probably some measure of protein precipitation, they dissolve and diffuse to the tissues in effective bactericidal and bacteriostatic concentration.

When applied to freshly "debrided" wounds, or wounds with little exudate, a chemical inflammation occurs, attended by superficial coagulation necrosis to a depth of 2 to 4 mm., due to solution of the sulphates in the tissue fluid to a protein-precipitating concentration. Healing, at first arrested, is generally retarded, occurring under or after separation of the eschar. In saline solution the lowest concentration of these salts at which protein precipitation occurs is 0.6% and 0.25% of neutral proflavine sulphate monoacid and proflavine sulphate *B.P.* respectively. It is suggested, therefore, that the optimum concentration of these salts in a sulphathiazole-proflavine powder, for use prophylactically in war wounds, should be 0.5% for neutral proflavine sulphate and 0.2% for proflavine sulphate *B.P.*, and, having regard to the difficulty of effectively applying powders to irregular wound cavities, saline-tragacanth gels of either of the proflavine sulphates, alone, and in the strengths mentioned, might be employed instead.

Our thanks are due to Col. F. Holmes, O.B.E., for his kind permission to carry out this investigation in the hospital under his command; and to Dr. R. P. Liston, of Imperial Chemical (Pharmaceuticals) Ltd., for providing the sample of neutral proflavine sulphate. To Major O. N. Bailey, R.A.M.C., surgical specialist at the hospital, and Capt. H. R. W. Lunt, R.A.M.C., graded orthopaedic surgeon to the hospital, our thanks are also due for the notes on the cases under their care and for their observations on the treatment.

REFERENCES

- Albert, A., and Gledhill, W. (1943). *Pharm. J.*, **151**, 87.
 Browning, C. H., Kennaway, E. L., Gulbransen, R., and Thornton, L. H. D. (1917). *British Medical Journal*, **1**, 73.
 Garrod, L. P. (1940). *Lancet*, **1**, 802.
 Heggie, J. F., Kendall, A. W., and Heggie, R. M. (1942). *British Medical Journal*, **2**, 655.
 McIntosh, J., and Selbie, F. R. (1943). *Lancet*, **1**, 793.
 Mitchell, G. A. G., and Buttle, G. A. H. (1942). *Ibid.*, **2**, 416.
 Raven, R. W. (1944). *Ibid.*, **2**, 73.
 Russell, D. S., and Falconer, M. A. (1943). *Ibid.*, **1**, 580.

HEREDITARY HAEMORRHAGIC TELANGIECTASIS

BY

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This comparatively rare disease is also known as Rendu-Osler-Weber's disease or Goldstein's hereditary familial angiomas. It is defined as a hereditary dystrophy of the capillaries, the disease being transmitted as a Mendelian dominant and affecting both sexes equally. The diagnosis is based on the following criteria: (1) it is hereditary and not sex-bound; (2) the presence of telangiectases or angiomas; (3) the tendency to haemorrhage (Goldstein); to which a fourth may be added—the multiplicity of the lesions.

Although W. Legg first described a case in 1876, he presented it as a case of "haemophilia." Rendu in 1896 distinguished it from haemophilia, and Osler in 1901 described all its features. Parkes Weber described a family in 1907 and reviewed the literature in 1924. Goldstein (1932) made an extensive review of the literature, mentioning about 100 families (affecting 600 persons); but Hurst thought this review only caused confusion, since it included every kind of purpura and haemophilia and even a case of familial nephritis. Hurst and Plummer believed this condition to be more common than the number of cases on record would indicate; Price goes further in expressing the belief that it may be as common as haemophilia. The disease should not be diagnosed unless the patient shows multiple telangiectases with haemorrhage and comes from a family one or more members of which are known to have been affected in the past (Hurst and Plummer). Levy in 1933 stated that there were 320 known cases, all familial, though sporadic forms may arise. Hurst and Plummer (1932), Hampson (1932), and Yates (1932) made valuable contributions by reporting complete family histories of this condition. More recently (in 1944), while the case recorded below was being studied with a view to publication, since it shows unusual features, Campbell published a very similar case,

and, following this, Wolfsohn added yet a further case to the literature.

Case Record

Mrs. C., aged 31, was admitted to hospital for a dilatation and curettage for incomplete abortion. Apart from this she wanted her blood condition investigated as she felt worried about it. The history was that since the age of 10 she had suffered from epistaxis, which soon became frequent and uncontrollable. She bled more from the right nostril than the left. Bleeding would start spontaneously at any time, day or night. It increased in frequency from once fortnightly to nearly every alternate day. It formerly ceased spontaneously, but now she has to control it by application of cold water and finger pressure. In some severe attacks she "bled white," having lost 1 to 1½ pints (her estimate) in the course of 1½ to 2 hours, which was the longest time bleeding continued. As a result she was always anaemic as a girl, suffering from lassitude and periodic swelling of the ankles, which prevented her from carrying out ordinary household duties. When free from attacks for a more prolonged period—i.e., two or three weeks—a strikingly high colouring of the cheeks had been observed. In such an instance her blood picture was: Hb, 112%; R.B.C.s, 6,250,000; C.I., 0.9; W.B.C.s, 7,000; and on the strength of this and the history of bleeding she was diagnosed as a possible case of "polycythaemia" and told to avoid red meat, and the iron which she was taking regularly was discontinued. She also complained of headaches, which started at the age of 10. Often these headaches, which were parietal and frontal, preceded the epistaxis, and the only thing which seemed to make them better was a "good nose-bleed." She never had vomiting, fortification spectra, or blurring of vision. At about the same age she noticed that some red spots had appeared on her face. The first was on the right upper lip, then next on the tongue and cheeks and in the mouth. Later she had discovered a few on the ends of her fingers. She has never bled from these spots. In the family history we find that her father had similar spots all over his face and fingers. One such bigger spot used to bleed. He also had severe headaches preceding epistaxis; the latter came from spidery naevi on both sides of the nasal septum. His case was regarded as thrombocytopenic purpura, but no blood reports are available. Transfusion was considered, though his heart condition was thought to make this too hazardous a procedure, so it was not performed. These symptoms increased in severity in middle life, and he died at 66 from severe loss of blood and mitral stenosis.

Further details of the family history are shown in the accompanying chart. Nearly all had epistaxis and telangiectasis, but a history of headaches could not be ascertained.

The obstetric history is that the patient has two live children and there have been two miscarriages. Menses are regular; there is no excessive loss. On the contrary, the tendency is to lose less now than formerly. Tonsillectomy was performed at 18, after which she had violent bleeding. There is no history of haemorrhage from anywhere else. A careful examination of the patient was carried out 8 weeks after her dilatation and curettage. Signs of anaemia were present—e.g., pale conjunctivae, lips, and koilonychia. Multiple asymmetrical telangiectases were present, mostly distributed over the upper lip and tip of the tongue, and on the cheeks; they were flat round spots with a red centre and paler periphery. It was remarkable that their colour disappeared, diminished, or remained unchanged on vitreopression, according to their size, intensity of colour, and situation. Thus the very dark ones and the larger spots on the cheeks would be unaffected, but the smaller spots on the lips and tongue would disappear. This confirms the finding of Aubertin *et al.* (1933). The blood pressure was 130/70, and Hess's test was negative. Anterior rhinoscopy revealed a spidery naevus 1½ in. by 1/4 in. on the right septum about 2 in. from the external nares. This had a scab on it, and bled with the slightest trauma. Several other spots were present on the left anterior turbinate and in the hypopharynx. The liver and spleen were impalpable. The cardio-respiratory system showed nothing of note. The blood picture (after

a haemorrhage) was: Hb, 66% (Sahli); erythrocytes, 4,400,000; C.I., 0.8; leucocytes, 9,500; reticulocytes, 1.5%; Wintrobe V.I., 0.80; differential white count, normal; coagulation time, 2½ minutes; bleeding time and clot retraction, normal; platelets, 400,000. Some weeks later the blood showed: Hb, 55%; R.B.C.s, 4,500,000; C.I., 0.6; platelets, plentiful.

A series of skull radiographs were taken and examined stereoscopically for a calcified angioma or naevus, but no abnormality was found.

Discussion

In the differential diagnosis haemophilia, pseudo-haemophilia, erythraemia, the purpuras, leukaemia, Banti's syndrome, and hypertension have been excluded. The unusual feature was that in this case epistaxis caused evident clinical anaemia (Lévy), but at times there was polycythaemia. In view of the fact that this occurred when the patient was free from an attack for some 3 weeks or more, it is suggested that it was due to a physiological bone-marrow response to repeated haemorrhages: such over-compensation is known to result from excessively frequent blood donation by healthy persons. No previous reference to polycythaemia in this condition could be found, though Lévy in 1933 stated that the haematological picture may vary. The headaches were another unusual feature. Campbell's case differs in that the headaches have a more pronounced migrainous character, though they do not justify the use of the term "migraine," which is usually applied to a well-defined clinical entity. There are no previous case reports of this condition being associated with headaches, excepting the mention by Osler (1938) of the association of Lindau's syndrome in angiomatosis—a calcified pial angioma accounting for the headaches and other complaints (glaucoma); Hurst in 1932 also mentioned the possibility of angioma of the brain. It is suggested that in both Campbell's case and the present one the headaches may be due to a relative polycythaemia causing engorgement of the cerebral vessels and terminating by epistaxis. In two cases of the second generation of this patient's family epistaxis could not be definitely ascertained; the presence of the malady is, however, not excluded, since they may have been "frustrated forms" (Lévy, 1933). It is interesting to note that this patient did not suffer from menorrhagia—which is in agreement with the literature. A study of the family history favours Campbell's view that the prognosis of the disease is better than that given by Osler, although the condition obviously aggravates concomitant disorders. The immediate treatment of epistaxis is to control it by the inflated-finger-stall method; later, cauterization of the bigger naevi may be carried out—though new naevi are likely to appear.

Summary

A case of hereditary haemorrhagic telangiectasis with a family history of four generations and presenting some unusual features is reported. The literature is discussed in relation to this case.

My warm thanks are due to Mr. G. O. Taylor, Honorary Surgeon, Dorset County Hospital, for allowing me to publish the case; and to Dr. T. V. Cooper, County Pathologist, for the pathological investigations, for indicating the probable diagnosis of the case, and for help and encouragement in preparing this report.

BIBLIOGRAPHY

- Aubertin, C., Lévy, R., and Baclesse, Mme. (1933). *Presse méd.*, **41**, 185.
Goldstein, H. I. (1932). *Arch. Derm. Syph.*, N.Y., **26**, 282.
Hampson, A. C. (1932). *Guy's Hosp. Rep.*, **82**, 87.
Hurst, A. F., and Plummer, N. S. (1932). *Ibid.*, **82**, 81.
Osler, W. (1938). *Principles and Practice of Medicine*, 13th ed., p. 1090, New York.
Price, F. W. (1941). *Textbook of Practice of Medicine*, 6th ed., p. 889, London.
Whitby, L. E. H., and Britton, C. J. C. (1939). *Disorders of the Blood*, 3rd ed., p. 307, London.
Yates, A. G. (1932). *Guy's Hosp. Rep.*, **82**, 96.

The Ministry of War Transport has notified owners and masters of merchant ships that dimethyl phthalate is now included in the medical scales and should be used as a repellent against mosquitoes when vessels are in malarious areas. The instructions for use are: After sunset a few drops of the liquid should be placed in the hand and applied to the exposed parts of the body, avoiding the eyes, the forehead just above the eyebrows, and areas around the mouth. The liquid should also be applied to the clothing if mosquitoes are biting through it. The effect of the repellent lasts for only about four hours, and a further application should be made when mosquitoes renew their attack. As dimethyl phthalate is a solvent for lacquer, paint, and plastic articles, it should not be smeared on spectacles, watch-glasses, fountain-pens, etc.

NEWER CONCEPTS OF BREAST-FEEDING

BY

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The decline of breast-feeding among civilized races gives the greatest concern to the medical profession. It is the endeavour of the paediatrician to explore every possible avenue in order to remedy this disconcerting state of affairs. With this objective I wish to draw attention to the importance of expressing some breast milk immediately *before* each feed, and the advantage of giving at each nursing *both* breasts instead of one. This routine distinctly helps the proper establishment of lactation.

Most textbooks on paediatrics stress the importance of completely emptying the breast *after* each feed. It is claimed that this is a *sine qua non* for the maintenance and stimulation of an abundant milk supply. It is mainly for this reason that only one breast is permitted at a feed. Although mothers are instructed to empty the breast completely after each nursing, relatively few carry this out—first, because it is very irksome, and, secondly, because the improvement observed is almost negligible. The emptying of the breast at the *end* of each nursing is in my experience not nearly so helpful as the expression of some milk *just before* baby is fed.

The lactating mammary gland consists of 12 to 20 lobes round the nipple. Each lobe is a miniature mammary gland and is composed of smaller lobules. Each lobule consists of a number of alveoli lined with large epithelial cells. The relationship of the lobules and the alveoli to the main duct of the lobe is analogous to that of a bunch of grapes to its stalk. Each alveolus possesses a duct, and the alveolar ducts from each lobule unite to form larger ducts, and finally form the main canal which expands in the areola as the lactiferous sinus. These sinuses, which serve as milk reservoirs, contract at the base of the nipple and pursue a straight course to its summit, perforating it by separate orifices considerably narrower than the ducts themselves. The surface of the nipple is corrugated and provided with secondary papillae. It is perforated by from 12 to 20 excretory ducts, the orifices of which appear at the bottom of these corrugated papillae.

Failure in a large percentage of breast-feeders can be traced to a block either within the lumen of these fine ducts or at their exits. Such obstruction must lead to an accumulation and damming back of milk in the sinuses, ducts, and the whole parenchyma concerned. The effect of such back pressure is engorgement and a lumpiness in the mammary gland which, unless relieved, may lead to resolution and retrogression of that particular sector of the breast. In this way the baby is deprived of a quantity of milk which would otherwise have been contributed by that portion of the gland. In the course of days other ducts may become similarly occluded, with further loss of nourishment for the baby. Thus the condition becomes distinctly aggravated. The mother finds that she has on her hands a restless and unhappy baby, failing to thrive and plagued with intestinal colic as a result of insufficient food and excessive swallowing of air.

The destiny of the breast parenchyma, when there is duct occlusion within either the areola or the nipple, is as follows: There is a damming back of the milk throughout the lobe concerned, and if several are so involved the breast becomes distinctly heavy and painful. Instead of the tissues being lax and freely mobile so as to enable the infant to pull the areola well into its mouth, they become tense, hot, and unyielding, and the baby finds great difficulty in grasping the breast. Its efforts are mostly spent on the base of the nipple rather than well over the areola, with the result that the papilla often cracks. This not infrequently leads to an ascending infection and sometimes to a breast abscess.

Importance of Expressing Some Milk before Feeding

The question which immediately presents itself is, What factors are responsible for occlusion of the lactiferous ducts? It is quite obvious that such minute canals could easily become blocked either by milk products within the areolar sinuses or nipple ducts, or through the viscosity of colostrum in the early days affording resistance to outflow. The openings of the ducts on the surface of the mammary papilla are often occluded by coagulated milk, toilet powder, or desquamated epithelium due to the friction of clothing on the nipple itself.

If we examine the manner in which milk leaves the breast of a mother whose baby is adequately and successfully fed we find, on manual expression, the number of jets expelled from the nipple are many—12 or more. They do not appear all at once—usually three or four at a time—depending on the site of pressure exerted on the areola by the thumb and forefinger of the expressing hand. This demonstrates that the successful functioning of the breast depends mainly on the patency of the ducts. In cases where difficulty prevails we find only one or two jets appearing, and no more. This