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Antibacterial therapies aimed at killing these intra-cellular microbes have to contend with Jarisch-Herxheimer Shock (JHS) [28,29], "Jarisch-Herxheimer-Lukashevich syndrome" [30]. Mangin writes "patients are reporting periodic aggravation of their symptoms as an apparent direct response to the antibiotics .. these patients say that their treatment makes them feel much worse before they experience symptom-relief" [31].

Thus, JHS is at once a bad thing, because it exacerbates the suffering of the patient, and also a good thing, because it indicates that the bacteria are being effectively killed. In fact, it is our observation that those patients who do not experience significant JHS are not killing the bacteria at a rate fast enough to induce remission of their sarcoid inflammation. Out of our current subject cohort (n>100) only 5 patients have had significant difficulty finding an effective antibiotic regimen, and all have eventually been 'privileged' to suffer the effects of JHS.

Indeed, the greatest danger is that too powerful an antibiotic regimen will be put in place too soon, precipitating life-threatening JHS. Even while exercising great care, we have had two patients with life-threatening bradycardia and several with (temporary) debilitating pulmonary insufficiency. The two bradycardia events each lasted about two months and disappeared as the JHS subsided. In both cases the bradycardia was controlled with high doses of the Angiotensin Receptor Blocker, Olmesartan Medoxomil (Benicar/Olmetec).

A number of cases of skin rash have developed in subjects upon commencing the antibiotic therapy. For example, one subject reported that a skin rash was exacerbated when commencing the initial (minocycline) phase of the therapy. It cleared after 4 months but returned again (in a milder presentation) when Azithromycin was added. It disappeared again after another 3 months, and has not subsequently returned.