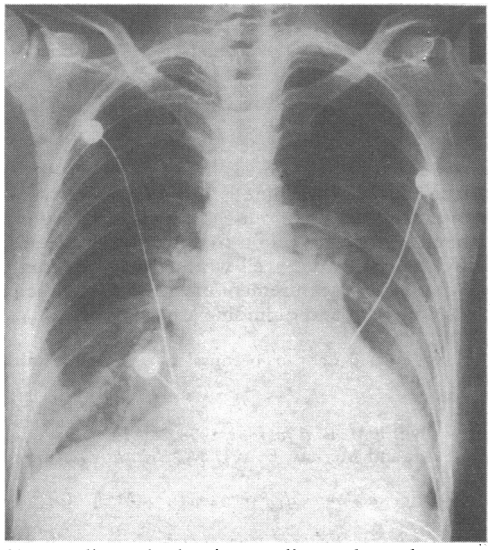
When seen in March 1977 she was well and managing her job. She had no signs of heart failure and the prosthetic sounds were normal. Four weeks later she suddenly developed severe chest pains and was admitted. She was in fast atrial fibrillation and was hypotensive. On auscultation the opening and closing clicks of the prosthesis could be heard and there were coarse crepitations at the lung bases. An electrocardiogram showed changes of an inferolateral myocardial infarction, and a chest radiograph showed cardiomegaly with pulmonary oedema (see figure). A provisional diagnosis of coronary embolism and cardiogenic shock was made, and, despite conservative treatment, she died the following day, 21 hours after her acute collapse.

At necropsy the Bjork-Shiley prosthesis was completely detached and lay free in the left atrium. The sutures were intact around the prosthesis ring although they were slightly loose. The edges of the mitral annulus were frayed but no thrombus or vegetations were present. Histological examination showed no evidence of infection or calcification, and there were no myxoid changes.



Chest radiograph showing cardiomegaly, pulmonary oedema, and an abnormal position of the Bjork-Shiley mitral prosthesis.

Comment

This is the first reported case of complete dehiscence of a Bjork-Shiley prosthetic valve. Embolism of the poppet or occluder from a prosthetic valve presents with pulmonary oedema, which is usually fatal within 36 hours,[3] although, in some, surgical correction has been described.[4] Incomplete prosthetic dehiscence, which is more common, has a less acute course and is associated with myxoid change,[2] calcification,[1] or infection affecting the valve annulus.[5] It is uncommon when the annulus is fibrosed from rheumatic heart disease.[2] There was no evidence of these predisposing factors in our patient.

The lack of clinical features suggesting valve malfunction failed to alert us to the diagnosis, which, if recognised, might have led to surgical correction. We suggest that all patients with prosthetic valves presenting with pulmonary oedema should be carefully assessed for serious malfunction of the prosthesis.

We thank Mr D B Clarke for permission to report this case.

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Spina bifida: early antenatal diagnosis using amniotic fluid alpha-fetoprotein heterogeneity

Affinity chromatography of human α-fetoprotein (AFP) on concanavalin-A-sepharose (con-A) yields two types of protein[1]—namely, a con-A-binding fraction (type *a*), which adheres to the lectin, and a con-A-non-binding fraction (type *b*), which does not. Type *b* contributes only 2-6% of the fetal serum, whereas the proportion of type *b* in second-trimester amniotic fluid ranges from 15-45%.[1] This difference is potentially useful for the early antenatal diagnosis of neural tube defects. In such cases the concentration of type *b* in the amniotic fluid is usually low, probably because of fetal serum leaking into the fluid.[2 3] We have assessed the importance of this finding by testing a series of samples wrongly diagnosed from assays of total fluid AFP.

Methods and results

Amniotic fluid samples obtained by amniocentesis were assayed for total AFP concentration and stored at −20°C. Reassay after storage showed no changes in values. Affinity chromatography on con-A-sepharose (Pharmacia) was performed essentially as described,[2] without the operator knowing the origin of the sample. Total AFP and the fraction passing through the column were measured using radioimmunoassay and the proportion of type *b* thus calculated.

Fifty-two samples of second-trimester amniotic fluid from normal singleton pregnancies were analysed. The mean proportion of type *b* detected was 25·6% ± SD from mean 9·4% (range 10·0-55·1%). No consistent change in values occurred with advancing gestation. Four samples that had been diagnosed as abnormal because total AFP concentrations exceeded five standard deviations above the mean contained a normal proportion of type *b*—that is, over 10% (table). In a fifth sample both total AFP concentration and the proportion of type *b* suggested an abnormal fetus, which was not confirmed at birth. Six further samples from pregnancies complicated by spina bifida that had been missed on total AFP assay were diagnosed correctly from the proportion of type *b* detected—that is, under 10%. In three of these six cases uncertainty over gestational age had contributed to the wrong diagnosis.

AFP concentrations in amniotic fluid samples that were false-positive or false-negative on total AFP assay

Sample No	Gestation (weeks)	Total AFP concentration (SDs above mean)	Con-A-non-binding AFP (% of total)	Outcome of pregnancy
1	17	5·1	19·5	Normal
2	19	10·0	18·1	Normal
3	19	7·2	29·2	Normal
4	21	6·1	37·6	Normal
5	18	19·7	9·1	Normal
6	17	4·1	8·7	Spina bifida
7	18	4·8	8·9	Spina bifida
8	19	2·7	8·6	Spina bifida
9	17 by scan	1·0	6·0	Spina bifida
	22 by dates	4·8		
10	17 by scan	1·1	5·2	Spina bifida
	20 by dates	3·3		
11	18 by scan	2·2	8·6	Spina bifida
	21 by dates	7·5		

SD = Standard deviation.

Comment

Though measurement of total amniotic fluid AFP concentration for the early antenatal diagnosis of open neural tube defects is reliable and widely used, it has several minor limitations.[4] Chief among these is the overlap of concentrations in normal pregnancies and those where the fetus has open spina bifida. In my experience, a cut-off value of three standard deviations above the mean results in a false-positive incidence of about 1% and a failure to diagnose 3% of open spina bifidas. At a cut-off value of five standard deviations above the mean the false-positive incidence falls to 0·2% but 10% of open spina bifidas are missed. Hence there is an urgent need for supplementary diagnostic procedures.

Measurement of concentrations of con-A-binding and con-A-non-binding fractions of amniotic fluid AFP may fulfil this need. Throughout gestation fetal serum AFP contains a low proportion of the con-A-non-binding fraction as compared with amniotic fluid.[1] If the increased concentrations of total amniotic fluid AFP associated with open neural tube defects originate from leakage of fetal serum into amniotic fluid[5] this AFP might be expected to be enriched in con-A-

non-binding fraction and the composition of amniotic fluid altered accordingly. This is apparently so.

Since affinity chromatography is more complicated than conventional AFP assay it will probably be used on selected samples. If the method proves as reliable as suggested here, an appropriate strategy would be to lower the cut-off value to three standard deviations above the mean total AFP concentration and subject all samples with concentrations above this value to con-A-binding studies. Theoretically, this would give a high incidence of detection of open spina bifida with a specificity of nearly 100%.

I am grateful to Sandra Brown and Lilias Barron for excellent technical help. The work was supported by a grant from the Medical Research Council.

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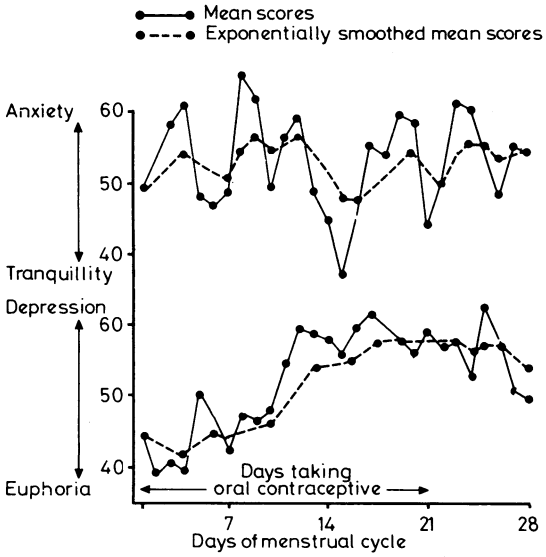
Cyclical variations in mood in normal women taking oral contraceptives

A significant number of women taking oral contraceptives develop pathological changes in their mood over a period of several months.[1] Little, however, is known of the mood changes within the menstrual cycle of normal women taking oral contraceptives.

Subjects, methods, and results

Twelve volunteers were studied. All were healthy women aged between 22 and 30. They were all taking oral contraceptives containing not more than 30 μg of ethinyloestradiol per tablet and were happy with this method of contraception. None had changed the brand of their oral contraceptive within three months of the start of the study. None had any history of mental illness. Each recorded an indication of her mood and affect every evening for 30 days. This was done by using visual analogue self-rating scales.[2] These were forms, one for each day, on which were two 10-cm lines separating statements describing extremes of mood (euphoria to depression) and anxiety level (tranquillity to anxiety). Before retiring for the night the subjects marked each of the lines in the position between the two sets of statements that best described their state of mind at the time. Subsequently, by measuring the position of the mark on the lines, numerical scores from zero to 100 for depression and anxiety for each subject each day were produced. To minimise any bias effects each woman started keeping records on a different day in the menstrual cycle and the records from the first two days for each woman were not analysed. The results from the group as a whole were pooled, and mean scores for depression and for anxiety for each day of the menstrual cycle, counting day 1 as the first day on which an oral contraceptive pill was taken, were calculated. The mean scores were then analysed by computer, calculating exponentially smoothed scores for each day and using Cembrowski *et al's*[3] modification of Triggs's technique for trend detection. There was a steady increase in mean depression scores while the subjects were taking oral contraceptives. Triggs's technique showed that when the depression scores were smoothed so that the exponential mean corresponded to a moving average containing seven observations this trend was significant ($P < 0.05$) from days 11 to 19 of the cycle. The anxiety self-rating scores did not show any significant trend throughout the cycle.



Variation of mean mood and anxiety level scores during menstrual cycle.

Comment

The steady increase of the self-rating scores for depression during the menstrual cycle is consistent with the theory that depression associated with oral contraception has a metabolic basis. The metabolic lesion may be a functional deficiency of pyridoxine.[4] Thus it would be of interest to investigate the effect, if any, that pyridoxine has on these cyclical mood changes. There was no tendency for anxiety self-rating scores to increase towards the end of the cycle, and this would support the use of oral contraceptives for the treatment of premenstrual tension.

I thank the volunteers for their co-operation in this study.

A listing of the computer program in BASIC used for the calculation of Triggs's statistic and for exponential smoothing is available from the author.

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How to evaluate papers given at medical meetings: Use of the SPEAKER Index

The objective finding that fewer than half the speakers at a surgical meeting were able to communicate effectively[1] confirms subjective experience that the standard of oral presentations at medical meetings is disappointingly low. There are many reasons why speakers communicate poorly. Two are interrelated: few doctors are taught how to communicate the results of their work (whether by the spoken or the written word), and most doctors therefore evaluate talks subjectively rather than objectively. In teaching doctors how to speak effectively, I emphasise the need for objective evaluation of oral presentations, and one result of this emphasis has been the development of the SPEAKER Index.

The Index

The SPEAKER Index is an aid to evaluating the performance of speakers. The mnemonic acronym SPEAKER refers to the initial letters of the seven primary performance characteristics that are listed in part 1 of the Index (see figure). These primary characteristics may be rated from low to high on a five-point scale contained on the first side of a single sheet; the overall rating may be displayed numerically, by calculating a mean score, or graphically, by joining numerical points. The other side of the sheet, which is given over to part 2, lists the many secondary characteristics that can be subsumed under the primary characteristics. These secondary characteristics may be