mean arterial pressure alone could account for the large decrease in limb flow, and it is concluded that the change is due to a relative vasoconstriction in the peripheral circulation.

The rapid onset of vasodilatation after induction of anaesthesia suggests that the primary alteration in calibre of the peripheral vessels is an expression of release of normal vasomotor tone, and is due to the action of the anaesthetic agent on the vasomotor centre. But the vasodilatation could also be due to a direct action of the anaesthetic agent on the walls of the blood vessels (Cushny, 1928). The large distribution of vasomotor nerves to the skin, and the great number of arterio-venous anastomoses in the foot and hand (Lewis, 1936) could both equally well account for the relatively greater flows which develop in these regions.

The development of relative vasoconstriction in the later stages of the major operations probably expresses a circulatory readjustment for maintenance of the blood pressure. It occurs despite the fact that the amount of anaesthetic used increases in proportion to the length of the operation.

There is a limit to the duration and degree of vasoconstriction that can occur during lengthy major operations. This limit has not been determined, and may indeed vary from patient to patient. But it can be inferred that intense and prolonged vasoconstriction is indicative of maximal compensation and presages overt clinical shock.

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A hundred years ago last month the first conference on international health problems was held in Paris, when the representatives of 12 nations met to discuss quarantines and lazarettos in the Mediterranean. After six months' work the first International Sanitary Convention was signed. The Republic of Venice had introduced quarantine in the fifteenth century, and every maritime country eventually followed suit; but the systems used by the various governments were different and often conflicting. Also the cholera epidemic of 1832 had shown that the quarantine systems were unable to prevent the spread of disease and badly needed re-examination; the result of this situation was the Paris conference. From that time international co-operation steadily increased, and in 1907 the first permanent health organization was founded in Paris—the Office International d'Hygiène Publique. Its duties were primarily to ensure the functioning of the quarantine conventions, to draw up new ones, and to act as a centre of information on pestilential diseases. Until it was absorbed by the World Health Organization in 1947 the Paris Office performed these tasks with ever-increasing efficiency. By the end of the 1914–18 war other health problems which required international action had arisen, such as the control of narcotic drugs, the international standardization of sera, vaccines, and so on; also there was the need for uniformity in the classification of the causes of death. The League of Nations Health Organization, the predecessor of W.H.O., was established in 1921. (A fuller account appeared in *The Times* of July 23.)

THE FAMILY DOCTOR AND DIABETIC COMA

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Diabetic coma most frequently develops outside hospital, therefore we feel it important to draw attention to some of the ways in which the patient and his family doctor can prevent the onset of coma.

Since 1922 there has been a fall in the incidence of diabetic coma (Joslin, 1946). The reasons for this decline are the introduction of insulin therapy, the use of sulphonamide and antibiotic drugs, the increasing number of diabetic clinics—the last resulting in better control of both patient and disease—and a greater understanding of the disease by patient and doctor.

The mortality of diabetic ketosis has fallen because diabetic precoma is treated more effectively, with the result that fewer cases progress to coma, and also because the management of diabetic coma is improving each year. The chief factors responsible are the larger doses of soluble insulin given in the early stages of the illness, the more liberal use of intravenous fluids—a form of therapy revolutionized by the introduction of the “drip” method of transfusion (Marriott and Kekwick, 1935)—a clearer understanding of the type of fluids which should be given, the realization of the importance of gastric lavage, and the control of infections by sulphonamides and antibiotics. In spite of all these advances in treatment diabetic coma still occurs and all too often results in death.

The figures given in Table I illustrate the difference in prognosis between precoma and coma. The cases have been divided into two periods, each of nine years. October, 1941, was taken as the mid-point of the 18 years because the Radcliffe Infirmary Diabetic Clinic was started in that month. In defining diabetic precoma and coma we have used the former expression when a patient is conscious but drowsy from diabetic ketosis, and the latter when he is unconscious from the same cause.

TABLE I.—Outcome of 111 Cases of Precoma and 74 Cases of Coma, 1932–50

Year		Alive	Dead	Survival
Coma	1932–1941 (Sept.)	2	34	6%
	1941 (Oct.)–1950	15	23	39%
Precoma	1932–1941 (Sept.)	22	5	81%
	1941 (Oct.)–1950	80	4	95%

The four deaths which occurred between 1941 and 1950 in the precoma group were due to (1) pulmonary embolus, (2) toxæmia after amputation of a gangrenous leg (an anaerobic infection), (3) acute dilatation of the stomach, and (4) a cerebral vascular accident. In each instance the patient had recovered from the precoma but died within a few days of admission.

Comparison of the two periods shows that the prognosis for both precoma and coma has improved since 1941, but that the death rate for diabetic coma is still very high.

Illustrative Cases

The following four cases illustrate some of the common faults in dealing with ill diabetic patients.

Case 1.—A 36-year-old woman in whom diabetes had been diagnosed six years ago. At the time of her illness she was 18 weeks pregnant. Three days before admission she developed acute pyelonephritis, and on the following day was vomiting. She took insulin on the morning of the day before admission, but not afterwards, and by the evening was drowsy. At 2 a.m. next day her doctor gave her a sedative because she was restless; by 7 a.m. she was unconscious. She arrived in hospital four hours later.

Comment.—The doctor was called in too late, the insulin dosage was reduced instead of being increased, and there was great delay in sending the patient to hospital.

Case 2.—A 63-year-old woman known to have had diabetes for years. Five days before admission she developed cough, headache, and vomiting. She had had no insulin until admitted to a cottage hospital on the fourth day of the illness: then she was given 20 units of soluble insulin, 20 units of zinc-protamine insulin, and $\frac{1}{2}$ oz. (14 g.) of glucose in water to drink. She received a further 20 units of insulin on the following morning; she did not reach the Radcliffe Infirmary until 12.30 p.m.

Comment.—The patient was given an inadequate dose of insulin and would have been better sent to the Radcliffe Infirmary direct instead of being admitted to the local hospital.

Case 3.—A man aged 69 not known to have diabetes. He was seen by his doctor five days before admission to hospital, when he gave a history of one month's loss of weight, lassitude, and thirst. Three days later the patient was drowsy and dehydrated, and his urine contained a large amount of sugar. He was admitted to hospital two days later, at 4.45 p.m.

Comment.—The delay in diagnosis and initiation of treatment proved fatal to the patient.

Case 4.—A girl aged 19. Diabetes had been diagnosed in 1940, four years before her death. Eighteen months before death she gave up her diabetic diet, and twelve months later stopped taking insulin. The symptoms and signs of uncontrolled diabetes returned. On the day before coming into hospital she became feverish and began to vomit. That evening she was warned by her doctor to "take care" because she was a diabetic. No insulin was given. Next morning she was admitted to hospital in deep coma with signs of peripheral circulatory failure.

Comment.—Insulin should have been given immediately and the patient sent to hospital that night.

Education of the Patient

The amount which a patient is able to learn varies according to his individual capacity, but almost every diabetic is capable of learning a few fundamental facts—a matter of great importance, since the education received in the management of his diabetes should act as his first line of defence against the onset of diabetic ketosis. The patient should know amongst other things that: (1) one of the essentials in treatment is to take enough insulin to keep glycosuria and ketonuria under control; (2) infections make diabetes worse; (3) urine testing is more than ever important during illness; (4) if a full diet is not acceptable, at least the carbohydrate of the diet should be eaten; and (5) if vomiting is persistent half the normal dose of insulin should be taken as a "first-aid" measure and a doctor summoned immediately.

At the Radcliffe Infirmary Diabetic Clinic patients are given some instruction at their first visit and also receive

a copy of a pamphlet in which are set out clearly and simply the main facts about diabetes. This dual method of instruction is used because many patients are unable to recall the spoken word by the time they reach home, because another member of the family may, after reading the pamphlet, be able to help the patient, and because in a busy out-patient department there is not time to give each patient a comprehensive dissertation on diabetes. At subsequent visits the patients are encouraged to ask questions, and any part of the book which has not been understood is explained. There is a section describing what to do if ill, and another section dealing very simply with the symptoms and signs of diabetic coma; the patients are urged at the first sign of trouble to call in a doctor at once. Patients with a low intelligence are made to understand at least that if they are unwell a doctor should be consulted straight away.

Bruch (1949) has shown that the education of the diabetic child and his parents is also of great importance, as often the doctor can improve the child's diabetes by helping the parents to adopt a more reasonable attitude towards the child's disease.

The Family Doctor's Part

The doctor's part in dealing with ill diabetic patients is described in textbooks of medicine and medical treatment, and recently in two informative articles by Oakley (1949). Analysis of the 23 fatal cases of coma occurring at the Radcliffe Infirmary between 1941 and 1950 has served to emphasize the following facts.

Infection was the precipitating factor in 19 instances. Lowered carbohydrate tolerance occurs in infection, and unless treatment is adequate ketosis develops. This may lead to or be accompanied by anorexia or vomiting, which will increase the state of dehydration already induced by the fever and polyuria. If this train of events is not checked precoma will develop, followed in its turn, after a period of days or even hours, by coma. If the doctor is unable to check the development of ketosis he should immediately send the patient to the nearest hospital capable of dealing with such an emergency.

Delay.—Joslin (1950) quotes Root as saying that the lowness of the death rate among those of his patients who were admitted in diabetic coma was to a large extent because the family doctor had given insulin before the patient was placed in the ambulance. In the 23 present cases only four received insulin before coming into hospital (see Table II). In at least eight instances, from a study of the doctor's letter and the time of arrival of the patient at the hospital, it seemed probable that unnecessary delay had occurred. The usual causes of delay are wrong assessment of the urgency of the case, delay in getting ambulances in country districts, and the distances patients have to travel. The last point is brought out in Table II.

TABLE II.—*Distance Travelled by 23 Fatal Cases of Diabetic Coma (1941–50), and the Number Given Insulin Before Being Sent to Hospital*

Distance Travelled	No. of Patients	No. Given Insulin Before Starting
Patients in Oxford City ..	7	2
6–10 miles	4	1
11–15 "	4	0
16–20 "	6	1
Over 21 "	2	0

It is difficult to estimate how long a patient has been comatose before admission. In Table III the duration of the coma in hospital in the 23 fatal cases is shown.

TABLE III.—Time in Hospital of 23 Cases of Fatal Diabetic Coma

Time in Hours	No. of Patients
0-1	2
2-12	7
13-24	6
25 or more	7
Unknown	1

We feel that some of the patients would have arrived in better condition if an adequate dose of insulin had been given by the doctor outside.

Insulin.—It is suggested that the doctor sending to hospital a patient with either precoma or coma should adopt the following scheme of emergency treatment.

TABLE IV.—Showing the Basic Insulin Dosage as Judged by the Reaction of the Urine to Benedict's Test

Colour of Benedict's Test	Dosage of Soluble Insulin
Red	40 units
Orange	32 "
Yellow	20 "

(1) Add 20 units of soluble insulin if the ferric chloride test is positive for diacetic acid (N.B. salicylates give a false positive reaction); (2) Add a further 20 units if the patient is drowsy, or 40 units if he is unconscious, from diabetic ketosis; (3) If the patient is unconscious, and circumstances permit, a stomach tube should be passed and the gastric contents aspirated before sending the patient to hospital (Micks, 1948); (4) Carbohydrate should not be given as a "cover" to the insulin.

This scheme is not a substitute for the full treatment of diabetic coma, but is the "first aid" to be applied before the patient reaches hospital, when every hour counts.

A doctor who may have to treat diabetic patients should always have available one bottle of soluble insulin, strength 80 units per 1 ml. Insulin, if stored in a cool place, will maintain its potency for two years.

Summary and Conclusion

The outcome of 111 cases of diabetic precoma and 74 cases of coma admitted to the Radcliffe Infirmary between 1932 and 1950 is presented.

Suggestions are made for improving the prognosis for diabetic patients with progressive ketosis, and stress is laid on the important part that the family doctor can play in improving the outlook for these patients. The prognosis for diabetic precoma is good; the prognosis for diabetic coma is bad. It should be the aim of every doctor dealing with diabetic patients to correct any tendency to progressive ketosis and, if unable to do so, to admit the patient to hospital without delay. Every opportunity should be taken to teach diabetic patients about their disease and how to look after themselves when ill.

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In December, 1950, a nightly average of 2,239 people slept in reception centres (*Ministry of Labour Gazette*, July, 1951), many of them young men presumably fit and capable of earning their living. During the year the National Assistance Board used for the first time its power to prosecute anyone who had to be helped by the Board because of persistent refusals to work; four men were sent to prison in Birmingham. The effort to help men to settle in jobs has not diminished, and no one was prosecuted without being warned and given another chance.

Medical Memoranda

Ascaris Impacted in Common Bile Duct

Routine stool examination in the hospital laboratory during the last 30 years has shown that ascaris infection among Syrians is almost universal. *Ascarides* have been found in a liver abscess, in the appendix, and as the primary cause of intestinal obstruction; they have often been suspected of causing cholecystitis. This is the first proved case recorded here of their causing obstruction of the common duct.

Case Report

A woman aged 45 was admitted to hospital complaining of severe epigastric pain, extending towards the left, of eight days' duration. Nausea had been persistent, and for this she had obtained relief by making herself vomit. She had had similar attacks, though not so severe, during the last 30 years.

Physical findings were as follows: no jaundice; acute tenderness over the gall-bladder; temperature 101.3° F. (38.5° C.); pulse 128; B.P. 100/75. Urine: albumin +, chlorides 3 g./l.; W.B.C., 17,500. During the next three days under penicillin and sulphathiazole the temperature settled and the urine cleared. Cholecystography revealed a non-functioning gall-bladder. The abdomen was opened on the fourth day with a diagnosis of subsiding cholecystitis.

Operation.—Under rectal ether, spinal procaine, procaine infiltration, and endotracheal ether and oxygen, an 8-in. (20-cm.) right paramedian incision was made. The gall-bladder was adherent to the duodenum and distended but contained no stones, and the cystic duct up to its junction was perfectly normal. The common duct presented as a thick white cord of the calibre of small intestine arched tensely forwards and outward. It appeared to be the site of some long-standing fibrous change. Suspecting malignancy, the duodenum was mobilized and turned inwards, but no thickening of the ampulla was felt on palpation. Finally it was decided to open the duct. The lumen was found to be entirely filled by a firm white, twisted, cable-like mass consisting of 21 large ascarides. These were removed by forceps and suction. Both hepatic ducts were grossly dilated and one or two worms were removed from the right hepatic duct. The entry into the duodenum was free and dilated rather than constricted. The actual wall of the duct was not thickened, and the appearance of general fibrosis had been produced entirely by the compact mass of worms. Drainage was established by a Y tube snugly stitched round the duct opening, and a second tube was brought out alongside, draining the peritoneal cavity in the region of the duct incision. The liver itself presented many scars on the surface and one fibrous nodule, which was removed for biopsy. The omentum was brought over to wall off the drainage area.

The patient made a smooth recovery. On the tenth day a cholangiography with lipiodol through the intrabiliary tube showed dilated hepatic and common ducts but free passage into the duodenum. Three more worms were evacuated through the common-duct tube. This was removed on the 13th day, and the peritoneal tube on the 17th. There has been no further trouble. Stools contained ascaris ova on the 12th day, but were clear by the 16th after the administration of santonin. Biopsy of the liver showed considerable periportal fibrosis with bile-duct hyperplasia and round-cell infiltration.

Ascarides should always be regarded as a surgical hazard and be cleared out if possible before any laparotomy. The hospital records show at least one case in which ascarides were found free in the peritoneal cavity