A retrospective study was made of 40 consecutive children admitted to the children's ward with febrile convulsion not associated with campylobacter enteritis. The mean age of these children was 22 months (range: 5 months to 4 years 2 months).

Comment

Convulsion as a manifestation of campylobacter enteritis has not been reported. The nine children in our series who had convulsions had the unusually high mean age of 5 years 8 months, which is outside the range normally associated with febrile convulsions. Rutter and Smales³ reported a mean age for febrile convulsions of about 22 months, which is similar to that in our own small series.

Convulsion is known to occur with shigellosis. The incidence is about 10%, and convulsions may occur in children with only slightly increased or normal body temperatures.⁴

There are several explanations that may partly account for this unusual incidence of convulsion and high mean age: firstly, the outbreak largely affected schoolchildren and not younger children; secondly, the outbreak was large with 223 proved cases in children aged 10 years or less, of whom only 14 were admitted to hospital and only nine had convulsions; and thirdly, campylobacter, like shigella, may predispose children to convulsions.

¹ Skirrow MB. Campylobacter enteritis: a "new" disease. *Br Med J* 1977;iii:9-11.

² Communicable Disease Surveillance Centre. Explosive outbreak of campylobacter enteritis. *Communicable Disease Report* 1979;No. 13.

³ Rutter N, Smales ORC. Role of routine investigations in children presenting with their first febrile convulsion. *Arch Dis Child* 1977;52:188-91.

⁴ Smith DH. Shigellosis. In: *Nelson textbook of pediatrics*. Philadelphia: W B Saunders, 1975:602-6.

(Accepted 11 January 1980)

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Paget's disease of bone in two American cities

Paget's disease is rarely found among African blacks.¹ Although it is known to occur among blacks in the USA the prevalence has not been documented. We carried out a radiological survey of the prevalence of the disease in hospital patients in two American cities to compare prevalences in blacks and whites. One of the cities selected was in the north and the other in the south to investigate further a suggestion that prevalence is lower in the south.²

Methods and results

The cities selected were New York and Atlanta. In each city a survey was made in two hospitals, one privately and one publicly funded, serving mainly white and black patients respectively. Samples of about 1000 abdominal radiographs of people aged 55 and over were taken from the files of stored films in each hospital. The sampling procedure was the same as that used in earlier surveys.^{3,4} Only patients whose films showed the pelvis, sacrum, femoral heads, and all lumbar vertebrae were included. Diagnosis was by one observer (PBG) using the same standardised criteria used in the previous surveys.

The medical records were consulted to determine the patients' ethnic group. At each hospital medical records were available for all patients with Paget's disease and for more than 80% of those without the disease. The ethnic distribution of the remaining patients was assumed to be identical with that of the patients whose records were available. In Atlanta all but one of the patients whose notes were consulted were found to be classified as either black or white; but in New York about 5% were of other ethnic groups and were excluded from the survey.

The table shows the prevalence of Paget's disease by sex and ethnic group in New York and Atlanta. Each prevalence was standardised⁵ for age against the aggregate population of a large British survey,³ so that the results could be compared with those already reported for Britain³ and Australia.⁴ In each city the prevalence in blacks was similar to that in whites. In addition the

Prevalence of Paget's disease by ethnic group. Numbers of cases are given in parentheses

City	Ethnic group	No of patients	Age-standardised prevalence (%)		
			Men	Women	Both sexes
New York	Black	950	3.3 (12)	2.0 (12)	2.6 (24)
New York	White	1082	5.2 (30)	2.5 (13)	3.9 (43)
Atlanta	Black	1111	1.9 (11)	0.6 (3)	1.2 (14)
Atlanta	White	1563	0.9 (7)	0.8 (6)	0.9 (13)

blacks showed a higher prevalence among men, similar to the well-documented male preponderance of the disease in European whites. The higher prevalence in New York than in Atlanta was shown by both sexes and both ethnic groups. Previous surveys have shown that the radiological prevalence of Paget's disease is higher in radiographs taken specifically to show the skeleton than in other radiographs of the pelvis. The proportions of skeletal radiographs were, however, similar in the two cities and ethnic groups (blacks in New York 12%, in Atlanta 12%; whites in New York 13%, in Atlanta 15%).

Comment

In contrast to the position in black Africans, in whom Paget's disease is rare, the condition is similarly prevalent in blacks and whites in the USA. This finding provides further evidence of the influence of environment in the aetiology of the disease. The difference between the northern and southern cities, found in both sexes and in both ethnic groups, indicates a pronounced variation in prevalence within the USA. The figure for the whites in New York is comparable with the lowest values found in a survey of 14 towns in Britain,³ and similar to the prevalence in Perth, Australia (3.6%).⁴

We thank Armour Laboratories (USA) and the Wessex Regional Health Authority, who generously funded the study; Professor J Farman and his colleagues of the Downstate Medical Centre, Brooklyn, New York, and Dr L Clements and his colleagues of Emory University, Atlanta, for their help and kindness.

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² Rosenbaum HD, Hanson DJ. Geographic variation in the prevalence of Paget's disease of bone. *Radiology* 1969;92:959-63.

³ Barker DJP, Clough PWL, Guyer PB, Gardner MJ. Paget's disease of bone in 14 British towns. *Br Med J* 1977;ii:1181-3.

⁴ Gardner MJ, Guyer PB, Barker DJP. Radiological prevalence of Paget's disease of bone in British migrants to Australia. *Br Med J* 1978;ii:1655-7.

⁵ Bradford Hill A. *A short textbook of medical statistics*. London: Hodder and Stoughton, 1977.

(Accepted 5 December 1979)

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Eclampsia in multipara

Three consecutive eclamptic patients seen by me at the Ikedife Hospital within one week were multiparous. Routine history taking revealed changes in paternity in the first two cases. This led me to make confidential inquiries about the actual paternity in the pregnancies of other eclamptic multiparous patients.

Patients, methods, and results

Forty-six eclamptic multiparous patients with singleton pregnancies were seen between August 1968 and July 1978 in a rural practice centred on Nnewi, Anambra State, Nigeria. The Ikedife Hospital serves as an unofficial reference centre for 22 small maternity hospitals within a radius of 35 km. I have excluded in this series three multiparous patients brought in dead after eclamptic fits, two cases of epilepsy, two other patients whose blood pressure was still over 160/90 mm Hg six weeks postpartum, and one uncontrolled diabetic multiparous patient who had fits during the last week of pregnancy. The 46 patients were confidentially interviewed by me

about the *true* paternity of the pregnancy that was affected with eclampsia. They were interviewed either antepartum, postpartum, or at a postnatal visit, but always after confidentiality had been established. The questioning was either direct or oblique, depending on my assessment of the patient's personality. The placenta was examined after delivery in all cases to exclude gross placental abnormalities such as hydatidiform mole coexisting with a normal fetus. To assess the prevalence of change of paternity in this area two random groups of 300 and 200 pregnant multiparous patients were questioned in 1971 and 1977 respectively.

Thirty-four (74%) out of the 46 multiparous eclamptic patients seen in my practice during the 10 years admitted that they had had a new partner for the affected pregnancy. Four were not sure, and eight were certain that there was no new factor in the affected pregnancy, which varied between their 3rd and 8th. In 20 cases the fact of a new partner was easily elicited during routine history taking, and subsequent interview was unnecessary. These new partnerships were owing either to death of the previous husband, separation, divorce and remarriage, unfaithfulness, or unmarried multiparous patients (Nnachi custom practised by the Igbo people). So far 14 of these patients have had subsequent deliveries under my supervision. Three had oedema and albuminuria but none had a recurrence of eclampsia. Two of them reported another change of partner.

Of the random groups of 300 and 200 women questioned in 1971 and 1977, respectively, for change of paternity, 31 and 12 had had changes in the preceding three years. Out of these 43, one had had intrapartum eclampsia and three had had only albuminuria and ankle oedema in their pregnancies.

Comment

The cause of gestosis remains enigmatic but may have an immunological basis.¹ The disease is associated with many and profound changes in the maternal state.² One view widely held is that primigravida patients are more commonly affected than multigravida. In Nigeria we certainly see multiparous patients with eclampsia. In this study of 46 multiparous eclamptic patients with singleton pregnancies 34 had had new partners for their pregnancies. The pregnancies in these multiparous patients became complicated by eclampsia, which is otherwise commoner in primigravida patients. For each particular new couple, however, the pregnancy was primary, irrespective of the gravidity of the woman. Whatever the aetiological factors, the manifestational sequence of gestosis seems the reverse of rhesus sensitisation, in which the chances of being affected increases with the number of pregnancies. This study suggests that when a multiparous patient with a singleton pregnancy develops eclampsia it is likely to be a primary pregnancy.

I thank Professor W I B Onuigbo, University of Nigeria Teaching Hospital, Enugu, Nigeria, for his advice and criticism.

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² Leading article. Immunological factor in pre-eclampsia. *Br Med J* 1976; ii:604-5.

(Accepted 10 January 1980)

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Mittelschmerz is a preovulatory symptom

Many women experience mid-cycle lower abdominal pain (mittelschmerz) during ovulatory cycles. It may be caused by intraperitoneal release of follicular contents at ovulation or by muscular cramps in the uterus, tubes, or large bowel,¹ but the exact relation of mittelschmerz to ovulation has not been defined. We investigated its timing as part of a study of the periovulatory period using ultrasonic techniques.

Patients, methods, and results

A group of 96 women were studied. All had regular ovulatory menstrual cycles and were attending the reproductive biology clinic of the Royal Women's Hospital. From the mid-follicular phase of the cycle (day 9-11) onwards blood was drawn daily to measure luteinising hormone (LH)

concentration by rapid radioimmunoassay, and each patient was questioned about her symptoms during the preceding 24 hours. The diameter of the developing follicle was measured in three planes and a mean calculated using either a static B-scanner (Diasonograph N E 4200) or a real-time device (A D R Model 2130).² This procedure was continued daily until the follicular appearances of ovulation² were first noted.

Thirty-four women (35%) noticed lower abdominal pain, usually lasting six to 12 hours, during the mid-cycle period. In 27 discomfort was localised to one or other iliac fossa, which in all but two cases corresponded to the

Relationship of mid-cycle pain to the peak plasma LH concentration (day 0) in 34 women

	<Day -2	Day -1	Day 0 (LH peak)	Day +1
Timing of pain relative to peak LH concentration	2	2	25	5*

*Four of these patients did not ovulate on ultrasound evidence until day +2.

side of the developing follicle. In seven patients the pain was central and suprapubic. Most patients (77%) felt pain on the day of the peak plasma LH concentration (table). When the data were analysed in relation to the ultrasonically determined time of ovulation it was found that in 31 (91%) the pain had occurred in the previous 24-48 hours. The mean ($\pm$ SD) follicular diameter on the day of pain was 19.3 ± 2.2 mm. Ovulation was confirmed in every cycle by a mid-luteal 24-hour urinary pregnanediol content of greater than 2.0 mg,³ and four of the cycles were conceptual. Pain did not last longer than 24 hours in any patient and none required analgesia.

Comment

In all but one patient, whose pain might have coincided with follicular rupture, the intact follicle could still be seen after the pain had disappeared. As in previous studies,² the plasma LH peak concentration preceded "ultrasonic ovulation" by about 24 hours. In most cases mittelschmerz coincided with the plasma LH peak—that is, when the follicle was still enlarging. Nevertheless, simple follicular distension is unlikely to be the cause of pain since even on the day of peak volume many follicles were distorted by distension of the bladder, suggesting a low intrafollicular pressure. Furthermore, in anovulatory cycles cystic follicles may become much bigger without producing pain. In mammals intrafollicular pressures are low and no significant preovulatory pressure differentials have been noted.⁴ On the other hand, the mid-cycle LH rise induces increased contractility in ovarian perfollicular smooth muscle,⁵ an effect which is partially mediated through production of prostaglandin $F_{2\alpha}$. This may be the origin of mittelschmerz.

The recognition of mittelschmerz as a preovulatory event has useful implications. It may help to identify the most fertile day of mid-cycle, and thus help the timing of coitus or artificial insemination, and may also improve the efficacy of natural family planning techniques.

¹ Jeffcoate TNA. *Principles of gynaecology*, 4th ed. London: Butterworth, 1975:544.

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³ Pepperell RJ, Brown JB, Evans JH, Rennie GL, Burger H. Investigation of ovarian function by measurement of urinary oestrogen and pregnanediol excretion. *Br J Obstet Gynaec* 1975;80:321-33.

⁴ Espey LL, Lipner H. Measurements of intrafollicular pressures in the rabbit ovary. *Am J Physiol* 1963;205:1067-72.

⁵ Wright KH, Wallach EE, Fromm E, Jeutter DC. Studies of rabbit ovarian contractility using chronically implanted transducers. *Fertil Steril* 1976;27:310-8.

(Accepted 18 January 1980)

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