A. E. A. RIDGWAY: SOLAR RETINOPATHY

FIG. 1.—Case 3. Photograph taken on 24 May. Kodachrome II. Kowa fundus camera.

FIG. 2.—Case 5. Photograph taken on 3 June. Kodachrome II. Nordens fundus camera.

P. D. GULATI *ET AL.*:
ENGLEMAN'S DISEASE

FIG. 1

FIG. 2

FIG. 3

FIG. 1.—Radiograph showing increased density of the diaphysis of long bones of the lower extremity. Epiphysial and metaphysial zones show normal density.

FIG. 2.—Radiograph showing increased density of the diaphysial zones of long bones of the upper extremity.

FIG. 3.—Radiograph of skull showing osteosclerosis of base of skull as well as bones of calvarium.

myeloma, lymphoma, and occasionally in other malignant disease. It is of interest that in the case of Domz and Chapman (1961) resection of the tumour led to a fall in the level of circulating cryoglobulins. The level, however, remained high and the cryoglobulinaemia could have been due to the coexisting myelofibrosis. Waldenström (1961) found that in 276 unselected cases of macroglobulinaemia 10 suffered from malignant disease. He concluded that the finding of macroglobulins in any patient should warrant further investigation to exclude neoplasm. Bohrod (1957) thought that cryoglobulinaemia, which can occur together with plasmacytosis and urticaria, represented an immunity reaction between tumour and host. In our cases cryoglobulins were looked for in all patients except Case 1, and in her case the electrophoretic strip was normal. They were found to be present only in Case 6, who suffered from Hodgkin's disease.

On the basis of the six cases described above it is suggested that malignant disease may give rise to digital ischaemia, and this may be the presenting or the only symptom. How this occurs is a matter for speculation. The incidence of "Raynaud's disease" in the older age groups of the general population is not known. It is possible that the occurrence of attacks of blue and numb fingers in association with malignant disease was coincidental in cases 3 and 5. The presentation in the remaining four cases, in which necrosis was an early feature and the onset more or less sudden, is unusual enough to suggest a more than fortuitous relationship. All of these patients with the exception of the case of Hodgkin's disease died within 18 months of the onset of the ischaemic changes. In some the malignancy remained undiagnosed until the patient's death and in others was advanced when eventually discovered. Patients who present with digital ischaemia, often loosely termed "Raynaud's disease," in whom the accepted causes have been excluded, deserve the fullest investigation in an attempt to discover an occult neoplasm.

The variety of neoplasms described in our series would suggest that the search must be wide and may even necessitate laparotomy in certain cases. If thorough investigation proves negative a careful follow-up should be undertaken. In two further cases not described in this series, the patients presented with digital ischaemia and remained undiagnosed despite the fullest investigation. They were well at nine months and three years respectively from the onset of their symptoms. In such cases a careful follow-up should always be undertaken. If the possibility of occult neoplasm as a cause of digital ischaemia is

kept in mind diagnosis may be hastened and radical treatment made possible.

Summary

Six cases are described suggesting an association between digital ischaemia and malignant disease. They were a group of middle-aged women who either presented with atypical Raynaud's phenomenon or in whom this was a prominent feature. The symptoms were of sudden onset, bilateral in five cases, and in four rapidly progressed to gangrene. None of the commonly accepted causes of digital ischaemia was present. On investigation three patients were found to have a primary carcinoma of the maxillary antrum, kidney, and ovary, respectively; one patient had primary carcinomata of both the colon and corpus uteri, and in another the origin was not established but was probably the ovary or pancreas; the remaining patient was suffering from Hodgkin's disease. All of the patients with the exception of the last died within 18 months of the onset of the ischaemic changes. In some the malignancy remained undiagnosed until the patient's death and in others was advanced when eventually discovered. It is suggested that malignant disease may give rise to digital ischaemia and that it may be the presenting or only symptom. Patients who suddenly develop "Raynaud's disease" in whom the accepted causes have been excluded deserve the fullest investigation in an attempt to discover an occult neoplasm.

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REFERENCES

- Bennett, T. I., and Poulton, E. P. (1928). *Amer. J. med. Sci.*, 176, 654.
 Bohrod, M. G. (1957). *J. Amer. med. Ass.*, 164, 18.
 Domz, C. A., and Chapman, C. G. (1961). *Calif. Med.*, 95, 391.
 Fagge, C. H., and Pye-Smith, P. H. (1891). *Text Book of the Principles and Practice of Medicine*, 3rd ed., vol. 2, p. 118. London.
 Hamilton, W. F. (1920). *Canad. med. Ass. J.*, 10, 670.
 Gifford, R. W., jun. (1963). *Circulation*, 27, 970.
 — and Pickering, G. W. (1933-4). *Clin. Sci.*, 1, 327.
 Mackay, I. R., Eriksen, N., Motulsky, A. G., and Volwiler, W. (1956). *Amer. J. Med.*, 20, 564.
 Nielsen, B. L., and Petri, C. (1963). *Nord. Med.*, 69, 237.
 O'Connor, B. (1884). *Brit. med. J.*, 1, 460.
 Pasteur and Price-Jones (1901). *Trans. clin. Soc. Lond.*, 34, 160.
 Trouseau, A. (1865). *Clinique Médicale de l'Hôtel-Dieu de Paris*, 2nd ed. Paris.
 Waldenström, J. (1961). *Acta med. scand.*, Suppl. No. 367, p. 110.

Solar Retinopathy

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[WITH SPECIAL PLATE]

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Five cases of solar retinopathy presenting in Oxford within three months of the partial eclipse of 20 May 1966 are described.

Pathogenesis

Of the sum of solar radiation reaching the surface of the earth, only the visible and short infrared wavelengths reach the retina, the ultraviolet and longer infrared rays being absorbed in the cornea and lens (Verhoeff *et al.*, 1916; Irvine, 1945). The 60 to 70% of solar energy reaching the retina is absorbed by its pigment epithelium with the liberation of heat. With a meiosis of 2 mm., a 30-second exposure leads to the produc-

tion of heat at a rate of 70 calories/sq. cm./min., which is more than enough to produce a severe retinopathy experimentally (Verhoeff *et al.*, 1916; Ludvig and McCarthy, 1938; Eccles and Flynn, 1944).

Exposure of pigmented rabbit retinae to xenon arc photo-coagulator light has been shown to produce very similar lesions to human solar retinopathy, but similar exposures of albino rabbit retinae were tolerated without damage, and ten times the dose was needed to produce equivalent burns (Geeraets *et al.*, 1962; Geeraets and Ridgeway, 1963).

Further experimental work with xenon and carbon arc photo-coagulators, the emissions of which are close to that of sunlight,

has shown that the earliest damage is pyknosis of pigment epithelium cells with loss of fine structure in rods and cones, and local oedema, which may cause bulging of the retina into the vitreous cavity with resolution after a few days (Ham *et al.*, 1958).

The other factors influencing the severity of solar retinopathy are the intensity of the incident light—never less than 60% of full sunlight in the eclipse of 20 May 1966—and the duration of exposure.

Reflex meiosis of the order of 1.6 to 2 mm. (Verhoeff *et al.*, 1916) and refractive error tend to protect against damage. It has been shown that uncorrected myopes, who do not focus the sun's image on the retina, are relatively protected by reason of the lower density of illumination of the exposed areas (Das *et al.*, 1956). Physiological movements of fixation result in the exposure of a crescentic arc of retina on sun-gazing with a central zone always exposed and a peripheral zone intermittently exposed. Lastly, constancy of exposure is of importance in that repeated short exposures at short intervals do not summate in effect because of the good thermal conduction away from the macular area by its excellent blood supply.

Case 1

A 21-year-old labourer looked at the eclipse with both eyes without any protection for "about five minutes." He presented on 22 May 1966 complaining of dazzling when looking into the distance.

On examination the right visual acuity (R.V.A.) was 6/12 unaided, and the left visual acuity (L.V.A.) 6/9 unaided. On refraction both eyes were emmetropic, he had no strabismus, and no scotoma was demonstrable in either visual field. There was slight prominence of both maculae, and he was offered no treatment.

Progress.—His right macula became more prominent over the next three days and then subsided, and by 3 June was thought to be within normal limits. His symptoms improved and on 17 June his visual acuity was 6/4 unaided in both eyes, and his only complaint was that he had to alter fixation to read the middle two letters of the bottom row of the test chart with each eye. It was impossible to demonstrate a scotoma objectively in either eye, but ophthalmoscopically minute yellow spots were visible at both maculae.

Case 2

A 19-year-old male undergraduate reading physics presented on 24 May, having watched the eclipse through a plain glass window for a few seconds, and with sun-glasses for half a minute. He complained of a black spot in his right central field.

On examination the R.V.A. was 6/4 and the L.V.A. 6/9 (parts) uncorrected. On refraction the right eye was emmetropic and the left 0.25 dioptre sphere myopic, with which correction he achieved 6/4. He had no strabismus. Projection campimetry showed a small scotoma across and below fixation to a 3/2000 mm. red object, using a green fixation object and coloured goggles, green before the left and red before the right eye (Gambis *Perimeter Handbook*, p. 10, 1966). He had moderate macular oedema in the right fundus. He was treated with prednisolone 5 mg. three times a day.

Progress.—Over the next two weeks the macular oedema cleared and the prednisolone was tailed off and stopped. On 15 June his black spot was smaller and less dense, he had no visible macular abnormality, and on campimetry the scotoma was minute.

Case 3

A manual worker aged 36 presented on 22 May after watching the eclipse "for a few minutes" with his right eye through the gaps between his fingers. He complained of blurring of central vision.

On examination the R.V.A. was 6/6 (parts) and the L.V.A. 6/4. On refraction the left eye was emmetropic and the right eye had one dioptre sphere of hypermetropia. With this correction his R.V.A. was 6/5. He had no strabismus.

Projection campimetry showed a circular blurred area to 10 degrees from fixation and he had gross perimacular oedema. He was started on prednisolone 10 mg. three times a day by mouth.

Progress.—On 24 May the perimacular oedema was less but there was a swelling at the macula (Special Plate, Fig. 1). On 27 May the oedema had receded further and there was a definite macular cyst. The prednisolone was reduced to 10 mg. twice daily. On 1 June his blurred patch was much smaller but considerably denser and a scotoma of 1 degree diameter was demonstrated at fixation to a 3/2000 mm. red object. The R.V.A. had dropped to 6/9 corrected. The prednisolone was tailed off and stopped over the next seven days. During the next two weeks the macular cyst became smaller, as did his scotoma, and on 17 June he managed R.V.A. 6/5 corrected, scanning the smaller letters.

Case 4

A schoolboy aged 13 presented on 11 August complaining of blurring of vision in the right eye after viewing the eclipse. He had gazed at the sun through two sun-glasses for two to three minutes between classes. Three hours later he noticed green flashes in the right eye and that evening his vision in this eye became very blurred. It had now recovered somewhat.

On examination the R.V.A. was 6/6 (parts) and the L.V.A. 6/4. Both eyes were emmetropic and there was no strabismus. Campimetry showed a small scotoma between one and two degrees, nasal to (binocular) fixation and extending five degrees up from the horizontal meridian. He had a flat lesion below a moderate-sized paramacular hole in the right fundus.

Progress.—In view of the lateness of presentation no treatment was offered, and to allay parental anxiety follow-up has been deferred for six months.

Case 5

An accounting-machine operator aged 25 presented on 3 June. She had been sunbathing without sun-glasses five days previously, and had been "squinting at passers-by against the sun" through her left eye. On awaking the next morning she had noticed a shimmering patch in her left central field, which had become smaller and was now opaque.

For 10 years she had had migraine two to four times a year. This consisted of flashing lights in the right eye spreading to the left, followed by a generalized headache not relieved by aspirin.

On examination the R.V.A. was 6/5 and the L.V.A. 6/36+2. On refraction both eyes were emmetropic and she had no strabismus. Campimetry showed a crescentic scotoma in the upper and lateral macular region and she had much macular and perimacular oedema maximal below a small macular hole (Special Plate, Fig. 2). She was treated with prednisolone 5 mg. four times a day by mouth.

Progress.—On 11 June her field defect had contracted to a 2 by 1 degree scotoma across and above fixation on projection campimetry to a 3/2000 mm. red object, and her L.V.A. had improved to 6/18 unaided. The macular oedema had disappeared and the macular hole was less prominent. Her corticosteroids were tailed off over the next week. On 17 June her L.V.A. had improved to 6/9 part unaided and she was no longer conscious of the field defect, which was unchanged. There was some fine pigmentation around the macular hole.

Discussion

The first four cases demonstrate the damage which may be caused by short exposure of the retina to the sun. In the past, damage caused by watching eclipses has been related to similar changes seen in people who have looked at the sun for other reasons.

Das *et al.* (1956) reported cases among Punjabis who look at the sun for religious reasons. Pittar (1943) reported one case in an anti-aircraft gunner firing at an enemy plane flying out of the sun, and fighter pilots performing the same manoeuvre have suffered similar consequences. Irvine (1945) has recalled the case of a seaman "steering into the sun's eye." Bates (1920) advocated prolonged sun-gazing as the treatment of myopia, with disastrous results.

However, solar retinopathy takes on a much more sinister aspect with the presentation of the fifth case. Here a healthy young woman was sunbathing, and it seems probable that as a consequence of this innocent act she sustained a severe macular burn, leaving her with a permanent central field defect in one eye, an anatomical lesion, and possibly the predisposition to macular degeneration in this eye in later life (Corcelle, 1958).

The warnings made by Knudtzon (1948) need repeating, especially as they bear on the false sense of protection afforded by dark glasses, red glass filters, smoked glass, and overexposed negative films. The only safe way of observing the sun, the method used by astronomers, is to project the sun's image on to a screen. For the amateur a pin-hole and sheet of matt paper held in the shade behind it will suffice (Gilkes *et al.*, 1961).

In the case of accidental solar retinopathy it is difficult to suggest any protective measure other than greater public awareness of the dangers involved. Publicity before the eclipse of 20 May 1966 was responsible for much sun-gazing, and no safe protective methods were advocated. In effect, interest was aroused and the dangers were minimized.

Summary

Four cases of eclipse burn and one of solar retinopathy due to sunbathing are presented. The seriousness of an apparently

trivial action in terms of visual function is demonstrated and attention is drawn to the possibility of accidental macular burns.

More effective methods of prevention are advocated and it is urged that public awareness of the dangers be aroused.

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REFERENCES

- Bates, W. H. (1920). *The Cure of Imperfect Sight by Treatment Without Glasses*. London.
 Corcelle, L. (1958). *Arch. Ophthalm. (Paris)*, **18**, 555.
 Das, T., Nirankari, M. S., and Chaddah, M. R. (1956). *Amer. J. Ophthalm.*, **41**, 1048.
 Eccles, J. C., and Flynn, A. J. (1944). *Med. J. Aust.*, **1**, 339.
 Geeraets, W. J., Everett, W. G., and Guerry, D., III (1962). *Amer. J. Ophthalm.*, **54**, 393.
 — and Ridgeway, D. (1963). *Acta ophthalm. (Kbh.)*, Suppl. No. 76, p. 109.
 Gilkes, M. J., Roberts, D. St. C., Osmond, A. H., and Thorne Thorne, B. (1961). *Lancet*, **1**, 109.
 Ham, W. T., jun., *et al.* (1958). *Amer. J. Ophthalm.*, **46**, 700.
 Irvine, S. R. (1945). *Ibid.*, **28**, 1158.
 Knudtzon, K. (1948). *Acta Ophthalm. (Kbh.)*, **26**, 469.
 Ludvig, E., and McCarthy, E. F. (1938). *Arch. Ophthalm.*, **20**, 37.
 Pittar, C. A. (1943). *Brit. J. ophthalm.*, **27**, 36.
 Verhoeff, F. H., Bell, L., and Walker, C. B. (1916). *Proc. Amer. Acad. Arts Sci.*, **51**, No. 13.

Effect of Oral Contraceptives on Erythrocyte Sedimentation Rate in Healthy Young Women

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The erythrocyte sedimentation rate (E.S.R.) is a useful non-specific test for a variety of diseases accompanied by inflammation or breakdown of tissue. It is often used as a screening test for occult disease in the absence of physical signs, and it is also useful for assessing progress in chronic or relapsing disease.

A recent reply in the "Any Questions?" column of the *British Medical Journal* (25 March 1967) has implied that the E.S.R. in women taking oral contraceptives is normal. It has long been known that the E.S.R. is raised above the usually accepted limits of normal in pregnancy (Fåhræus, 1918; Bochner and Wassing, 1925; Dawson, 1960), and since the physiological effects of oral contraceptives are in many ways similar to those of pregnancy it was decided to investigate their effect on the E.S.R. in normal young women.

Methods

Women attending a routine family planning clinic were divided into two groups according to whether they used oral contraceptives¹ or an occlusive pessary. After a medical interview a 2.5-ml. sample of venous blood was obtained for the estimation of haemoglobin, packed cell volume, and E.S.R.

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¹ The oral contraceptives used in this study were: Ovulen (mestranol 0.1 mg. and ethynodiol diacetate 1 mg.), Lyndiol (mestranol 0.15 mg. and lynoestrenol 5 mg.), Lyndiol 2.5 (mestranol 0.075 mg. and lynoestrenol 2.5 mg.), and Gynovlar (ethinyloestradiol 0.05 mg. and norethisterone acetate 3 mg.).

All women included in the final study conformed to the following criteria: (1) they were below 40 years of age; (2) they had been using the same contraceptive method for at least six months previously; (3) they gave no history of previous serious or chronic illness, and had not suffered from overt infection during the previous four weeks; (4) they felt perfectly well on the day of attendance at the clinic; and (5) they had haemoglobin levels and packed cell volumes within normal limits (Hb 11.5–16.4 g./100 ml.; P.C.V. 36–47%).

The relation of the timing of the blood sample to the menstrual period was not determined, since this was a random variant in both groups, and in any case the fluctuation of E.S.R. with menstruation is insignificant (Greisheimer, 1927; Wintrobe, 1961).

Seventy-eight women were included in the study, of whom 42 had been taking oral contraceptives and 36 had been using an occlusive pessary.

The blood samples were transported in polystyrene bottles containing Sequestrene anticoagulant, and the E.S.R. was determined within six hours by the Westergren method, a one-fourth part of 3.8% sodium citrate solution (Dacie and Lewis, 1963) being used. Various authors have given results varying from 7 to 25 mm. in the first hour for the upper limit of the normal E.S.R. in women (Westergren, 1926; Dawson, 1960; Dacie and Lewis, 1963; Hilder and Gunz, 1964; Böttiger and Svedberg, 1967). The normal value used in this laboratory for young women is up to 7 mm. in the first hour (Dacie and Lewis, 1963), and this is also the value proposed by the British Standards Institution Technical Committee on Haematological Equipment and Methods (Lewis, 1965).