to protect against death from malarial infection by prompt haemolysis of infected erythrocytes.

Another feature of homozygous Hb O disease is that the three patients were all of small stature and had young, almost childish faces.

The inheritance of homozygosity for Hb O Arab in this family was also associated with another rare recessive inheritance for conjugated hyperbilirubinaemia of the Dubin-Johnson type. The total coproporphyrin concentration in the urine of the three patients and that of their brother was within normal limits. A preponderance of isomer I varying in total between 76% and 85% clearly suggested, however, the Dubin-Johnson syndrome.[3] In all cases liver function tests gave normal results.

One of the largest surveys conducted in Saudi Arabia did not detect Hb O Arab.[4] The finding of Hb O Arab in isolated cases in other countries is probably the result of gene transmission from the Sudan in these countries through historical links.[5]

We thank Professor Sir David Weatherall for confirming the finding of Hb O Arab and Mrs Samia Bakhit for secretarial help.

1 Ranot B, Fisher S, Remez D, Schneerson R, Kahane D, Ager JAM, et al. Haemoglobin O in an Arab family; sickle cell haemoglobin O trait. BMJ 1960;ii:1262-4.
2 Vella F, Beale D, Lehmann H. Haemoglobin O Arab in Sudanese. Nature 1966;209:309.
3 Koskelo P, Toivonen I, Adlercreutz H. Urinary coproporphyrin isomer distribution in the Dubin-Johnson syndrome. Clin Chem 1967;13:1006-9.
4 Lehmann H, Maranjian G, Mourant AE. Distribution of sickle-cell haemoglobin in Saudi Arabia. Nature 1963;198:492-3.
5 Coqulet ML, Jaeger G, Mullender N. Anamolies de l'hémoglobine et données médico-biologiques chez 10 000 Africans. Nouv Rev Fr Hematol 1978;20: 465-77.

Side of origin of epithelial ovarian cancer

Catherine B Johannes, David W Kaufman,
Lynn Rosenberg, Julie R Palmer, Paul D Stolley,
John L Lewis Jr, Ann G Zauber,
M Ellen Warshauer, Samuel Shapiro

Slone Epidemiology Unit, Boston University School of Medicine, Brookline, MA 02146, USA
Catherine B Johannes, PHD, epidemiologist
David W Kaufman, SCD, assistant director
Lynn Rosenberg, SCD, assistant director
Julie R Palmer, SCD, epidemiologist
Samuel Shapiro, FRCPED, director

Clinical Epidemiology Unit, Department of Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA 19104, USA
Paul D Stolley, MD, professor of medicine

Gynaecology Service, Department of Surgery, Memorial Sloan-Kettering Cancer Center, New York, NY 10021, USA
John L Lewis Jr, MD, gynaecologist

Memorial Sloan-Kettering Cancer Center, Box 44, New York, NY 10021, USA
Ann G Zauber, PHD, assistant attending epidemiologist

Cancer Care and Research Program, New York Hospital, Box 17, New York, NY 10021, USA
M Ellen Warshauer, MA, director

Correspondence to:
Dr Johannes.

Incessant ovulation has been suggested to increase the risk of ovarian malignancy because of repeated trauma to the ovarian epithelium.[1] Recently Cruickshank hypothesised that if this is so tumours should arise more often on the right than on the left side[2] as ovulation occurs more frequently on the right.[3] Among 214 cases of epithelial ovarian cancer he found that significantly more tumours (59%) arose in the right ovary. We investigated the hypothesis with data from our case-control surveillance study.[4]

Patients, methods, and results

Patients aged 18-69 years admitted to hospitals in several metropolitan areas of the United States and Canada with ovarian cancer as well as other selected conditions were administered standard questionnaires on personal characteristics, medical history, habits, and use of medication.[4] Hospital records were abstracted for information on the diagnostic and histological classification of ovarian cancer. Patients included in this analysis had been interviewed from October 1976 to June 1989. All cases of epithelial ovarian cancer, stage I or greater, that occurred in women with both ovaries intact at diagnosis and for which a pathology report was present were reviewed (341 of 612 cases).

The side of origin was determined by two independent reviewers—one who knew the hypothesis and one who did not. The reviewers agreed on 289 (85%) of the cases. (Results were similar whichever reviewer's classification was used, so knowledge of the hypothesis did not appear to influence the results.) The 52 cases classified differently were decided on by a third reviewer, who knew the hypothesis. (Results were similar whether or not these 52 cases were included in the analysis.) Proportions were compared by using the one sample test of significance for binomial proportions.[5]

The median age of the 341 women reviewed was 51 years; 314 were white. The stage was determined in 253 cases (74%): 66 stage I, 25 stage II, 124 stage III, and 38 stage IV.

The side of origin could be determined for 264 cases. In the others widely disseminated disease made identification of the side impossible. The proportions with right and left unilateral tumours were similar (table). A somewhat greater proportion of the bilateral tumours were right than left dominant (dominance was determined according to the side with the larger tumour) but the difference was not significant ($z=1.7$, $p=0.1$). Thus the totals for tumours of right and left sided origin were 141 and 123 respectively ($z=1.1$, $p=0.3$).

Distributions according to age, race, parity, and ages at menarche, first pregnancy, and menopause were similar for right and left sided tumours. The stage could be determined for 95 of the patients with right sided and 91 patients with left sided tumours. Similar proportions of women with right (76; 54%) and left sided (68; 55%) disease presented at a late stage (III or IV).

Comment

Our results give scant support for Cruickshank's hypothesis. We found that 53% of the determinable tumours were of right sided origin compared with Cruickshank's 59%.[2] Although his results were statistically significant and ours were not, the 95% confidence interval for our estimate (0.47 to 0.59) includes his estimate; thus we cannot rule out his findings. In both studies the findings were mainly accounted for by right sided dominance in bilateral tumours. As Cruickshank discusses, it is necessary to make the unproved assumption for bilateral disease that the side with the larger tumour is the side of origin.

Cruickshank found that a significantly higher proportion of patients with left sided tumours (60%) presented at an earlier stage (I and II); we observed no difference. Cruickshank's study may have been subject to observer bias because the single reviewer who examined the records was aware of the hypothesis. Such bias is unlikely in our study as cases were classified by two reviewers, one of whom was unaware of the hypothesis. On the other hand, his study was population based and selection bias was unlikely; we could not exclude the possibility of such bias.

We conclude that the hypothesis that epithelial ovarian cancer arises more commonly on the right remains unproved. Since both studies are consistent with a possible right sided predominance for bilateral tumours, however, this relation should be examined further in a larger series.

hospitals: Beth Israel, Newton-Wellesley, Mount Auburn, and Massachusetts General Hospitals, Boston; Memorial Sloan-Kettering Medical Center, New York, and Lennox Hill Hospitals, New York; University of Pennsylvania, Hahnemann, Lankenau, Medical College of Pennsylvania, Montgomery, Pennsylvania, Presbyterian, and Thomas Jefferson Hospitals, Philadelphia; and Johns Hopkins Hospital, Baltimore. Supported by contracts 223-76-3016, 223-80-3001, and 226-82-0007 and cooperative agreements UO1 FD01222 and FD-U-000082 from the Food and Drug Administration and by grant 5 R37 CA45762 from the National Cancer Institute.

1 Fathalla MF. Incessant ovulation—a factor in ovarian neoplasia? Lancet 1971;ii:163.
2 Cruickshank DJ. Aetiological importance of ovulation in epithelial ovarian cancer: a population based study. BMJ 1990;301:524-5.
3 Potashink G, Insler V, Meizner I. Frequency, sequence, and side of ovulation in women menstruating normally. BMJ 1987;294:219.
4 Slone D, Shapiro S, Miettinen OS. Case-control surveillance of serious illnesses attributable to ambulatory drug use. In: Colombo F, Shapiro S, Slone D, et al. eds. Epidemiological evaluation of drugs. Littleton, Massachusetts: PSG Publishing, 1977:59-74.
5 Snedecor GW, Cochran WG. Statistical methods. 6th ed. Ames, Iowa: Iowa State University Press, 1967:211.

School attendance as a factor in deliberate self poisoning by 12-15 year old adolescents

L McGibben, C G Ballard, S Handy, W R Silveira

Department of Psychiatry, Walsgrave Hospital, Coventry CV2 2DX
L McGibben, MRCPSYCH, senior registrar in child and adolescent psychiatry
C G Ballard, MB, registrar in psychiatry
S Handy, MRCPSYCH, registrar in psychiatry
W R Silveira, MRCPSYCH, consultant child and family psychiatrist

Correspondence to:
Dr McGibben.

Published reports on deliberate self poisoning among school age adolescents are scarce. We found only one study, in North America, discussing the importance of the school term as a possible risk factor.[1] We studied the influence of school terms on deliberate self poisoning among schoolchildren from Coventry.

Subjects, methods, and results

We studied the medical records of all schoolchildren aged 12-15 in Coventry who had been admitted to hospital for deliberate self poisoning and hence referred for child psychiatric assessment. The completeness of data was assessed by cross checking the records by name and date with the list in the hospital admissions unit and with discharge summaries from paediatric wards.

Details of the dates of school terms were obtained from Coventry education department, and the rates of hospital admission for deliberate self poisoning were calculated per 1000 schoolchildren aged 12-15 per year for school terms and holidays. The relative risk of adolescents being admitted for deliberate self poisoning during school terms compared with during holidays and its 95% confidence interval were adjusted for the length of school holidays and terms. The various school holidays were compared by the same method.

The average number of 12-15 year olds in Coventry during 1982-90 was 18 265. Of these, 380 were admitted to hospital for deliberate self poisoning, 340 (89%) having been referred to the child psychiatric services.

The 95% confidence intervals of the relative risk showed that there were significantly more hospital admissions during school terms than holidays (table). There were also significantly fewer admissions during the Christmas and summer holidays than during school term time, and there was a non-significant trend for

Rates of deliberate self poisoning and relative risks of its occurring during school terms among adolescents aged 12-15 in Coventry, 1982-90

	Rate (per 1000/year)	Relative risk (95% confidence interval) during school term
School term (n=292)	2·86	
School holidays:		
Overall* (n=48)	1·06	2·73 (1·68 to 4·66)
Christmas (n=1)	0·13	22·23 (7·5 to 395·4)
Easter (n=14)	1·94	1·49 (0·97 to 2·24)
Summer (n=25)	1·27	2·28 (1·40 to 3·78)

*Including half term holidays.

fewer admissions during the Easter holidays. When the different holiday periods were compared there were fewer admissions during the Christmas holidays than during either the Easter holidays (relative risk 14·92, 4·95 to 265·07) or the summer holidays (9·77, 3·13 to 170·72); there was also a non-significant trend for fewer admissions during the summer holidays than the Easter holidays.

Comment

As almost 90% of the adolescents in our study were referred for child psychiatric assessment we consider that the medical records gave a sufficiently reliable estimate of deliberate self poisoning among 12-15 year old adolescents.

We chose an upper age limit of 15 because after 16 a young person is exposed to a more adult environment, with different expectations, responsibilities, and pressures from those of childhood. Also, 16 was the lower age limit in studies of parasuicide in adults.[2]

We found appreciable differences between the rates of deliberate self poisoning during school terms and holidays, the rate of admission being lowest during the Christmas and summer holidays. We tested seven comparisons for significance, which increases the risk of a spurious positive result. Most of the significant differences were, however, substantially above the conventional 95% level.

The data suggest that two main factors are operating. The larger is the influence of Christmas holidays in substantially reducing the rate and the smaller the direct influence of school attendance in increasing it. Possible risk factors that may increase the risk during school terms are the stress of school work, the competitive nature of much schooling, difficulties in fitting into a peer group, and bullying. Teachers' vigilance may have led to a slight increase in referral rates during school terms but it was probably not a major confounding factor. Alternatively, school terms may coincide with a seasonal variation. The data did not suggest a spring or autumn peak other than that limited to school holidays. This absence of seasonal variation is consistent with other data.[3]

The reduction in risk during the Christmas holidays may reflect the strength of family cohesion then. Both factors favour a sociological rather than a biological explanation and support the view that holiday times are a time of reduced sense of isolation[4] and of enhanced family purpose.

1 Christoffel KK, Marcus D, Sagerman S, Bennett S. Adolescent suicide and suicide attempts: a population study. Pediatr Emerg Care 1988;4:32-40.
2 Masterton G. Monthly and seasonal variation in parasuicide. Br J Psychiatry 1991;158:155-7.
3 Papathomopoulos E, Vlachos P, Kalamara D, Papadatos C. Suicidal attempts by ingestion of various substances in 2050 children and adolescents in Greece. Can J Psychiatry 1989;34:205-10.
4 Durkheim E. Suicide: a study in sociology. London: Routledge and Kegan Paul, 1952. (Translated by J Spaulding and C Simpson.)