore, biopsies are often essential to excluding non-vasculitic conditions and to determining prognosis. In the evaluation of patients with possible vasculitis, I routinely obtain biopsies in several situations. But I couldn’t imagine then how information from a kidney biopsy would change either our diagnosis or its treatment. This couldn’t be anything except Wegener’s – Wegener’s behaving in a way the disease is known to behave. The kidneys are typically the last organ system to improve, and sometimes they get worse before

getting better. We opted against the kidney biopsy.

In entertaining ideas of other treatments, I also considered the other side of the treatment coin. Battering away with higher and higher doses of toxic medications sometimes risks dangers even greater than dialysis; namely, life-threatening infections that Mr. Standon’s compromised immune system could not fight. After discussions with the Standons, we decided to stay the course, neither to add new treatments nor to intensify those Mr. Standon was already on, and to give the therapy we had chosen more time to work. I told them that I hoped dialysis could still be averted, and that if he needed dialysis I hoped it would be temporary. But there was no guarantee.

### Chapter 7

It struck some of my nephrology colleagues as misguided, but as Mr. Standon’s kidney function declined I *decreased* his cyclophosphamide dose. Adequate kidney function is essential to metabolizing full doses of cyclophosphamide. Failing to adjust the dose when the kidneys are failing can lead to major side-effects, particularly low white blood cell counts and infections. I didn’t want to save the kidneys while losing Mr. Standon.

When his creatinine rose to 8.1, I admitted him for the third time, this time to begin dialysis.

### Chapter 8

I often marvel at how patients shoulder adversity. Only weeks before, Mr. Standon had never heard the term “creatinine”. Now he kept tabs on his creatinine level as carefully as a stockbroker follows the Dow. He quickly settled into his dialysis routine three times a week. From my vantage point, it seemed that Mr. Standon had become a favorite of the dialysis nurses: they doted on him. Several weeks later, Mr. Standon spoke to me on the telephone while he was hooked to the dialysis machine. I asked what his creatinine had been that day. His reply: “3.7”. “*He’s getting there!*”, I thought, with a guarded sense of relief. One week later the number was 1.8, and the dialysis catheter was removed from his chest. Six weeks after his kidneys had failed, Mr. Standon had become dialysis-free.



**Chapter 9**

After 6 months, Mr. Standon tapered off of prednisone and switched from cyclophosphamide to azathioprine. The lung mass was gone. He remained off dialysis and had enough good kidney to last for decades – assuming the Wegener’s didn’t come back. His energy had returned. His ANCA test was negative. His disease was solidly in remission.

He served as the Best Man in the wedding of his son, Erik, to his new daughter-in-law, Stephanie. He began to think about living again, about getting back to the things he had enjoyed before getting sick, about retiring to have more time to fish. I included a discussion of his case in a talk at the American College of Rheumatology meetings, emphasizing one of the major lessons illustrated by his case: that sometimes in the treatment of vasculitis, less is more. The lowering of his cyclophosphamide dose even as his kidney function worsened had shown this. Mr. Standon also graciously came to “Topics in Internal Medicine”, the annual Continuing Medical Education program at Hopkins attended by physicians from all over the globe. There, he helped teach an auditorium full of doctors about his disease and the nuances of its treatment.

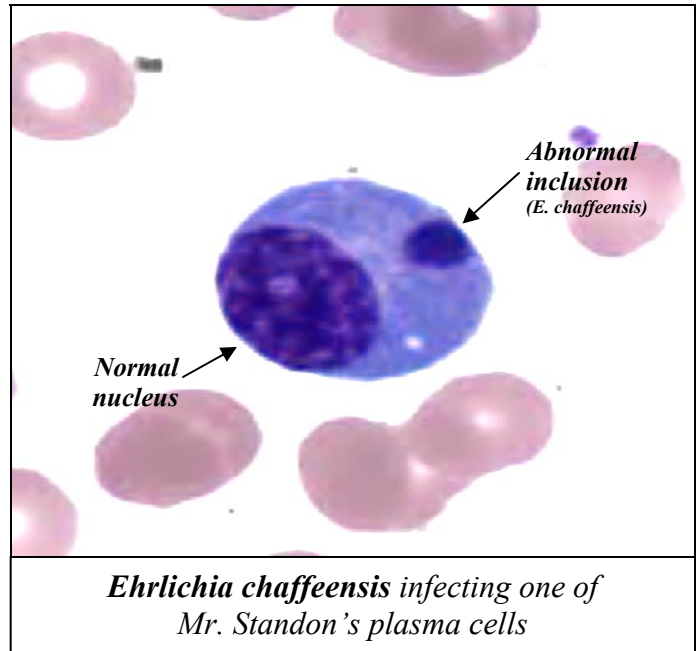
As we closed that presentation, I mentioned that Mr. Standon was now in the market for retirement property. He and Chris were actively looking for a place near the water on Maryland’s Eastern Shore.

**PART TWO****Chapter 10**

Two weeks after the Topics course, Mr. Standon called to say he hadn’t felt well for several days. Fever, sore throat, fatigue... “*Sounds like a viral thing*”, I told him. But I could tell by the sound of his voice that he wasn’t so sure.

**Chapter 11**

His symptoms persisted through the weekend. I had him come in to clinic for a quick look. One glance at his eyes – now red again – spoke volumes. Something was going on. Was it Wegener’s again? His creatinine had jumped from 1.9 to 3.8.



In contrast to his classic presentation with Wegener’s 16 months earlier, this time Mr. Standon’s symptoms didn’t add up. Although he was clearly quite ill, he had no nasal symptoms, no arthritis, no painful tongue ulcer, no lung lesions, no red blood cells in his urine. In short, with the exception of red eyes, he had almost none of the features of Wegener’s that he had shown before. Furthermore, his ANCA test was also still negative. On the other hand, he did have striking temperature elevations, a faint but diffuse skin rash, low platelets, and elevations of his liver function tests, all of which were new. *Could this really be a flare?*

**Chapter 12**

In the hospital, there was no quick answer. On the contrary, only more confusion. The dry hospital air brought a return of Mr. Standon’s nasal crusting, renewing concerns about an unusual disease flare. Despite Tylenol, his temperature soared to > 104°F. He spent most of one day with severe gastrointestinal upset (deepening the mystery further. This is not a feature of Wegener’s). With IV fluids, his creatinine rise leveled off at 4.6, but he was growing more and more short of breath by the hour. His chest X-ray, still normal, revealed no clues. The interns, residents, medical students, and I were mystified. Something was badly wrong, getting worse, and we hadn’t figured it out yet. We asked the Infectious Disease consultants to see Mr. Standon.

**Chapter 13**

That night the intern, Dr. Ghazaleh Aram, called me at home. Mr. Standon's condition had worsened substantially in the hours since I had left the hospital. Dr. Aram was preparing to transfer him to the Intensive Care Unit; he was no longer stable enough for the regular floor.

Just then, in short succession, Dr. Aram received two pages from the lab. The first page told her that Mr. Standon's cardiac enzyme levels were sky high. This suggested myocarditis – inflammation in the heart wall muscle – as the cause of Mr. Standon's shortness of breath. Though a plausible enough explanation for difficulty breathing, myocarditis made no sense whatsoever in the context of Wegener's (Wegener's seldom involves the heart, and never does so in the form of myocarditis). The second page from the lab provided the full answer: Mr. Standon's blood test for antibodies to *Ehrlichia chaffeensis* was strongly positive. This was the "Eureka! Moment" we had sought: Mr. Standon had *ehrlichiosis*.

**Chapter 14**

Ehrlichiosis, first identified as a disease in 1986, is an infectious disease caused by an organism transmitted by the bite of a tick. In retrospect, as it always does, this diagnosis made perfect sense. With his Wegener's in remission, Mr. Standon had traveled to the Eastern Shore of Maryland (a tick-endemic region) and tramped through the woods looking at retirement properties. Ticks, as everyone knows, carry the organism that causes Lyme disease. They also carry *E. chaffeensis*, and sometimes manage to transmit both ehrlichiosis and Lyme disease to the same unfortunate individual. (If there was a bright side to Mr. Standon's close encounter of the ticklish kind, it's that he didn't get Lyme disease at the same time). *E. chaffeensis* enters the "host" through

the skin as the infected tick is doing what ticks do: sucking the host's blood. Once it gains access to the bloodstream, *E. chaffeensis* mediates its mischief in part by infecting white blood cells. In fact, after we suspected the diagnosis on the basis of his blood test, a careful review of Mr. Standon's blood smear from the day of admission showed the organism within one of his white blood cells [Figure].

**Chapter 15**

Even with this new diagnosis in hand, we were not yet – to use a relevant phrase – out of the woods. Mr. Standon not only had ehrlichiosis, he had it **BAD**. Myocarditis is an unusual but reported complication of ehrlichiosis. It can be fatal. The severity of Mr. Standon's case of ehrlichiosis probably stemmed in part from the immunosuppressive medicines he had required to treat his Wegener's. The inflammation in his heart led to a profound impairment of that organ's pumping ability. With each heartbeat, normal hearts eject 55-60% of the blood within the pumping chamber, distributing it to all parts of the body. Mr. Standon's "ejection fraction" at the height of his illness was only about 15%. Ejection fractions lower than 10% are not compatible with survival. Once again, Mr. Standon had started treatment in the nick of time.

**Chapter 16**

Like many diseases known to be caused by microbes, ehrlichiosis has a potent antidote: antibiotics. Specifically, doxycycline. After one dose of "Doxy", Mr. Standon's fevers vanished. He began to feel better in small but unmistakable ways, and became certain of his recovery far earlier than I.

During his recuperative period, we again had to fend off sub-specialists who wanted to perform biopsies (now the cardiologists!).

**Lessons from Mr. Standon's Case**

- *The kidneys are often the final organ system to improve in Wegener's granulomatosis. They sometimes get worse before getting better.*
- *Nasal crusting can be the result of previous damage to the nasal epithelium (the top layer of tissue). In patients with Wegener's, it does not always signal active disease. Dry air (like that present in most hospitals) can make nasal crusting worse.*
- *Systemic infections can mimic active vasculitis very closely, posing challenges in diagnosis.*

## THE ODYSSEY OF DAVID STANDON (Continued)

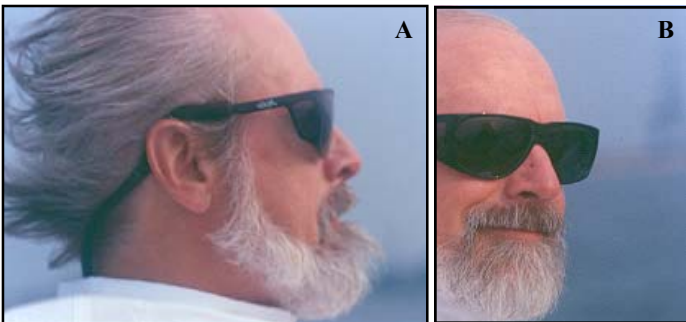
Mr. Standon was happy to agree with me (again) that a biopsy, this time of his heart, was unlikely to change our treatment approach. Now that he again had the proper diagnosis and therapy, we hoped for slow but consistent improvement in his heart function. And we got it. Serial echocardiograms showed an ejection fraction of 35% at one month, 45% at three months, and 55% (normal) five months after discharge. For the second time in the year 2003, Mr. Standon felt like a new man, and was.

### Epilogue

*Mr. Standon's New Year's Resolution for 2004 is simple: not to develop any more extraordinary illnesses. I plan on holding him to that. Even so, he has already taught us a great deal about his two remarkable illnesses. A summary of some of the lessons derived from his case is shown in the Box on page 5. Most of these lessons are broadly applicable to all forms of vasculitis and their treatment.*

*On a fishing trip that he and I took recently, Mr. Standon illustrated the completeness of his recovery. With the patience, steadiness, and tenacity that marked his approach to sickness and treatment, Mr. Standon reeled in fish after fish and made it look effortless all the while. In contrast, even with his selection of lures working in my favor, I could manage only one solitary nibble all evening. (It was a hungry one, though. Bit the lure in half – the half without the hook!). And on that trip I discovered one more thing about David Standon. His reasons for loving to fish have less to do with catching fish than with driving his boat — and driving it fast!*

*John H. Stone*



**On the Harbor: A) 80 m.p.h.; B) Rest**

## FAQ — HANGING OUT SHINGLES

*Clare Goodman is a person with Takayasu's arteritis. She has been followed for many years at the Vasculitis Center by Dr. David Hellmann. Therapies for her condition have included prednisone, immunosuppressive medications, blood thinners, and other treatments. She is an extremely well-informed patient who understands the potential for complications that her medications bring. Recently her husband Keith, otherwise hale and hearty, developed "shingles", a painful rash caused by the Varicella zoster virus (VZV), the same virus that causes chickenpox. Was there anything for Clare to be worried about with regard to her own health?*



**Clare Goodman with her husband, Keith, on vacation in Amsterdam**

The short answer to Mrs. Goodman's question — fortunately — is "No". The longer answer is somewhat more complicated, and raises many issues about both the function of the immune system and the interaction between this unusual virus and its host, humans.

Chickenpox is the result of an individual's first exposure to VZV. This of course, usually occurs in childhood; essentially all adults had chickenpox when they were kids. Once the fevers and itchy red rash of chickenpox subside, however, VZV does not go away entirely. Rather, it becomes dormant, "hibernating" in the body (often for decades) within nerve roots along the spine.

Years after the initial chickenpox infection, VZV can re-emerge, this time not as chickenpox but as "shingles", a painful red rash that usually occurs in the distribution of a single nerve. The classic appearance of the rash is what dermatologists call "grouped vesicles on a red base" [Figure, page 7]. The nerve distribution involved corresponds to the nerve root in which the virus has hidden (near the spine) ever since the original chickenpox rash resolved. The rash of shingles is usually confined to only one side of the body — either the right or the left — because a single nerve root receives sensory input from only one half of the body.



Shingles is a common problem, both among patients with vasculitis and (less often) among their family members. *What other questions does Mr. Goodman's case of shingles raise?*

### **Who gets shingles?**

Most cases of shingles occur in individuals who, like Mr. Goodman, are generally well. There is some tendency for shingles to occur in older individuals, when a subtle waning of immune system function occurs. Shingles, or “zoster” as it is sometimes known, is usually a one-time, self-limited episode that resolves within a few weeks. The nerve root inflammation that occurs with shingles, however, can be very painful. Moreover, about 20% of patients continue to have pain for six months or longer after their rash has subsided. Because VZV is a member of a family of viruses known as the *herpesviruses*, this condition is known as “post-herpetic neuralgia”.

### **How common is shingles?**

Quite common, actually. Up to half of people who live to the age of 85 may get shingles at one time or another. There are an estimated half a million cases of shingles per year in the United States alone.

### **Isn't Mrs. Goodman at risk because of her immunosuppressive medications?**

Mrs. Goodman had chickenpox when she was a child. Consequently, re-exposure to VZV because of her husband's shingles does not pose any new risk to her. (In fact, she undoubtedly has some VZV residing in one of her nerve roots, too, left over from when she had chickenpox at age 4). The dormant



**Grouped vesicles on a red base:  
The classic shingles rash**

virus may or may not ever lead to an eruption of shingles in her.

Any risk that Mrs. Goodman has for shingles stems not from her husband's rash but from the “latent” virus that

she – like most of us – already has. Although Mrs. Goodman's immune system is dampened to some degree by the moderate doses of prednisone she takes, she is probably fully capable of holding at bay

any hibernating VZV. Ironically, the exposure that she has gotten to a new strain of VZV through contact with her husband's rash may serve as a “refresher course” for her own immune system, reminding it how to recognize this virus and keep it in check. This may actually lower her own risk of developing shingles.



**Scabbed over shingles lesions. This patient is no longer infectious**

### **But aren't patients on prednisone and other vasculitis treatments more likely to get shingles?**

Yes, compared to the general population, they are. In a recent Vasculitis Center clinical trial (see Research Updates, page 8) in which all patients received conventional immunosuppressive medications, there were 13 cases of shingles reported out of 180 patients in the trial – a statistically significant greater number than one would expect among individuals not on treatment for vasculitis over a similar time period.

### **What can be done to treat shingles?**

Several interventions can help. First, anti-viral medications decrease viral replication, shorten the duration of the rash, and diminish the severity and duration of pain associated with shingles. Anti-viral medications that are active against VZV include acyclovir, famciclovir, and valaciclovir. Patients with shingles generally take these medications for one or two weeks. Second, a group of medications known as “tri-cyclics” (because of their three-ringed chemical structure) can also be very helpful in relieving shingles-associated discomfort. Examples of tri-cyclic medications are amitriptyline and nortriptyline. Other medications that are useful for post-herpetic neuralgia are gabapentin and local anesthetic patches (lidocaine). Finally, many patients require short courses of narcotic medications to treat the pain associated with nerve root irritation.

### **Is there any way to prevent shingles from occurring again?**

Yes, but prophylactic therapy is usually not indicated. Fortunately, second bouts of shingles are rare, and their infrequent occurrence does not justify

## HANGING OUT SHINGLES (continued)

continuous preventive treatment with anti-viral medications. In most circumstances, no preventive therapy is required.

### *Are there any danger signs for people who get shingles?*

There are a couple of things to watch out for. First, shingles can sometimes involve a nerve distribution that includes the area around the eye. If the eye becomes involved (or threatens to), prompt consultation with an ophthalmologist is required. Second, as noted, most cases of shingles are localized to the distribution of a single nerve root (a region on the surface of the skin known as a “dermatome”). Spread of the rash outside the area of one dermatome may indicate that the VZV exacerbation has not been contained adequately by the immune system and poses the threat of becoming “disseminated”. This is a dangerous scenario that demands urgent anti-viral treatment.

### *Is there anyone whom Mr. Goodman should avoid while he has his rash?*

Yes. It would be best for Mr. Goodman to avoid contact with infants less than 18 months old. These little humans, never exposed to VZV before, are at greater risk for complications if they acquire this infection.

### *How will the recently-available chickenpox vaccine affect the frequency of shingles?*

Within the past decade, most children have been vaccinated against VZV. This vaccine is usually administered around the age of 1 year. The vaccine consists of a live, “attenuated” (weakened) strain VZV. This shot primes the immune system to recognize and destroy the virus without causing an active infection, thus making the child “immune” to VZV infections. The vaccination not only prevents the children from getting chickenpox when they are young, it will also greatly decrease the number of cases of shingles that occur several decades from now. This, as Mr. Goodman knows, will be a big blessing for his children, grandchildren, and great-grandchildren.

*Follow-up: Mr. Goodman's case of shingles has resolved without any long-term consequences.*

## RESEARCH UPDATES

Analysis of the Wegener’s Granulomatosis Etanercept Trial (WGET) is now under way and will be complete by Spring, 2004. Two interesting bits of information, however, have already emerged from this trial. This trial, the first multi-center NIH-funded trial in Wegener’s granulomatosis, has involved eight centers around the United States and is directed by the Vasculitis Center. Noteworthy results to date include:

- ***Gender disparity in Wegener’s?*** There appears to be a gender disparity with regard to certain clinical features of Wegener’s. Women are more likely than men to have “limited” disease; i.e., features of this condition that are limited to the upper respiratory tract and do not lead to vital organ involvement, such as kidney disease. Patients with limited disease also tend to be younger at the time of their disease onset and, unfortunately, to have disease that is more likely to recur.

The WGET researchers emphasize that there is a great deal of overlap in the clinical subsets of Wegener’s. Women can get severe disease, and men may certainly have limited Wegener’s, too. Further understanding of this apparent gender disparity, however — observed in this study for the first time — may yield important insights on the disease. Baseline data on the WGET cohort of patients, including these findings, were published in *Arthritis and Rheumatism* in August, 2003.

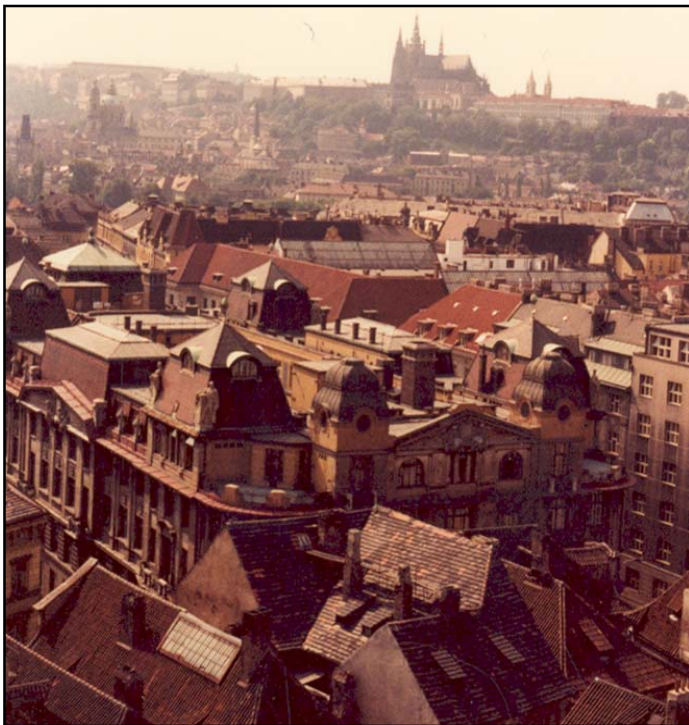
- Deep venous thrombosis in Wegener’s patients have a striking tendency to form blood clots in their legs, particularly during periods of active disease. These blood clots, known as “deep venous thromboses” (DVTs), are dangerous because they may break off from the places they form in large leg veins and travel to the lungs, potentially leading to death. DVTs that migrate to the lung are called “pulmonary emboli” (PEs).



## **Research Updates (continued)**

Twenty-nine of the 180 patients in WGET either developed a DVT or PE during the trial (16 patients) or had histories of a DVT/PE prior to trial entry (13 patients). The risk among Wegener's patients for a DVT or PE was 22 times higher than that of normal individuals, and 7 times higher than that of patients with lupus in the Johns Hopkins Lupus Cohort. (Lupus is a disease with a known clotting tendency). Of note, there was no increase in the risk of DVT or PE associated with the experimental treatment [etanercept; Enbrel]. During the trial eight DVT/PEs occurred in the etanercept group and eight in the comparison group.

Awareness of this major potential complication has immediate implications for the management of patients with this disease (See Box, to the right, on Mr. Robert Townsley). Data related to the frequent occurrence of DVTs and PEs in Wegener's were reported in October at the International Vasculitis/ANCA Meeting in Prague (Czech Republic).



***Prague: the site of the 2003 International Vasculitis/ANCA Meeting***

***The case of Mr. Robert Townsley of Sykesville, Maryland, exemplifies perfectly the research findings reported to date from WGET:***

Mr. Townsley, a lifelong Baltimore sports fan, has a history of severe Wegener's with kidney disease that threatened dialysis before he started (and responded to) treatment. In the Spring of 2002, after doing a bit of gardening, Mr. Townsley noticed redness and swelling of his right leg. He called the Vasculitis Center and wondered if there was anything he needed to do for his leg, which he presumed was swollen because of a bug bite.

Because of the data related to the occurrence of DVTs already emerging from WGET, red flags went up at the Vasculitis Center when Mr. Townsley called. Mr. Townsley was sent to a radiologist near his home in Sykesville for an urgent ultrasound of the leg in question. The ultrasound confirmed that the cause of the leg swelling [Figure] was a DVT, a blood clot in a large vein.



***Mr. Townsley's legs  
(swelling on his left)***

Mr. Townsley successfully completed a six-month course of coumadin (a blood thinner) and the clot in his leg resolved without complications. Two years after his Wegener's diagnosis, Mr. Townsley's disease remains in complete remission. He is back to his favorite pastime — attending sports memorabilia fairs — and warns everyone to watch out for the Ravens next year!

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**VOLUNTEER PROGRAM**

**Seeking Volunteers to assist Vasculitis Center Staff during Clinic hours!**

**Please contact:**

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**5501 Hopkins Bayview Circle**  
**JHAAC, Room 1B.22**  
**Baltimore, MD 21224**

**410-550-6816**  
**dward1@jhmi.edu**

**Clinic Corner: Spreading Her Talent Around**

For years, patients at the Vasculitis Center and their families have appreciated (no, revered) Judy Harrison and her talents as a Patient Care Coordinator. Judy possesses a personal warmth and an understanding for people's feelings and concerns that have set her apart during her tenure at Johns Hopkins, and made her a shining star in our Center.

patients, Judy now oversees the Patient Care Coordinators for the Scleroderma and Arthritis Clinics, as well. We are grateful for Judy's talents, and thankful for everything she does for the Vasculitis Center patients, staff, and faculty.



**JUDY HARRISON**  
 Rheumatology Division  
 Clinic Supervisor

Don't worry – she's not leaving! You will still find her buzzing around the Rheumatology Clinic: answering phones, talking with patients and their families, making us all feel better, and solving any number of problems that come up in the course of a normal day in the life of a busy clinic. But she is spreading her talents around. In addition to helping Cynthia Bethea and Sidone Lawrence assist Vasculitis Center

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