How’s Sean’s Health?

Two years after his last POZ blab, our longtime survivor goes head-to-head with his longtime doc on everything from drug holidays to hair loss. Bob Lederer catches up

January 1, 2003 By Bob Lederer

Soon after POZ’s launch in 1994, “What This Means,” which tracked the health of founder Sean Strub by spelling out his labwork in plain English, emerged as a must-read. Over the next four years, as Sean grew dangerously ill and then suddenly rebounded on combo therapy [see “The Strub Files,” below], this column made for high drama not only for other Lazaruses but for the voyeur in us all. Now, two years and six (unsupervised!) treatment breaks after his last check-in, Sean and his doctor, the pioneering Joseph Sonnabend, weigh in on Sean’s most recent (August 2002) labs. This time around, “I’m feeling self-conscious about sharing so much,” Sean confesses. “I know it served a purpose for readers in the past when I was often sick, but now that I am relatively healthy, I worry that it may sound like gloating.” But with a comeback complicated by lipo, fainting spells, high cholesterol, low testosterone and a grateful-but-hateful attitude toward his meds, the latest leg of Sean’s journey is likely to offer readers far more aid and comfort than he thinks.

Joseph Sonnabend: Sean has been doing just fine in the past two years. He periodically complains of fatigue, but overall, considering his trajectory over more than 20 years of having HIV, he’s quite functional. He doesn’t visit me regularly, and he goes on and off treatment on his own. I have many patients who take structured treatment interruptions (STIs), and I make recommendations on doing it (hopefully) safely. But Sean is his own doctor—he does what he wants.

Sean Strub: I’ve been on my current drug combo—indinavir (Crixivan), d4T (Zerit) and delavirdine (Rescriptor)—for almost seven years, but I’ve probably had six treatment interruptions since the last one I wrote about in POZ [see “Sean’s Trough Luck,” February/March 2001]. They may partly reflect simple laziness—my relatively good health allows me the perceived luxury of going off meds. I know my casual attitude bothers Joe, but my instinct has served me well—I’m more concerned about upsetting Joe than about the strategy being a mistake. I’ve only been on one triple combo, and if I develop resistance, other combinations are still available to me. Still, I have mixed feelings about these treatment breaks: The calm periods after stopping but before renewed viral activity are getting longer, but I fret that I’m risking resistance. Fatigue comes reliably after I’m off treatment for a while, but with each STI, the fatigue takes longer to develop.

JS: Sean’s CD4 count dropped from 452 in August 2001 to 392 one year later—not a significant

loss—and his viral load rose from undetectable to a bit below 1,000. Meanwhile, his CD4 percentages have gone steadily up, from 16.7 percent in March 2000 to 24.2 percent now. CD4 percentages measure what proportion of lymphocytes [antibody-making immune cells] are CD4s vs. CD8s. They're important because they're not affected by the total lymphocyte count, and are therefore less prone to variation than CD4 count. Sean was on meds at the time of these labs, but I believe he may be benefiting from past STIs. One rationale for STIs is that they may act as an auto-vaccine, in which newly replicating HIV stimulates the immune system. They also relieve drug toxicity.

SS: It would be a convenient lie for me to say that I go on STIs just to relieve drug toxicity, since I've had few serious side effects: occasional kidney sludge from Crixivan; occasional neuropathy, but nothing very painful. The worst is Crixivan-related anxiety for a couple of hours after each dose. I take a very small dose of Ativan to take the edge off anxiety.

I do the STIs because I like freeing myself from the tether of daily drugs. My body feels better overall, like it's fresh, clean, flushed out. I like the psychological freedom—it enables me to distance myself from the constant reminders that I have a disease.

I also think my HIV is reacting to treatment in a pattern that's similar to how my herpes has reacted. I was infected with herpes in the late 1970s, probably about the same time I got HIV. After I tested positive in 1985, I took acyclovir every day for almost 10 years. Over time, my semi-regular outbreaks and blisters lessened. Now I rarely get them. It's as if the herpes became less virulent with treatment. Similarly, I believe the HIV "outbreaks"—which is how I think of HIV in my body—are also lessening in virulence.

JS: I'm a proponent of the idea of "cycling" antiretrovirals. All these drugs are more or less toxic. Nobody can be expected to be on them for life without some modification. Cycling can reduce the toxicity—which all the drugs have, just affecting different organs (liver, kidney, bone marrow). I suggest periodically changing to drugs with different side effects—spreading the poison, if you like. Most patients are reluctant to go off treatments that work. But I believe that people's options could be extended if they switched while viral load is undetectable, as these drugs could likely be used again.

Over the past few years, Sean's viral load has bounced between undetectable and 1,713. I'm not bothered by that, but as soon as it becomes and stays undetectable, I'd like him to switch combinations. But I don't think he's willing to—yet.

SS: I'm warming up to the idea. But the awful, vain truth is that I am finding myself influenced by the fact that [POZ publisher] Brad Peebles is convinced that his hair has grown back on the top of his head because he went off Crixivan. For two years he's denied my jealous accusations that his new hair is the result of hair-growth drugs. I never had significant hair loss until the last few years. I assumed it was just aging, but then I noticed that my two brothers don't suffer the same loss. So maybe it is the Crixivan. With the incentive of my vanity plus the counsel of Joe, whom I respect enormously, I will probably change combos. Sometimes we PWAs make treatment decisions for

reasons that others may view as silly.

JS: Sean has a little lipodystrophy, but it's not too obvious. It's a combination of fat loss and fat accumulation. His arms are skinnier, his veins a bit more prominent, his checks slightly sunken. He's not that concerned about it, so I wouldn't say he needs to do anything.

SS: When I weigh around 160, the wasting in my face isn't visible. But when I get closer to 150, it becomes noticeable—first to me, then to other PWAs, then to friends and family.

JS: Sean has had several fainting spells. These most probably result from either vaso-vagal syncopal attacks (the vagus nerve's overstimulation of the body's blood-pressure-lowering function) or postural hypotension (sudden changes in posture producing low blood pressure). Some people, when they get up from a chair, don't compensate so quickly for the blood flowing to the lower part of the body. I don't know what's causing it, but I don't think it's particularly dire. However, if these spells get worse, I will have Sean checked.

SS: This has happened six or seven times over the past year, and while I've recovered fine each time, it is frightening. One evening I fainted a half-dozen times, starting at a restaurant and ending in an ambulance to a hospital emergency room. Twice I got minor bumps and bruises from falling—actually, more of a crumple in place than a "tim-ber-r-r-r-r"-type falling flat. Mostly it is embarrassing.

JS: In summer 2001, Sean's total cholesterol was 241—cause for concern. I had previously prescribed him the blood fat–reducing drug atorvastatin (Lipitor), but after a few months Sean stopped taking it—and anyway, we now know that protease inhibitors, and probably also delavirdine, can raise Lipitor in the blood to dangerous levels. We'll measure his lipids again next time. If they're elevated, I might recommend pravastatin (Pravachol).

SS: I'm surprised my cholesterol is high, because I got rid of my chickens on my farm in Pennsylvania and because I've been working out at the gym more than ever before. The chickens were great, but I found myself making four-egg omelettes every morning and then having eggs for lunch as well. Fresh eggs are incredible! But if Joe wants me to go on cholesterol-lowering drugs again, I probably will. I just have a hard time taking any meds other than what feels urgently necessary.

JS: Sean's testosterone is low, but so many HIV positive people have this deficiency—it seems to be part of the disease. In the past, he has sometimes used the gel. But now he says he prefers shots—which he gets when he feels like it.

SS: I'm not so sure I prefer shots, but I'm lackadaisical about using the gel. I see Joe rarely, but I try to get a shot when I do. The lack of libido bothers me, but I actually haven't had any problem Viagra can't fix. And working out has greatly improved my sexual function. I don't date much, so my biggest problem in that area actually has to do with opportunity, not performance.

JS: Moodwise, Sean seems to have acquired a beneficial attitude. He seems quite relieved by a recent event in his life.

SS: Joe is referring to my cathartic examination of my history of being sexually abused as a child [see “Sins of Transmission,” POZ, October 2002]. I know I sound like an Oprah cliché, but dealing with that has opened me up to a lot of growth. I was desperately searching for something my entire adult life. Now, at age 44, I’ve discovered it, and that’s given me a lot of power back.

Every day I feel incredibly lucky. I used to assume that the drugs would stop working at some point, but now I rarely fret about that. I may be living a dream with an ugly ending, but hope has been my sustenance for 20 years.

THE STRUB FILES

Throughout the ‘90s, top AIDS docs weighed in on Sean’s health roller-coaster for *POZ*:

August 1995. CD4 count: 6; Viral Load: 3.3 MILLION
“This is high-level viral replication going unchecked. Sean’s risk of an OI within the next year is high,” worries Michael Saag, MD. “Sean could achieve a lower viral burden by beginning antiretroviral therapy.” Soon after, Sean, a longtime AZT refusenik, starts ddI, which pushes his virus to 42,000. But pancreatitis pushes him off it—and virus rebounds to 705,000.

May 1996. CD4 count: 18; Viral Load: 63,000
Sean starts his first protease cocktail (Norvir and 3TC) in March, and by May his viral load has plummeted. Still, notes his longtime doc, Joe Sonnabend, “Sean has extensive KS on his face and in his lungs. He’s fatigued and has problems with his moods. He’s holding on, but he’s a very sick person.” Between the KS chemo and Norvir, Sean feels so lousy that two months later, Joe switches his cocktail to Crixivan, d4T and Rescriptor. He’s still on (and off) it.

March 1997. CD4 count: 77; Viral Load: undetectable
“After months of combination therapy,” reports Donald Abrams, MD, “Sean must be quite elated. He feels well with increased energy and improved KS”—and lungs are lesion-free!

July 1998. CD4 count: 154; Viral Load: 1 million
His CD4s reach 357 that spring, and then…“Sean initiated an unplanned drug holiday for two weeks,” explains Virginia Cafaro, MD. Once back on meds, Sean’s virus drops to 30K and, a year later, to undetectable—just as high cholesterol and facial lipo arrive. Still, “Sean’s story should be an encouragement to others,” writes Dr. Joe in the May ’99 *POZ*.
