

cular but glabrous men with small genitalia, and around dull normal intelligence (Méndez et al, *J Endocrinol Invest* 1995; 18:205–13).

Body hair and mesomorphism were rated, using reference sketches and pictures, in 51 self-identified Caucasian homosexual men, in a matched sample of 100 men from the general Caucasian population, and in all clearly printed pictures of 380 Caucasian men, aged 25–54, in Sheldon's Atlas of Men. Homosexuals were more hirsute ($P < 0.01$) but less muscular ($P < 0.001$); the controls were not different. Several samples of Caucasian and South Asian men were more hirsute ($P < 0.01$ and $P < 0.001$) than sufficiently matched control samples, either of less educated or with lower IQs. Three published studies (Danforth & Trotter, *Am J Phys Anthropol* 1922; 5: 259–65; Henry GW, *Sex Variants N.Y.*, Harper & brs. 1948; Lookingbill et al, *J Clin End Metab* 1991; 72:1242–8), done with different objectives, apparently confirm the findings.

Lower testosterone to dehydrotestosterone ratios may have something to do with the predisposition/development of homosexuality and intellectual giftedness in the male.

A SOCIAL CONSTRUCTIONIST RESEARCH APPROACH TO THERAPEUTIC NEGOTIATIONS BETWEEN FAMILY MEMBERS AND STAFF TEAM WHERE A FAMILY MEMBER HAS BEEN CHARACTERISED AS MENTALLY ILL

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A qualitative, social constructionist study of the accounts generated by family members and the staff team of reflective process meetings (Andersen 1990).

Given that the research aim was to provide information relevant to social constructionist practice, it was considered essential to use an inquiry paradigm consistent with the staff team's world view. This consideration suggested a constructionist methodology grounded in the experience of the researcher. Data collection and analysis was as follows: Meetings between families and the staff team were observed from behind a one way screen and field notes were recorded. Focussed group discussions with both the family members and the staff team around video material from the sessions were audiotaped and transcribed. Simultaneously, taking the view that what is described in the research has no separate existence outwith the researcher's lived experience (Steier 1991) the researcher kept a reflexive diary recording her relationship to the research and her emerging perceptions during the analysis of the data. Analysis of the data was by way of a grounded theory approach (Glaser & Strauss 1967).

Some provisional theoretical categories have emerged from the ongoing discussions and analysis of families' accounts of their meetings with the staff team. From these accounts, and subsequent discussions that have now begun with the staff team, some apparent contrasts regarding the different world views that family members and the staff team bring to the sessions have emerged. Of particular interest are questions relating to the respective theoretical positions taken by family members and the staff team.

The study raises further questions: i) How is one to proceed with a social constructionist research paradigm when classical methods of research make positivist assumptions about an independent final reality which can be known through investigations? ii) How can social constructionist conclusions be made relevant to an orthodox clinical practice which is based on positivist assumptions about privileged expert knowledge of an independent final reality?

MANAGEMENT OF A 25 YEAR OLD PATIENT WHO PRESENTED WITH AN ERECTILE PROBLEM AND LACK OF SEX DESIRE, AND WHO HAD A LOW DIHYDROTESTOSTERONE (DHT) BLOOD LEVEL

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A 25 year old man was seen at St. George's Hospital Sexual Dysfunction Clinic, London, together with his 29 year old wife for his erectile problems and lack of sexual desire. The couple, of Pakistani origin, married one year ago in an arranged marriage but the marriage had not been consummated yet.

The couple's knowledge of sex issues and their previous sex experience were found to be very poor. A clinical examination of the husband revealed a rather small penis and small testes, however normal hair distribution. A further laboratory investigation showed normal testosterone, SHBG, testosterone/SHBG ratio, prolactin, FSH, LH and caryotype, but low dihydrotestosterone (DHT) = 0.94 umol/l (normal range 1.3–2.5). The couple was offered sex therapy and the husband Caverject injections and tbs Mesterolone treatment for a short period. There was a marked improvement of the erectile problem but not remarkable results regarding his lack of sexual desire.

The management of this rare case is discussed and issues regarding further investigations and examinations, cultural issues, relationship problems, the wife's perception of the problem and other therapeutic options are addressed.

SPREADING DEPRESSION IN DENTATE GYRUS AT DIFFERENT BRAIN TEMPERATURES

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Spreading depression (SD) is a pathophysiological event of epileptic seizures. The role of temperature in the development of SD is unclear so far. We investigated how the time of SD onset changed at different brain temperatures and in dependence on preceding electrically induced epileptiform activity. In our experiments SD was elicited in dentate gyrus at electrical stimulation (trains of 10 s, pulse width 0.1 ms, biphasic, 80 V, 20 Hz, every 10 min) of perforant path in seventeen urethan-anesthetized (1.2 g/kg) rats. In 52 cases (13.7%) of 379 the electrically induced afterdischarge (AD) were followed by SD-negative a slow potential shift. SD was 20 ± 2 s (mean \pm s.e.m.) later than the AD end. The onset of SD was associated with paroxysmal firing of high-amplitude population spikes. The interval between the end of stimulation and the SD onset estimated as a latency of SD. Relations between the AD duration, the SD latency, and values of brain temperatures (within range 37–33°C) were analyzed. Positive correlation was observed between the AD duration and the SD latency ($r = +0.50$, $P < 0.001$). The AD duration correlated negatively with the brain temperature ($r = -0.37$, $P < 0.01$). The SD latency did not correlate with the brain temperature ($r = -0.13$, $P > 0.05$). Also the relations between the AD duration and period from the AD end to the SD onset revealed negative correlation ($r = -0.36$, $P < 0.01$). The data indicate: 1. that the afterdischarge interferes with the generation of SD the time of onset of which may reflect the cessation of refractory state to paroxysmal activity; 2. brain temperature probably also exerts some effect on the SD onset by means of the influence on the afterdischarge.