The RuffiniType-2 Corpuscle as an Explanation for Pain-only Decompression Sickness

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Introduction

The cause of pain in Type-1 (pain only) decompression sickness (DCS) remains elusive. Many explanations have been offered to explain why pain occurs in Type-1 DCS. To date, none adequately account for the pain only presentation and the clinical course–with its two *sine qua non* manifestations, namely:

- Universal absence of physical signs
- Immediate**,** dramatic pain relief with recompression

Joint pain is the predominant presentation in long duration dives… "Overwhelmingly the most common manifestation of DCS in saturation divers and aviators" (Francis & Mitchell)

We provide an explanation for the clinical observations and treatment responses based on the anatomy and physiology of the submicroscopic Ruffini type-2 sensory organelle.

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Background

Many hypotheses have been proposed to explain the etiology of pain only DCS, but none, heretofore, adequately explain the clinical course. Most of them... "Focus on an autochthonous bubble mechanism, but there is no agreement on the actual site" (Francis & Mitchell). Consider the following: (Red font indicates discrepancies, inaccuracies and/or inconsistencies with clinical observations)

- **Irritation of local nerve endings**--Clinical findings such as positive Tinel's sign and neuropathy are not observed in pain only DCS. Recovery of irritated nerve endings would be expected to be mediated with steroids or other anti-inflammatory medications and not disappear immediately with recompression
- **Bubbles in the joint**--Large bubbles inside joints do not produce symptoms (Vann & Thalmann) as is substantiated by gas insufflation of joints for arthroscopic surgeries and absence of gas in joints of pain only DCS patients with imaging studies (ultrasound and/or magnetic resonance)
- **Bubbles in articular cartilage**--Articular cartilage is devoid of nerve innervation. Pain from trauma, arthridities, degenerative joint disease, etc. is secondary to release of inflammatory mediators. These generate inflammation and pain in surrounding soft tissues with pain sensitive organelles such as synovium, joint capsule, retinaculum, tendon and ligament. Hyperbaric oxygen mediates some components of the inflammatory response such as vasodilation, but the effects are not as dramatic as pain relief for DCS with recompression alone nor would they be expected to occur so rapidly. Laboratory studies such as sedimentation rate and C-reactive protein would be expected to indicate the presence of inflammatory mediators
- **Bone marrow bubbles**--Possibly a source of pain, but generally silent until later manifestations such as bone marrow edema and/or microfractures develop. May be a precursor to dysbaric osteonecrosis (Lamphier). Magnetic resonance imaging virtually eliminates this as cause of pain only decompression sickness
- **Distention of bone marrow sinusoids**--Proposed as a link between bends and osteonecrosis (Walder). Sinusoids are devoid of sensory innervation. Sinusoid distention especially with bubbles would expected to have an identifiable "signature" with magnetic resonance imaging. Sinusoids are in marrow spaces rather than in the soft tissues around joints
- **Increased intramedullary pressure**--This has been postulated as a source of pain in bone tumors (Mercadante) and can be quantified by manometrics. The criticisms given for the three preceding hypotheses also apply to this explanation

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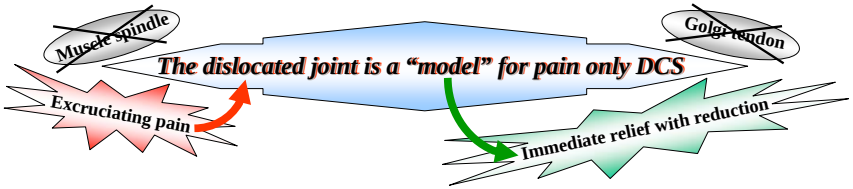
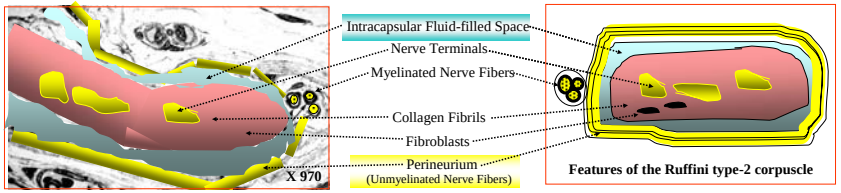
Evolution of the Ruffini Hypothesis

1. Hills-1970 Pain in decompression sickness is a mechanical problem… Hills statement is substantiated by the observation that immediate pain relief is the usual outcome with recompression whether with air or O_2 and is consistent with the mechanical reduction of a gas bubble predicated by Boyles Law. **Problem: Where is the gas bubble?**

2. Strauss-1980 There are four orthopaedic structures that are potential sources of pain in type 1(pain only) DCS: 1) Nerve stretch (e.g. sciatica), 2) Periostial elevation, 3) Muscle ischemia 4) Tendon & ligament stretch. Conclusions: Pain only DCS is a mechanical problem that involves relatively avascular structures. **Problem: What is the "avascular" structure?**



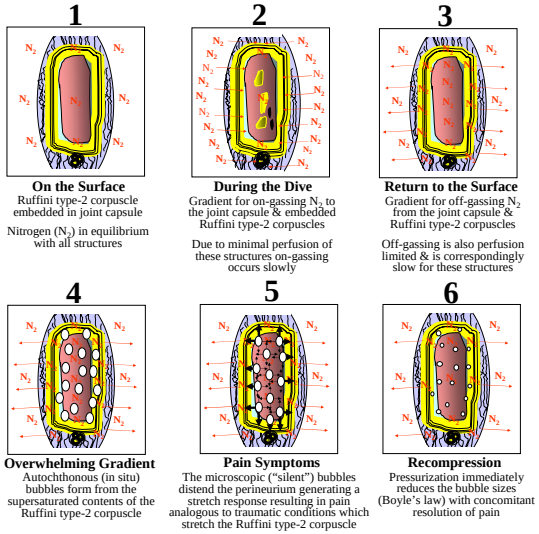
3. Halata-1984 The Ruffini type-2 corpuscle identified as a slow reacting pain sensor embedded in joint capsules with afferent C-fiber innervation. Stretching of the organelle generates the severe type of pain associated with dislocations, sprains, etc.



Solution: Ruffini Type-2 Corpuscle, the "Missing Link"

4. Strauss-1990 The Ruffini type-2 corpuscle is an "ideal model" for the pathophysiology of Type-1 (pain only) DCS. It is a pain sensing organelle that responds to stretch when its thick peripheral perineural capsule is distended. Its extensive intracapsular fluid-filled space is a site for autochthonous bubbles when the inert gas pressure differential is great enough. Once bubbles form in the microscopic intracapsular space, the perineurium distends & pain, without other detectable signs, occurs. Pressurization (recompression) immediately reduces the size/collapses the bubbles with immediate dramatic relief of symptoms.

A "Perfect" Fit



Conclusions

1. The histology & physiology of the Ruffini type-2 corpuscle explains the cause & character of pain… and the uniformly good responses to recompression observed with pain only DCS
2. The mechanism for bubble evolution in the Ruffini type-2 corpuscle fits Strauss's SPRM (Supersaturation Perfusion-Rate Model) explanation for autochthonous bubble formation
3. As imaging techniques improve, it is anticipated that the "silent" bubbles in the Ruffini type-2 corpuscle will become detectable …analogous to intravascular bubble detection with Doppler
4. "Silent" bubbles in adventitial tissues around nerves (and serosal membranes around abdominal organs) may explain other Type-1 DCS symptoms such as paresthesias, hypesthesias & fatigue

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