Rheumatology 2002;41:1070–1071

Hydrotherapy has had and has a rationale

Sir, Reilly and Bird’s [1] review of promising results of hydrotherapy such as swimming is tempered with scientific scepticism. Does this scepticism apply to all scientists?

By 1653 Olof Rudbeck in Uppsala, according to the Finnish physiologist Tigerstedt’s translations (Latin original at British Medical Association library in London) into Swedish [2], had found ligation of Asellio’s valvular lymphatics to induce swelling of the (lymph) glands and other tissues from which they stem. Familiar with Harvey’s circulation of blood from arteries to veins through invisible connections [2], Rudbeck found that hepatic lymph was filtered by some mechanism from blood, but he considered the function of lymph as yet to be shrouded in haze.

That haze had been dispelled by the time Ernest Starling at Guy’s Hospital clarified that hydrostatic and colloid osmotic pressure differences across capillary walls regulate exchange of fluid between plasma and connective tissues [3]. Starling found dropsy to consist in the accumulation of lymph, his term for the interstitial fluid filtered from blood [4: iii]. He had sought in vain for evidence for the absorption of ‘proteid’ by blood and concluded that escaping protein not used up by tissue elements must be collected by the lymphatics and restored to the blood by the thoracic or right lymphatic ducts. He found no lymph flow in resting limbs [4: iii].

Acknowledging his contemporary Tigerstedt’s contributions, Starling found that activity of muscle is followed by arterial dilatation, more plentiful blood supply and increased leakage of lymph and supply of protein to the cells. He found that hypertrophy involving not only growth of individual cells, but also multiplication of cells, can be brought about by increased supply of nutritive material, especially protein [4: i].

By 1900 it was known that (i) increased leakage from capillaries not compensated by lymphatic drainage, enhanced by movement, results in oedematous swelling of tissues, and that (ii) work increases local blood flow and leakage. Both have a bearing on hydrotherapy.

(i) Fisher [5] may have been the first to study lymphatics of (rabbit) joints. Potassium iodide absorption, enhanced by movement, may have taken place in synovial capillaries or lymphatics, or both. A colloid (colloidal silver) after 5 h of free movement, stained a synovial mesh of lymphatics, and also popliteal and other glands.
In the treatment of arthritic swelling (non-infective and non-tuberculous) Fisher found that a combination of rest and skilfully applied but not excessive movement may prevent ankylosis. In Fisher’s camera lucida drawings synovial bulges seem to be separated by a mesh [5], slightly wider than rabbit arteriolar–venular loops [6], of collecting lymphatics. Normal villi lack lymphatics [7]. The relationship between the microvascular loops, the parallel vessels (deep arteries?) in photographs [8] of Hunter’s [9] specimens, and lymphatics may not have been clarified [10]; this might elucidate the mechanism of lymph flow increase at change of joint angle linked to synovial fluid (SF) pressure [11], and the influence of arterial pulsation on lymph flow [12].

According to a review of lymphatic function [12], initial lymphatics, often provided with leaflets that function as one-way valves, are anchored to the interstitium by filaments. It seems widely accepted that traction by the filaments upon increase of interstitial fluid volume opens the leaflets and results in filling of initial lymphatics. The latter are considered to empty into collecting valvular lymphatics by intermittent external compression and pressure change on movement, massage, arterial pulsation, etc. Submersion of a limb in water increases pressure on and in it. Constant pressure in head-out-of-water immersion decreased lymph flow in spite of high interstitial pressure. Intermittent compression of sheep hooves increased lymph flow, but flow increase per compression fell when the interval between cuff inflations was reduced to below 8 s. Active movement, and passive motion at a frequency of 10–100 per min, increased lymph flow, etc. Fast walking did not increase lymph flow more than normal everyday activity. The possibility that lymph from positive tissue pressure compartiments such as the kidney and liver (swollen joints?) may be propelled into low-pressure ones could not be excluded. The review [12] might be useful for those who plan hydro- and physiotherapy.

(ii) In 1743 William Hunter [9] found it hard to believe that blood vessels could withstand the high pressures at loading of avascular joint cartilage, so he was not surprised by the large number of vessels in the soft (synovial) articular membrane. He could ‘not help observing that the Distribution of Blood-vessels to ... Cartilages ... seems calculated for obviating great Inconveniences’. He did not know if the vessels serve for nourishing (cartilage) only, or if they also ‘pour out a dewy (synovial) Fluid’. Both deductions were correct [11].

Recent data indicate that loading of cartilage increases the rate of processes involved in cartilage resistance to pressure [13] that may include protein synthesis [14]. Many of these processes require energy. Normally glycolysing cartilage might, at low SF glucose, start to oxidize fuel, possibly including amino acids [10]. In active muscle, Starling [4: i] found energy demands to be met by vasodilatory increase of capillary pressure and increased escape of (fluid and) protein. The conclusion agrees with a novel two-pore theory [15].

To sum up, (i) compression that increases tissue pressure and lymph flow occurs in any hydrotherapy that involves intermittent vertical movement of limbs in water. Swimming adds to this the exercise of muscles. The crawl and backstroke (at a slow pace?) ought to change pressures in forearms and hands more than breaststroke, and the former might spare neck (and low back?) joints. Effects on lymph flow of swimming, cheap and liked by patients [1], ought to be quantified. (ii) In many types of exercise, the cartilage of many joints is loaded more than in swimming. Does swimming minimize secondary vasodilatation [10] in the synovium?

British–Scandinavian co-operation might possibly benefit rheumatoid patients even more than it does today if attention were also paid to physiological research in this part of the world [2–7, 9–15]. Rudbeck has priority on lymphatic ligation-induced swelling, but this is, according to rather reliable Finnish and Swedish parish registers, also a homage to one of my many forefathers. Hydrotherapy theory might have evoked more interest had Starling not, because of bad timing, lost a Nobel prize for which he was twice a candidate.

J. AHLQVIST

Sibbvik, FIN-25830 Västanfjärd, Finland
Accepted 13 March 2002