could count fingers in the central area. The left fundus now showed a circular area of "oedema"—coagulation necrosis of the retina—covering an area some five disk diameters in width centred on the disk. This involved the macula, but no cherry-red spot was present. This was due to there being a small cilio-retinal artery supplying the macular area—the presence of which accounts for the survival of central vision from the beginning. In the next week the "oedema" of the retina gradually subsided, and as it did so the visual field gradually opened up; at the end of this time the patient could read the small print in a newspaper with her left eye. Thirty days after operation the left fundus showed generalized pallor of the optic disk and attenuation of the retinal arteries, the background of the fundus and the macular area appearing normal, the picture being typical of the late result of occlusion of the central retinal artery. At this time a full ocular examination was made. The right eye was found to be emmetropic, with vision 6/5 and a full peripheral field of vision. The left eye was also emmetropic, with vision 6/6. The left visual field was irregularly contracted, its periphery to a 1/330 white object varying between 30 and 50 degrees from the fixation point, while within this periphery there existed small blind islands.

The patient's recovery from the radical mastectomy operation was uneventful and serial blood pressures were not taken after her return to bed, as this was not thought to be necessary, but there was no suggestion of a falling off in blood pressure nor of circulatory insufficiency. On direct questioning she stated that she had had no previous eye trouble, and had never worn glasses, and there was no history of attacks of misty vision such as might suggest antecedent episodes of retinal angiospasm.

Discussion

It is clear from the clinical evolution of the case that the cause of the visual loss was retinal ischaemia serious enough to account for a severe immediate depression of function, but not prolonged enough to cause complete necrosis of those elements of the retina supplied by its central artery. The possible causes of such interruption of circulation are: thrombosis or embolism of the artery (including air embolism); pressure on the eye from without, increasing the intraocular pressure to a point where arterial circulation is mechanically impeded; arterial hypotension so low as not to maintain an adequate blood supply; and arterial spasm.

Thrombosis and embolism can be excluded on the clinical appearance of a re-established normal blood flow within 24 hours of the operation. Air embolism is admittedly a possibility, but there was nothing during the course of the operation to suggest its occurrence, and without postulating a patent interventricular septum it is unlikely that such would pass the sieve of the pulmonary capillaries.

Some four cases of occlusion of the central retinal artery after general anaesthesia have been recorded by Givner and Jaffe (1950), who attributed this complication to pressure on the globe when a patient is in a state of shock and suffering from loss of blood. With a lowered retinal arterial pressure any slight increase in the intraocular tension, the result of the pressure of a facepiece perhaps, might suffice to cut off the vascular circulation more or less completely. In the present case this explanation seems inadequate in view of the apparent survival of the area of retina, including the macula, supplied by the cilio-retinal artery, whose circulation should have been impeded by any external pressure on the globe equally with that of the central retinal artery. For the same reason we do not feel that pure insufficiency of circulation through arterial hypotension is a causative factor; if it were, such a complication should be the almost invariable concomitant of lowering the blood pressure to 70 mm. Hg for an hour or more. Furthermore, it has been calculated by Fritz (1950) that the retinal blood pressure corresponding to the amaurotic threshold is, at a minimum, 7, and at a maximum, 25 mm. Hg. The retinal ganglion and

bipolar cells will almost certainly remain viable, although perhaps temporarily functionless, at the minimum pressure, which will be considerably exceeded if the brachial blood pressure is at 50 mm. Hg.

This leaves, by exclusion, the possibility of spasm of the artery, which we believe to be the causative factor in the case. Spasm of the central retinal artery has often been described; it results usually in temporary obscuration of vision lasting a few seconds to a few minutes; if prolonged enough it may result in blindness. It is on the whole commoner in hypertensive subjects, but the mechanism of its causation is often completely obscure. In the present case what, if any, its relationship to the use of hexamethonium iodide may be it is impossible to say; it may be of significance, however, that this particular patient was to some extent resistant to the hypotensive action of the drug in that her arterial pressure did not fall below 70 mm. Hg; it is conceivable that this resistance is due to peripheral vascular contraction of unknown mechanism in which the retinal artery played so generous a part that its dependent structure, the retina, suffered severely.

Unfortunately we have no definite proof that the blood pressure did not fall to a low level after the patient's return to bed, but she had a competent special nurse and there was no suggestion of further hypotension in the one and a half hours before she recovered consciousness.

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Medical Memoranda

Exogenous Ochronosis

The clinical picture of ochronosis consists of a dark generalized pigmentation of skin and cartilage which is complicated by a marked tendency to osteoarthritis; it may or may not be associated with the excretion of homogentisic acid in the urine. The excretion of homogentisic acid is known as alkaptonuria and, according to Oppenheimer and Kline (1922), was first detected in 1858 by Bodeker. Garrod (1923) states that Virchow (1866) was the first to use the term "ochronosis" to describe the characteristic pigmentation; he used this word because histological sections of the affected tissues were stained yellow or yellowish brown.

It was Albrecht (1902), however, who first suggested that this pigmentation might be associated with alkaptonuria. It was later recognized that osteoarthritis formed the third link to make up the triad of signs in this inborn error of metabolism. It soon became firmly established that the same clinical picture, excepting the excretion of homogentisic acid, could be produced by the application of carbolic acid compresses to ulcerated areas—that is, an exogenous cause (Pick, 1906). A third group, of extreme rarity, is of endogenous origin, and consists of cases associated with the excretion of both alkapton and melanin (Duncan, 1942).

Duncan has stated that up to 1942 just under 200 cases of alkaptonuria had been described in the world literature; this contrasts with only 60 cases of ochronosis, and this discrepancy is due to many alkaptonurics not developing any pigmentation. With regard to the ochronotic cases, by far the greater number were of endogenous origin. According to Beddard and Plumtre (1912), out of 23 cases of ochronosis only 9 were of exogenous origin—that is, due to the application of carbolic acid compresses. Before 1920 it was fashionable to apply these dressings to ulcers, but now this technique has fallen into disuse. The following case is recorded because it was of exogenous origin, which is almost unknown in clinical practice to-day.

CASE REPORT

On February 2, 1949, a woman aged 58 attended the surgical out-patient department and complained of ulceration of the left leg. She had had puerperal fever 18 years previously, and this had been complicated by a "white leg" which had subsequently ulcerated. Over this period of time the ulcers had been treated with elastic adhesive bandages and various lotions. She had used compresses of dilute carbolic acid over long intervals—in spite of warnings from her own doctor. She had noticed that for about three years the whites of her eyes were gradually becoming pigmented, and this was accompanied by darkening of the skin, especially of the face and hands. She also admitted that this pigmentation would wax and wane. More recently the fingers had become painful on movement and the middle finger of the right hand often locked. She also complained of marked nocturnal frequency with intermittent dysuria, and the urine was dark on occasions. There was no headache, but she slept badly and the skin itched intolerably. There was nothing else significant in the history. Both her parents had lived to a ripe old age and none of her relations had had pigmentation.

On examination there was a bluish-black pigmentation over most of the body, but especially of the face and hands, and the axillae were also heavily involved. The cartilages of the ears were deeply pigmented, and on the conjunctivae there were bilateral patches of the same dark pigmentation. Pressure on the skin did not cause it to disappear. The knees were swollen and crepitus was easily elicited; in fact, most of the joints were the seat of advanced osteoarthritis. There was also some dorsal kyphosis. The heart was enlarged and an apical systolic murmur was present; the B.P. was 160/75. The abdomen was somewhat rigid; no gross changes were found in the lungs or nervous system. The urine appeared normal in colour and its S.G. was 1021; there was, however, albuminuria, and the guaiacum test for blood was positive. Neither Fehling's nor Benedict's solution was reduced.

It was obvious from the clinical picture that this was a case of ochronosis of exogenous origin. As the patient was pyrexial and her general condition was poor she was admitted to hospital. A blood count showed some hypochromic anaemia associated with a leucocytosis. A catheter specimen of urine contained many red cells and pus cells; the blood urea was 190 mg. per 100 ml. The Wassermann reaction was negative. She rapidly deteriorated in spite of symptomatic treatment and died in uraemic coma four days after admission.

The post-mortem examination was revealing. Besides the external pigmentation nearly all the cartilages were of the same dark colour, especially those of the knees, the sternum, and the intervertebral disks. With regard to the joints, it appeared that where the pigmentation was deepest the osteoarthritis was more advanced. The aorta, which was atheromatous, was also pigmented. It was rather surprising to find the brain stained a brownish colour. The gall-bladder contained one cholesterol stone and the spleen was slightly enlarged. The kidneys, which were pale and small, were secondarily contracted. The bladder was full of pus. There were no other significant findings.

COMMENT

The pigmentation in this case did not differ fundamentally from that described in previous ones. The patient was quite emphatic that its depth varied from time to time, but she did not correlate this variation with the use of carbolic acid. There is little doubt that, had she stopped using carbolic acid and had lived long enough, the pigmentation would have diminished considerably in intensity. The osteoarthritis is undoubtedly caused by the deposition of pigment in the cartilages. The differential diagnosis is not difficult. The colour picture is probably most closely simulated by argyria, but in the latter condition there is a history of using

a silver preparation and the pigmentation does not wax and wane. Addison's disease, haemochromatosis, acanthosis nigricans, and arsenical pigmentation may offer slight difficulties. Haemochromatosis may simulate it not only because of the pigmentation but because of the reducing substance—that is, sugar—found in the urine.

I should like to thank Dr. W. J. S. Reid for his advice and permission to publish.

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Traumatic Rupture of a Cyst of the Mesocolon

The case here recorded is of particular interest as it illustrates both traumatic rupture of loculi and probably spontaneous rupture of a loculus on a previous occasion. Only one case could be found in the literature.

CASE REPORT

Driver A first noticed increased abdominal girth in August, 1950; this was progressive, making it necessary for him to obtain a larger tunic. A few weeks afterwards he developed swelling of the left leg, which was diagnosed as thrombophlebitis and treated with rest in bed. The swelling did not subside completely, and he had two further attacks before the end of the year.

Apart from epigastric distension after meals, he had no further symptoms until April, 1951, when he was on leave. He woke up one day at 1 a.m. with severe continuous pain in the epigastrium and in both shoulders. There was no nausea or vomiting. He was admitted to a hospital for observation. The epigastric pain subsided, and had disappeared after three days, but the pain in both shoulders persisted for a further week.

After his discharge from hospital he had severe anorexia, and the epigastric distension increased. His bowels were regular, but he passed a great deal of flatus.

In July, 1951, he was taking his horses to a water-trough, when one of the animals kicked him in the epigastrium. He was admitted to the Cambridge Hospital, complaining of severe continuous pain all over the abdomen and in both shoulders. He was not severely shocked; B.P. 120/90, pulse 100. A hoof-mark could be seen in the epigastrium. There was tenderness and rigidity all over the abdomen, and dullness in the flanks. A fluid thrill was detected and shifting dullness was demonstrated. There were no abnormal physical signs in the chest, and the urine was normal.

Laparotomy was performed immediately through a mid-line epigastric incision. There was a considerable quantity of serous fluid in the peritoneal cavity, and a huge multi-locular cyst of the mesocolon was seen; two of the loculi had ruptured, but the remainder of the cyst was intact and was removed without difficulty. The cyst was approximately the size of a full-term foetus.

His convalescence was uneventful, and his legs are now of equal size.

We are indebted to the Director of Surgery to the Army for permission to publish this case.

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