platelets. The blood in most hospital transfusion departments is usually at least two to three days old. Arrangements should be made to obtain freshly drawn blood (less than six hours old) from the regional transfusion centre; alternatively, donors from a local or national panel should be bled.

Hypofibrinogenaemia is best controlled by using freeze-dried fibrinogen. Each bottle contains 1.5-2.5 g of dried product, which is usually reconstituted in 200 ml of sterile water. If there is a problem with fluid overload the amount of water can be reduced to 100 ml.

Thrombocytopenia should be treated with platelet concentrates. In most hospitals there is a delay while platelet concentrates are being prepared or transported from the regional transfusion centre. Each unit consists of the platelets from four to five donors. Two units should be given as soon as they are available.

At this point the clinical and haematological effect of replacement treatment should be assessed. If bleeding continues heparin treatment should be tried. The dosage is started at the rate of 10 000 units every 24 hours by constant intravenous infusion or 5000 units subcutaneously every 12 hours. This may

be cautiously increased if indicated. Heparin treatment should be stopped when the platelet count reaches $100 \times 10^9/l$ ($100\,000/mm^3$).

Erythrocytosis

Carcinoma of the kidney, other renal tumours, polycystic kidneys, and a hydronephrosis may be accompanied by an increase in the red cell mass. The incidence of erythrocytosis in carcinoma of the kidney has been variously reported as 1-10%. Erythropoietin may be produced by some tumours. The increased erythropoietin concentration, however, is more likely to originate from the distorted renal tissue surrounding the various lesions. Removal of the diseased kidney results in the red cell mass returning to normal.

Renal transplantation is usually followed by a progressive rise in packed cell volume. It is not uncommon for the haemoglobin concentration to reach 17-18 g/dl or higher. Sometimes patients must be treated by venesection to avoid thromboembolic episodes.

Clinical Topics

Menstruation and control of diabetes

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Summary

Seventy per cent of the patients aged 45 years or under and suffering from diabetic ketoacidosis who were seen in one diabetic clinic over five years were women. The association of menstruation with ketoacidosis was assessed over two and a half years, and it was found that menstruation was associated with ketoacidosis more often than would be expected by chance ($P < 0.01$).

Two hundred women were interviewed and 76 observed that menstruation changed their diabetic control. Fifty-three found that control deteriorated and hyperglycaemia occurred, while 23 found that control improved and hypoglycaemia was a common problem.

Menstruation appears to be an important factor in influencing control of diabetes. The mechanism of the changes observed has not yet been determined, but it seems to be a subject worthy of further investigation.

Introduction

Several textbooks^{1,2} indicate that control of diabetes may be impaired around the time of menstruation. These observations

are based largely on clinical impressions and are supported by occasional case reports of severe and sometimes fatal ketoacidosis occurring at the onset of menstruation.^{3,4} They do not, however, indicate the frequency of changes in diabetic control at the time of menstruation, nor do they indicate whether changes other than hyperglycaemia and ketoacidosis occur. Clearly such information is necessary so that patients can be advised about this problem and acute metabolic disturbances minimised. We undertook a study to determine the type of changes in diabetic control that occur at menstruation and their frequency.

Patients and methods

The study was carried out in three parts.

The incidence of diabetic ketoacidosis was assessed over five years (1971-5) and related to sex and age. Diabetic ketoacidosis was defined as uncontrolled diabetes requiring intravenous fluid and electrolyte replacement, the presence of ketonuria ++ or +++, and a standard bicarbonate concentration of 17.5 mmol (mEq)/l or less.

Association between diabetic ketoacidosis and menstruation—This was determined in women who were admitted from January 1974 to June 1976. Throughout this time we took a detailed menstrual history from all except nine of the women with ketoacidosis once they had recovered from the acute metabolic disturbance. All the patients who complied with our criteria for ketoacidosis were included in this part of the study, except those in whom menstruation had stopped because of pregnancy, hysterectomy, or the menopause. Six patients whose presenting feature of diabetes was diabetic ketoacidosis were also excluded, though it is noteworthy that ketoacidosis was associated with menstruation in five of them. Diabetic ketoacidosis was considered to be associated with menstruation when menstruation was present on admission or on either of the two days before or after admission.

Questionnaire—In the third part of the study 200 consecutive women

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aged under 46 years who attended the diabetic clinic were interviewed by a doctor. They completed a standardised questionnaire, which recorded their observations on the effect of menstruation on diabetic control. During the interview particular attention was paid to the results of urine tests throughout the menstrual cycle. The occurrence of symptoms of hyperglycaemia or hypoglycaemia was noted, and any adjustment in insulin dose at the time of menstruation was recorded. Changes in activity or dietary habits before or during menstruation were also noted. The use of anovulatory agents was also recorded, and their effect on diabetic control was assessed on the basis of urine tests, symptoms, and the need to change the insulin dose. Only patients in whom diabetes had been diagnosed for at least six months and who had at least four menstrual periods a year were included in this part of the study.

Results

The number of patients suffering from one or more episodes of ketoacidosis in 1971-5 is shown in table I. In both age groups diabetic ketoacidosis occurred more often in women than in men.

TABLE I—Number of patients suffering from one or more episodes of ketoacidosis (1971-5)

	Total No of patients	No ≤45 years old	No >45 years old
Women	129	87	42
Men	56	33	23
Women:men	2.3	2.6	1.8

From January 1974 to June 1976 there were 53 episodes of diabetic ketoacidosis (in 48 patients) that could be assessed in relation to menstruation, and over half these episodes were associated with menstruation (table II). Two patients were using anovulatory agents and had just completed a course when the ketoacidosis occurred. In no patient was ketoacidosis associated with menstruation more than once. Twenty-two episodes of ketoacidosis were not associated with menstruation, but an adequate menstrual history was not recorded in nine cases. To avoid bias, these were considered to have been unassociated with menstruation, though in some of the patients there were strong reasons for suspecting that menstruation was in progress.

TABLE II—Details of the 53 episodes of ketoacidosis according to whether or not they were associated with menstruation

	Total No of episodes	Mean duration of diabetes	Mean blood sugar (mmol/l)	Mean H ⁺ (nmol/l)	Mean bicarbonate (mmol/l)
Menstruating	31	17.7 years (±6.19)	28.8 (±11.9)	78.4 (±27.5)	10.0 (±3.75)
Non-menstruating	22	14.1 years (±4.9)	25.1 (±9.9)	63.3 (±13.0)	12.1 (±2.75)
P value				<0.02	<0.05

Conversion: SI to traditional units—Blood sugar: 1 mmol/l ≈ 18 mg/100 ml. Bicarbonate: 1 mmol/l = 1 mEq/l.

Among the 31 cases in which ketoacidosis was associated with menstruation an apparent precipitating factor was found in only 16 cases (52%) (infection in 15 cases and insulin error in one). In contrast, an apparent precipitating factor was observed in 82% of cases in which there was no association (infection was a precipitating factor in 11 cases and insulin errors or other causes in seven). The differences were significant ($P < 0.01$).

Seventy-six out of the 200 women interviewed thought that their diabetic control changed around the time of menstruation. Fifty-three of these (26.5%) felt that diabetic control deteriorated—that is, glycosuria increased and ketonuria was observed by those who tested for it. Thirst and polyuria were also common, and visual disturbances were occasionally noticed. These features usually became apparent one to three days before menstruation occurred, though sometimes

they occurred as much as a week before. Almost invariably the changes were abruptly reversed as soon as menstruation was established.

The other 23 patients thought that their diabetic control improved around the time of menstruation—that is, glycosuria diminished and they tended to have hypoglycaemic episodes. In one patient these were so severe that she had to stop driving for three days before each period. Another patient was admitted to hospital with hypoglycaemic convulsions at the start of every period. Though all patients were questioned about dietary habits at the time of menstruation, no evidence emerged to suggest that this was a factor in the alteration of diabetic control.

Of the 48 patients (out of 194 on whom information was available) who had used oral contraceptives since their diabetes was diagnosed 39 observed no deterioration in diabetic control. Three patients actually found that using oral contraceptives corrected the altered diabetic control that they had previously noted at the time of menstruation. Of the nine patients whose diabetic control was upset by anovulatory agents six noted symptoms of hyperglycaemia, which required increases in the insulin dosage of up to 16 units daily. Two patients experienced recurrent hypoglycaemic reactions while using anovulatory agents, and in one patient control became extremely unstable, with episodes of hypoglycaemia alternating with periods of hyperglycaemia. These three patients discontinued the anovulatory agents of their own accord after one cycle.

Discussion

Our finding that diabetic ketoacidosis occurs more often in women than in men agrees with previous observations.^{2,5} This female preponderance is greatest in the younger patients. In patients aged 45 years or less we observed a female:male ratio of 2.6, which is the same as that recorded by Cramer.⁶ But about 50% more men than women in this age group attend our clinic, so the female preponderance is even more striking. The excess of women over men among the older patients is probably explained by the fact that diabetes in this age group is more common among women. Also, about two-thirds of the insulin-requiring patients (and therefore presumably those who are more prone to ketosis) among the older age group attending our clinic are women.

Our observations indicate that menstruation may have a profound effect on control of diabetes. This may vary from recurrent hypoglycaemia, on the one hand, to hyperglycaemia and even ketoacidosis, on the other.

Previous reports have emphasised the occurrence of hyperglycaemia,^{3,4,7,8} but our present findings show that hypoglycaemia, though less common than hyperglycaemia, is also a real problem. It may be severe enough to seriously disrupt the daily routine and require hospital admission. The frequency with which changes in diabetic control occur around the time of menstruation does not seem to be generally recognised. About 40% of the patients interviewed were aware of altered diabetic control, but this is likely to be an underestimate. Eighty-seven per cent of patients who found that control became worse tested their urine regularly, while only 57% of those who observed no effect of menstruation on diabetic control did so. If more of the latter group had tested their urine then a higher proportion would probably have observed at least minor alterations in control. We found no relation between observations of altered diabetic control and the occurrence of dysmenorrhoea in these patients.

On our criteria for associating ketoacidosis with menstruation the two events would be expected to be associated by chance alone in 32% of cases. We observed such an association in 58% of cases ($P < 0.01$), which suggests that menstruation may predispose to or precipitate ketoacidosis. This is further supported by the observation that other factors that might predispose to the metabolic upset were found less often ($P < 0.01$) in patients in whom ketoacidosis and menstruation were associated than in those in whom they were not associated. Infection was a precipitating factor in more cases in our series than in others. This difference is not easily explained, but we

were dealing entirely with women in a limited age range. There is also some evidence that infections are more common around the time of menstruation.⁹ It might be argued that the ketoacidosis precipitated menstruation. But this is unlikely; in only three cases in which ketoacidosis and menstruation were associated did menstruation occur at an unexpected time. In addition, menstruation was associated with admission in only 30% of a control group of non-diabetic women admitted to this hospital with acute illnesses.

It is well recognised that behavioural changes, accidents, and hospital admissions may increase in the premenstrual period and during menstruation.⁹ Might premenstrual tension have therefore predisposed to errors of insulin administration in the patients in whom ketoacidosis and menstruation were associated? Detailed questioning of the patients indicated that error in insulin administration was a factor in only one of these patients. Most of the patients had undergone at least one pregnancy while diabetic, and the close supervision and discipline then had made most of them careful and reliable patients.

It might be considered that to include patients with a standard bicarbonate of 17.5 mmol/l or less allows the inclusion of patients with very mild ketoacidosis. This is true, but our main object was to define a group of patients in whom there was an unequivocal acute metabolic disturbance. All of the patients in this study had such a disturbance, which required intensive treatment. The acidosis was more severe in the patients in whom it was associated with menstruation (table II). Indeed, in 20 of these patients the initial bicarbonate level was 10 mmol/l or less, and it was 15 mmol/l or less in 27 cases. On the other hand, the standard bicarbonate concentration was 10 mmol/l or less in only seven of the 22 patients in whom menstruation and ketoacidosis were not associated. Had we included only patients with severe ketoacidosis the association between menstruation and ketoacidosis would have been much greater.

Our observations underline the influence of menstruation on diabetic control. All diabetic women likely to menstruate should be routinely educated about this problem and encouraged to

determine, by testing urine and recording symptoms and dates of menstruation, the effect of menstruation on their diabetic control. In our experience, if changes in diabetic control do occur, the same pattern of changes almost always recurs in any individual. Our present advice to patients encountering symptoms of hyperglycaemia is to increase the dose of insulin in the days before menstruation. It is important that the dose be reduced again when menstruation starts, otherwise hypoglycaemia may be a problem. Though this advice is usually helpful, it is sometimes unsuccessful as menstruation is not always as regular as many women believe. In patients in whom hypoglycaemia is a problem, reduction of the dose of insulin may help, though again unexpected menstruation can lead to difficulties. Occasionally, anovulatory agents may lessen severe hypoglycaemic episodes and this beneficial effect may be maintained after they have been discontinued.

The average woman has 13 menstrual periods a year for about 30 years. The effect that menstruation may have on diabetic control is therefore clearly important. We are at present carrying out further investigations in an attempt to elucidate the mechanism of the changes we have observed.

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Are there any health hazards associated with the use of saunas, either to attendant staff or to users?

The health hazards of sauna baths were highlighted when 60 people were reported to have died taking saunas in Finland in 1970. Before then most scientific investigations were confined to countries in which their use is part of the cultural scene—notably Finland—but major investigations have since been reported from Britain¹ and the US.² The two types of sauna in common use are the Finnish ("dry") sauna and the Turkish, Japanese, or steam bath ("wet") sauna. The risk from heat strain in both types is similar, except for exposure of less than 10 minutes, in which the wet sauna probably carries a lesser risk.² There is an element of risk to people who are prone to coronary insufficiency, and researchers are unanimous in recommending "a cautious approach for the middleaged and elderly sauna novice,"¹ with a time limit of five minutes in high-temperature saunas, which are common in Britain, and 10 minutes in the cooler saunas of Scandinavia. The Finnish sauna, however, produces a training effect with regular use. Apart from sensible time limits, safeguards include careful control over temperature, a half-hour rest before entering the bath, and adequate hydration (at least 250 ml water) before and after exposure. Possibly everyone over 45 should undergo a thorough physical examination, including ergometry, before exposure.² Fungal infections can be transmitted on bare footways, and bathing shoes are recommended.

Conversely, saunas are claimed to have beneficial psychological effects. Indeed, "if it is true that pleasant states of mind can be obtained only by first activating the body, then going to sauna is a rather pleasant way of accomplishing this." A significantly greater reduction in anxiety has been shown in healthy young men taking a Finnish sauna bath as against taking a shower. No difference was shown

in the same volunteers in performing psychomotor tasks after saunas and showers respectively.³ "Sex saunas" raise other issues of a more traditional character. Perhaps curiously, a considerable increase in sexual arousal has been shown after cold showers compared with a decrease (albeit insignificant) in arousal after a sauna.³ No reference can be found to specific hazards to staff, which presumably relate, as in other occupations, to sampling their own product.

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On a recent edition of the BBC television programme "Tomorrow's World" an electronic device was demonstrated that may be of possible value to stutterers. What recent developments have there been?

It has long been known that stammerers speak fluently when they cannot hear their own voices. Various masking techniques have been devised, usually playing white noise (comprised of all audible frequencies) through headphones. The device in question is fitted with a throat microphone that picks up laryngeal movement and then plays a single tone, similar in frequency to the one the stammerer is about to produce, through earpieces. The masking tone thus mirrors the speaker's pitch changes rendering his voice inaudible. This technique avoids problems of monotony plus the discomfort of prolonged white noise. Although still in the experimental stage the machine has generated a great deal of interest, including offers of industrial help, and it is likely that it will become commercially available in the next few years. Meanwhile many speech clinics use white-noise masking among other remedial methods.