

THE AQUATIC APE THEORY AND SOME COMMON DISEASES

M. J. B. VERHAEGEN *Medical Hypotheses* 24: 293-300 (1987)

Abstract

The Aquatic Ape Theory claims that human ancestors once lived in a semi-aquatic habitat. Some human diseases might be explained by our aquatic past. Such problems include hyperventilation, periodic breathing, laryngo- and bronchospasm, vasomotor rhinopathy, seborrhea, dandruff, male pattern alopecia, rhinophyma, osteoarthritis, inguinal hernias, varicose veins, common obesity, myopia, and ear-wax.

Key words

Cheyne-Stokes, Pickwickian syndrome, sleep apnea syndrome, SIDS, asthma, allergy, schistozomiasis, acne, seborrheic dermatitis, rosacea.

Introduction

According to Sir Alister Hardy's Aquatic Ape Theory (1), our ancestors a few million years ago spent a lot of time swimming and diving. Present-day humans have many rudimentary characteristics that support Hardy's theory (1-12). It is conceivable that some diseases may find their origins in our aquatic past. Such "ex-aquatic" diseases must be: almost uniquely human (absent from most non-human primates); largely hereditary (often polygenetic); and usually not very harmful. (Objections against diseases being selected for are answered by Dawkins (13): "A certain amount of bad bodies seems to be an almost inevitable consequence of selection for good genes, where good refers to the average effects of a gene on a statistical sample of bodies in which it finds itself permuted with other genes".)

Respiratory centres

Hyperventilation (HV) is prolonged rapid and deep breathing. Reflex HV in mammals occurs during exercise or hyperthermia (but panting in dogs is rapid, undep breathing which reduces body temperature). Intentional HV is seen in aquatic mammals and man before diving. HV crises, quite common in general practice, are often seen in emotional states. The prime cause may be habitually unstable breathing (14). In a patient who hyperventilates for a few minutes, the hypocapnia (reduced CO₂) causes cerebral vasoconstriction with dizziness and paresthesias, and the respiratory alkalosis (increased pH) can produce tetany after several minutes (15). Though the HV never lasted for more than one minute in our diving ancestor, the preparation for a dive was obviously a somewhat stressing moment. In modern humans after voluntary breathing for two minutes, there is a minute or so of apnea followed by periodic breathing as in Cheyne-Stokes respiration: a few breaths, several seconds of apnea, again a few breaths, etc. Marine mammals, too, have a cycle of HV before diving, apnea for several seconds or even minutes during diving, again HV at the water surface, etc. In man, Cheyne-Stokes breathing can occur in heart failure, extreme obesity (Pickwickian syndrome), brain injury, uremia, at high altitudes, during sleep (sleep apnea syndrome), and in premature infants (15-17).

Sleep apnea syndrome (18-20), very frequent in humans (mostly middle-aged males), can be obstructive (blockage of the oropharynx, often cyclic), central (cessation of respiratory movements), or mixed. The apneic episodes often last for 30 or 40 seconds, and can be accompanied by bradycardia (30/minute). It is correlated with obesity (especially a short, fat

neck), polycythemia, and pulmonary or systemic hypertension (20-21).

It is thought that some cases of Sudden Infant Death Syndrome (SIDS) are due to an “immaturity” of brain centres that control the breathing and heart rhythm. SIDS is believed to be a real danger in babies with central apneas longer than 15 seconds or with excessive periodic breathing.

Airway obstructions

Others say that SIDS is caused by a mixed central/obstructive apnea, or by an obstructive apnea. A newborn baby, like most – young and adult – mammals, has a high-positioned, almost intranasal larynx, but older children and adult humans have a descended larynx (Adam’s apple). This is possibly an aquatic adaptation (5-6). E. Crelin thinks that when a sleeping baby is startled and wakes suddenly, its “relaxed” (descended) larynx may slide back up to a position where the soft palate can block it and cut off the laryngeal air passage (22). Just as do some drowned porpoises entangled in fishing-nets, some SIDS victims show bleeding from the mouth.

In drowned humans, unlike animal models, the amount of fluid aspirated is usually small (23). Complete laryngospasm even occurs in about 10% of human drownings: it is assumed that, after gasping under water, the first gulp causes laryngeal obstruction, the victim never aspirates water (“dry drowning”), and some patients, even with wide, fixed pupils, have been resuscitated after more than half an hour (3,23).

Asthma (24) is a bronchial hypersensitivity that gives a reversible air-way obstruction mostly due to constriction of the bronchial musculature. Expiration is delayed, so that more air is kept in the lungs during an asthma crisis. Bronchospasm can be due to irritating inhalants, viral, helminthic and other infections, allergy, face immersion (25), and HV (exercise, emotion or voluntary HV). Hyperventilation reduces the temperature and saturation of the breathing-air. In susceptible individuals this induces bronchospasm, which can be a preparation for diving. Asthma seems to be unknown in apes, but according to T. B. Anderson, seals do have bronchoconstriction while diving (3,25). Deep-diving mammals (Weddell seals, many cetaceans) close the bronchi completely during diving (26).

We recently suggested that vasomotor rhinopathy, a hypertrophy and hypersensitivity of the plexus cavernosi of the inferior nasal conchae, which can suddenly block the nose passage, is an aquatic rudiment (11).

Nasal obstruction, asthma and laryngospasm can all be precipitated by allergy type I. This type of allergy is remarkably common in humans. Allergic asthma is a familial disorder, which combines a bronchial hypersensitivity with a hyperglobulinemia IgE (both total and specific IgE). Allergy type I is actually a strong immunologic defense mechanism against metazoa. Both the allergy and the metazoan infection typically induce local and often systemic hyper IgE. This activates the basophil white blood cells that start the allergic reaction by releasing several substances: while histamin, SRS-A, etc. cause vasodilation and itching, ECFA attract eosinophils to attack the antigen. C. Anderson recently demonstrated that in schistosomiasis, the T cells (lymphocytic white blood cells) that identify the worm produce interleukin IL-4. IL-4 induces proliferation of B cells that make specific IgE, and of eosinophils in the bone marrow (27). Hyper IgE and eosinophilia protect against mite (house dust allergy), worm (*Necator americanus*) and fluke infections (27-30). Fluke infections, unlike other worm infections, are usually water-born (*Schistosoma*, *Fasciolopsis*, *Fasciola*, *Clonorchis*, *Paragonimus*), and much more common in humans than in apes (31-32). In schistosomiasis, e.g., one of the world’s most important diseases, the penetration of the fluke larvae in wading or swimming people causes urticaria, with itching and scratching to remove the parasites. In endemic areas we may expect a greater diathesis for

allergic reactions upon a schistosome infection.

Head and trunk dermatoses

There is a group of skin and hair disorders of head and upper trunk that are very common, uniquely human, mainly hereditary, androgen-induced, non-itching, painless, and merely cosmetic (33-34).

Acne vulgaris, an inflammation of the sebaceous follicles (SFs), is more prevalent around puberty and in males. SFs exist only in humans and are activated by dihydrotestosterone (DHT). In the affected areas, the patients have an increased 5- α -reductase activity, that converts testosterone into DHT. SFs are mainly situated at the forehead, cheeks, chin, upper ventral chest, and between the shoulders. Recently we suggested that the sebum of the SFs once kept the hairs waterproof and well-fitting to the body, so that the male's neck was streamlined for a swimming life-style (11). A similar combination of hairs and sebum is shown by the adult male Steller's sea-lion *Eumetopias*.

Seborrheic dermatitis (35) is partly hereditary (caused by *Pityrosporum* yeasts belonging to our normal microflora), and more common in young adult men. It mainly consists of scales and scurf at the naked/hairy boundary of the scalp (dandruff), beside the nose, beneath the brows, inside and behind the ears, and presternally. When we sketch seborrheic dermatitis, SFs and head hairs (DHT often induces longer-growing brows, male pattern alopecia, and presternal hair) upon a reconstruction of a Pliocene male ancestor swimming under water (11), the localization is very remarkable and typical: scales at the origins of the hairs, and sebum under the shafts of the hairs. Since our ancestors never used shampoo, we may suppose that the scales stuck amid the hairs, and even made a scaly layer over them, as in serious cases of dandruff. Scales and hairs were glued together by the sebum oozed by SFs underneath the hairs.

Also in the axillar and ano-genital areas, where atypical localizations of acne and seborrheic dermatitis can be found, there possibly existed similar combinations of hairs, sebum and scales.

Rosacea, a mainly hereditary disease of middle-aged people, more common in women, consists of erythema, telangiectases, papules, pustules and seborrhea in the middle of the face. In men, it is often accompanied by rhinophyma, a hyperplasia of the soft tissues of the nose. The most similar nose is seen in the male proboscis monkey *Nasalis*, which is the best swimming and diving non-human primate (3,12,36). Remarkably, *Nasalis* infants have little upward-pointing noses, almost like human babies (12).

Figure – Upon a reconstruction of the head and upper chest of a swimming ancestor of *Homo erectus* (11), I have sketched the localization of the head hairs, seborrheic dermatitis and acne. The typical relations of hairs, scales and sebum are schematically represented on the right.

Absence of surrounding water

Humans are very prone to degenerative joint disease of spine and legs (osteoarthritis of cervical and lumbar vertebrae, hips and knees), and – in adult men – to inguinal hernias. Possibly, these disorders were less common in a previous watery habitat, because they seem to be due to our present-day vertical stance, and to the absence of the counter-pressure of surrounding water (3). The same can be said about varicose veins and their complications (ulcus cruris). The causes of these troubles – the upright posture and the occurrence of an extensive network of limb veins – may lie in our semi-aquatic past (11).

Also ordinary obesity, a mainly hereditary disease very common in humans, which worsens several other diseases (osteoarthritis, diabetes mellitus, heart failure, periodic breathing, bronchitis, etc.), could be an aquatic rudiment. All sea mammals are thick-bellied. In an aquatic habitat, even extreme obesity would have less complications (e.g. heart failure, osteoarthritis), since the fat is supported by surrounding water (11).

Sense organs

In myopia, or nearsightedness, the eyeball is too long. Myopia usually starts in puberty and is more prevalent in boys. Also whales, seals and penguins are nearsighted outside the water (37-38). This is an adaptation to the refractive power of water (1.33; air, 1.00).

Human ear-wax, consisting of impacted cerumen and skin scales, can completely occlude the ear canal, especially during showering or swimming, when the ear-wax absorbs water and swells, and even can hinder hearing. In whales the ear canal is completely occluded by a string of connective tissue, by scaling epidermal cells or by a horny wax plug (39): in water an open ear canal could be the source of infections, and does not improve hearing since the sound waves are not transmitted through the air.

Conclusion

The abundance of ex-aquatic features and a fortiori of ex-aquatic diseases in man is an indication for a rather recent (semi)aquatic phase in our evolutionary history. In my opinion, the ancestors of *Homo erectus* – perhaps no longer than two million years ago – were highly aquatic. Some of these possibly ex-aquatic disorders are more common in men, e.g. sleep apnea, acne, alopecia, dandruff, rhinophyma, myopia. Probably the aquatic customs of our predecessors – e.g. in food gathering – showed a marked sexual dimorphism.

Acknowledgements

I wish to thank Mrs E. Morgan, Dr D. F. Horrobin and Janssen Pharmaceutica for corrections and help.

References

1. Hardy A. Was man more aquatic in the past? *New Scientist* 7: 642, 1960.
2. Morgan E. *The descent of woman*. Souvenir, London, 1972.
3. ---. *The aquatic ape*. Souvenir, London, 1982.
4. ---. *The aquatic hypothesis*. *New Scientist* 1405: 17, 1984.
5. ---. *Sweaty old man and the sea*. *New Scientist* 1448: 27, 1985.
6. ---, Verhaegen MJB. *In the beginning was the water*. *New Scientist* 1498: 62, 1986.
7. Morris D. ch 1 in *The naked ape*. Cape, London, 1967.
8. ---. ch 58 in *Man-watching*. Cape, London, 1977.
9. Cunnane SC. *The aquatic ape theory reconsidered*. *Medical Hypotheses* 6: 49, 1980.
10. Gribbin J, Chervas J. p 163 in *The monkey puzzle*. Paladin, London, 1983.
11. Verhaegen MJB. *The aquatic ape theory: evidence and a possible scenario*. *Medical Hypotheses* 16: 17, 1985.
12. Ellis DV. *Proboscis monkey and aquatic ape*. *Sarawak Museum Journal* in press.
13. Dawkins R. p 52 in *The extended phenotype*. Oxford University Press, New York, 1983.
14. Lum LC. *Hyperventilation and anxiety states*. *Journal of the Royal Society of Medicine* 74: 1, 1981.

15. Ganong WF. p 503 in Review of medical physiology. Lange Medical Publications, Los Altos, California, 1981.
16. Chatton MJ, Ullman PM. Obesity. p 780 in Current medical diagnosis and treatment (CH Kempe, ed) Lange Medical Publications, Los Altos, California, 1981.
17. Brazie JV, Lubchenko LO. Periodic breathing. p 60 in Current Pediatric diagnosis and treatment (CH Kempe, ed) Lange Medical Publications, Los Altos, California, 1972.
18. Brophy JJ. Sleep apnea. p 638 in Current medical diagnosis and treatment (MA Krupp, ed) Lange Medical Publications, Los Altos, California, 1981.
19. Manson RM, Rushing JL. Diseases of the trachea. p 111 in Current medical diagnosis and treatment (MA Krupp, ed) Lange Medical Publications, Los Altos, California, 1981.
20. Guilleminault C, Cummiskey J, Dement WC. Sleep apnea syndrome: recent advances. *Advances in Internal Medicine* 26: 347, 1980.
21. Przybylski, Sabbah HN, Stein PD. Why do patients with essential hypertension experience sleep apnea syndrome? *Medical Hypotheses* 20: 173, 1986.
22. Nisbett A. The origin of cot deaths? *The Listener* 1.5.86: 17, 1986.
23. Chatton MJ. Drowning. p 944 in Current medical diagnosis and treatment (MA Krupp, ed) Lange Medical Publications, Los Altos, California, 1981.
24. Manson RM, Rushing JL. Asthma. p 115 in Current medical diagnosis and treatment (MA Krupp, ed) Lange Medical Publications, Los Altos, California, 1981.
25. Mukhtar MR, Patrick JM. Ventilatory drive during face immersion in man. *Journal of Physiology* 370: 13, 1986.
26. Slijper EJ. p 168 in *Walvissen. Centen*, Amsterdam, 1957.
27. Newell J. How the body puts the pressure on parasites. *New Scientist* 1510: 27, 1986.
28. David JR, Goetzl EJ, Austen KF. Immunology. p 219 in *Pathophysiology, vol I* (LH Smith Jr, ed) Saunders, Philadelphia, 1981.
29. Weir DM. p 135 in *Immunology*. Churchill Livingstone, Edinburgh, 1983.
30. Gleich GJ, Adolphson CR. The eosinophilic leukocyte. p 177 in *Advances in immunology* (FJ Dixon, ed) Academic Press, Orlando, 1986.
31. Goldsmith RS, Trematode (fluke) infections. p 876 in Current medical diagnosis and treatment (MA Krupp, ed) Lange Medical Publications, Los Altos, California, 1981.
32. Redmond I. Parasitologic research. app G p 258 in *Gorillas in the mist* (D Fossey) Hodder and Stoughton, London, 1983.
33. Hammerstein J, Lachnit-Fixson U, Neumann F, Plewig G, eds. *Androgenization in women*. Excerpta Medica, Amsterdam, 1980.
34. Rees RB. Seborrheic dermatitis and dandruff. p 51 in Current medical diagnosis and treatment (MA Krupp, ed) Lange Medical Publications, Los Altos, California, 1981.
35. Burton JL, Pye RJ. Seborrhoea is not a feature of seborrhoeic dermatitis. *British Medical Journal* 286: 1169, 1983.
36. Napier JR, Napier PH. p 158 in *The natural history of the primates*. British Museum, London, 1985.
37. Slijper EJ. p 274 in *Walvissen. Centen*, Amsterdam, 1957.
38. Martin G. Through a penguin's eye. *New Scientist* 1447: 29, 1985.
39. Slijper EJ. p 247 in *Walvissen. Centen*, Amsterdam, 1957.