volunteers who are probably not menstruating and are of ovulatory status.

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Differential rates of cerebral maturation between sexes

TAYLOR and Ounsted[1,2] have proposed a model to account for the age of onset of developmental disorders and their relative incidence between the two sexes. They propose that there is a period in the maturational continuum during which the child is particularly vulnerable to a particular disorder. Males develop more slowly than females and they should therefore reach this "dangerous state" later, and take longer to pass through it. It is therefore predicted for any developmental disorder, that males should show a later age of onset and a higher incidence than females[2]; but that females are more likely to suffer seriously than males[1], (although this does not follow directly from the model). Evidence relevant to these predictions is reported here for congenital hydrocephalus.

Medical records are available for 48 children who were referred to the Neurosurgical Unit at the Radcliffe Infirmary, Oxford, over the past 20 years, and who were diagnosed as suffering from congenital hydrocephalus. In most cases there was no associated spina bifida cystica or other complex multiple congenital deformities. For each patient, it was established whether surgical intervention had been necessary, or whether the hydrocephalus had arrested spontaneously (Table 1).

The series contained 33 males and 15 females. This is equivalent to a sex ratio of 220 males for every 100 females, and differs significantly from equiprobability according to the binomial test ($z = 2.56$, $P < 0.01$, two-tailed test). The median age at which the boys were referred was 26 weeks; the median age at which the girls were referred was 7 weeks. The Mann–Whitney U test demonstrated that the boys were significantly older when referred than the girls ($z = 1.69$, $P < 0.05$, one-tailed test).

In the case of 23 of the children (48%), the hydrocephalus arrested spontaneously. This compares closely with a figure of 46% reported by Laurence and Coates[3] from a much larger series. Considering boys and girls separately, it was found that spontaneous arrest occurred in 17 (52%) of the boys, and in 6 (40%) of the girls. Thus, the girls were more likely to require surgical intervention than the boys. The difference is not statistically significant, however, according to a χ^2 test which used the correction for continuity ($\chi^2 = 0.18$, d.f. $= 1$, $P > 0.5$). The median age at which a patient was referred if his hydrocephalus arrested spontaneously was 48 weeks; the median age at which he was referred if surgery was necessary was 17 weeks. The difference between these two medians approaches statistical significance, according to a Mann–Whitney U test ($z = 1.82$, $P < 0.1$, two-tailed test). Both groups, however, showed the effect of sex on the age at which they were referred, described above.

Table 1 Number of patients referred, by sex and age at referral

	Age (months)							
	0	1	2	3–5	6–8	9–11	12–23	24 and over
Males	1	5	3	7	5	3	5	4
Females	5	4	0	0	1	1	2	2

These results support the two principal hypotheses which can be derived from the model proposed by Taylor and Ounsted[2]. Congenital hydrocephalus occurs in boys later and more often than it does in girls. This constitutes further evidence for two generalisations in the epidemiology of disease: females are susceptible earlier than males; yet males are more susceptible than females. The model proposed by Taylor and Ounsted links these two generalisations by a single formal mechanism.

If the necessity for surgical intervention can be taken as a sign of the severity of hydrocephalus, there is also some evidence for their assertion that diseases tend to occur more severely in females than in males. This evidence is, however, not supported by statistics. Nevertheless, the findings of this study suggest that research into developmental disorders may increase our understanding of the nature of sex differences and the processes of cerebral maturation.

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Nude mice with normal thymus

MICE homozygous for the mutation, nude (*nu*) have been reported to have no thymus[1], although they retain a prelymphoid thymic rudiment[2]. Here, I report the occurrence of individuals that are by external signs (including hairlessness) a nude mutation but possess an apparently normal thymus.

The outbred nude stock in this laboratory has been repeatedly reinforced with $+/nu$ heterozygotes and some fertile nu/nu homozygotes (Laboratory Animals Centre, Carshalton). The ensuing appearance of 'nudes with thymus' suggested that this phenotype originated from Carshalton, but the alternative could not be excluded that either a new mutation or some infiltration of a hairless gene might have happened in this laboratory. Similar findings were communicated to me by Dr D. Wakelin of the Wellcome Laboratories for Experimental Parasitology at the University of Glasgow, who had obtained his breeding stock from me. A Carshalton origin, however, seemed to be confirmed when in November, 1974, I obtained from there a large group of nude mice aged about 3 months. Out of over 60 autopsied so far there was one with an apparently normal thymus and one with a minute 'thymus'. These tissues are being examined.