pointed out long ago by Sir Leonard Parsons and his associates.

All this raises the question whether Mollison *et al.* (1948) are right in maintaining that the evils which flow from Rh incompatibility can be put down to the effects of blood destruction or whether the Rh antibody is able to fasten on fixed somatic cells and wreak damage there. Support for the second view is given by Boorman and Dodd (1943), who have shown that the Rh factor is a general characteristic of many tissues, and consequently many if not all tissues are likely to be susceptible to injury by Rh antibodies. The Birmingham school (Baar, 1946) also produced evidence of what we may term primary hepatic damage by Rh antibodies.

Another point of interest is that the mother of our patients showed no Rh antibodies and failed to produce them after an injection of 0.05 ml. of Rh-positive blood obtained from one of her sons, but showed them after injection of 5 ml. of her husband's blood. Something similar was found by Drummond and Watkins (1946), and the assumption is that the antibodies which the mother had originally died out with the passage of time, only to reappear under suitable stimulation. On the other hand, Gerrard (personal communication, 1949) found blocking antibodies in a woman 18 years after she had given birth to a child with icterus gravis. The child concerned died of cirrhosis of the liver at the age of $3\frac{1}{2}$, and the case was reported by Braid and Ebbs (1937).

If, from the fate of my patients, I may be allowed to draw any conclusions about the outlook in similar cases, I may be tempted to say that splenectomy improves the prognosis, but cannot be sure that this is the case when I recall that Frank did not bleed until after his splenectomy, and that Kathleen's death was almost certainly the result of a fortuitous gastric erosion superimposed on a dilated vein. Whitby and Britton (1946), however, are of the opinion that splenectomy influences the prognosis favourably. It may be that in future instead of a simple splenectomy some form of porto-caval anastomosis will be undertaken in similar cases.

The three patients described, though they showed no leucopenia or progressive anaemia, would qualify for inclusion in the so-called Banti's syndrome. What Banti (1894, 1910) had in mind when he described the disease to which he gave his name is not absolutely plain, and subsequent commentaries have not helped to clarify the issue. It has been obvious for some time, however, that the name Banti's syndrome has been used as a cloak for a variety of diseases. It may be that further work will sort out one of the entities lumped together under the title of Banti's disease by confirming that fibrosis of the spleen and liver, with enlargement of these organs, and especially of the former, occurring in the young and young adults and accompanied by a tendency to haematemesis, is at times due to Rh incompatibility.

Summary

A family is reported in which three out of five sibs were affected with hepatomegaly and splenomegaly and a tendency to haematemesis.

Reasons are given why this may have been caused by Rh incompatibility.

It is suggested that the condition observed may eventually be segregated from the Banti syndrome.

Grateful thanks are due to Dr. W. Weiner, Director of the Midland Region Blood Transfusion Service, for details of the table setting out the various blood groups of family T. and for his helpful advice; and to Dr. H. S. Baar for his opinion on the pathological sections.

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MIKULICZ'S SYNDROME

BY

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A considerable number of cases of combined lacrimal and salivary glandular enlargement have been reported since Mikulicz (1888) first described the syndrome which now bears his name. Nevertheless the condition is rare enough to prompt the recording of a recent case seen in the Western Infirmary, Glasgow. Since this hospital was opened in 1873 there apparently have been only two other in-patients in whom this diagnosis has been made, both in 1936. On fuller investigation, however, it seems that neither showed enlargement of the lacrimal glands, and their cases can hardly be considered to fall into this syndrome. One was a boy aged 7 with acute myeloid leukaemia and moderate enlargement of the salivary glands, and the other a man of 26 who had bilateral parotid enlargement but no other abnormality. It would seem, therefore, that this is the first case of Mikulicz's syndrome to be seen, at any rate as an in-patient, in the Western Infirmary in 75 years.

Case Report

The patient, a man aged 73, was admitted for the first time on Sept. 30, 1947, complaining of swellings in front of both ears, under the lower jaw, and above both eyes. These had appeared gradually over a period of 12 months, and for a month before admission vision had been much impaired by the presence of the large supraorbital swellings. Apart from these swellings there was no complaint, and the patient had given up his job as a forester only on account of difficulty in seeing. He could remember no previous illnesses. Both parents died when over 90, and all siblings had survived to similar ages.

Examination revealed a well-built healthy-looking man. The most striking abnormality was the symmetrical enlargement of all salivary and both lacrimal glands (Fig. 1). Chemosis of the conjunctivae of both upper lids was present, but neither xerophthalmia nor xerostomia was a feature. In addition there was generalized enlargement of all groups of

FIG. 1.—Appearance on Oct. 6, 1947—before treatment.

superficial lymph glands and also of those of the mediastinum as shown on x-ray examination of the chest. The superficial glands ranged up to $\frac{1}{2}$ in. (1.25 cm.) in diameter and were moderately firm, mobile, discrete, and painless. The spleen was enlarged to 4 in. (10 cm.) below the costal margin.

Blood examination (see Table) showed normal values for haemoglobin, erythrocytes, and total leucocytes, but a neutropenia and lymphocytosis were features of the differential count

Table of Blood Findings

Date	Hb* (g.%)	P.C.V. (%)	R.B.C. (10 ⁶)	W.B.C.	Neut. (%)	Eos. (%)	Bas. (%)	Lymph. (%)	Mon. (%)
30/9/47	14.0	40.0	5.42	3,500	33.0	2.0	1.0	55.0	9.0
16/10/47	13.0	39.0	—	6,800	42.0	1.0	0.5	46.5	10.0
30/10/47	12.9	40.0	—	6,200	35.0	0.5	0.5	60.0	4.0
4/11/47	13.9	41.0	—	7,200	42.0	3.0	1.0	51.0	3.0
26/11/47	13.2	38.0	—	4,080	59.0	0	0	29.0	12.0
6/2/48	14.4	43.0	4.59	6,240	62.5	0	0.5	29.0	8.0
13/4/48	12.0	40.0	4.91	5,840	39.5	1.5	0.5	53.0	5.5
20/4/48	12.5	39.0	4.65	6,440	37.5	3.5	0.5	54.0	4.5
19/5/48	13.4	41.0	4.51	6,080	38.0	0	0.5	57.0	4.5
18/8/48	12.2	38.5	4.72	5,240	36.5	1.0	0	59.0	3.5
26/8/48	12.5	42.0	4.63	6,600	34.0	0.5	0	57.0	8.5

before treatment. Sternal-marrow examination revealed normal erythroid and myeloid series with possibly a slight increase in lymphocytes. The Wassermann reaction was negative. The erythrocyte sedimentation rate was never more than 5 mm. in one hour by Wintrobe's method. Plasma protein levels were albumin 4.04 g. %, globulin 1.81 g., and fibrinogen 0.47 g.

A lymph-gland biopsy was carried out and Professor D. F. Cappell reported as follows: "The lymph node shows general overrunning of the architecture with small lymphocytes, which infiltrate the capsule and extend from the hilum into the surrounding fat. Only traces of a follicular structure are identifiable. The condition is one of the ill-defined systematized lymphoid hyperplasias. The association with lacrimal and parotid enlargement suggests Mikulicz's disease, of which the lymphoid form is one variety. It would not be surprising if this condition terminated with lymphatic leukaemia."

Following this report an attempt was made to alleviate the patient's main symptoms by deep x-ray therapy, applied both generally and locally. Within a fortnight of beginning treatment there was obvious diminution in the size of the salivary and lacrimal glands and to a smaller extent of the cervical lymph glands. By Nov. 29 there was very pronounced regression of salivary (especially parotid) and lacrimal glands, but the spleen and lymph glands were not greatly altered. The patient was discharged at this time.

When seen again on Feb. 6, 1948, he looked and felt extremely well. Lacrimal and parotid glands were no longer visibly enlarged (Fig. 2). The submandibular glands were still palpable

FIG. 2.—Appearance on Feb. 6, 1948—three months after first course of treatment.

and the spleen and lymph glands unchanged. Blood examination showed essentially normal values, the previously noted lymphocytosis having disappeared.

The patient was seen again on April 12; there was then moderate recurrence of the swelling of both lacrimal and submandibular glands. Neither parotid was palpably enlarged and the condition of lymph glands and spleen was unaltered. Blood

examination showed a slight fall in haemoglobin and a return of the lymphocytosis, although the total white count remained normal. A further sternal-marrow examination, however, showed gross infiltration with lymphocytes, which comprised about 90% of the total nucleated count. X-ray therapy was

reinstated on April 14, and a month later complete regression of lacrimal and submandibular glands had taken place, but other findings, including the leucocyte picture, remained unchanged.

By Aug. 17 there had been sufficient recurrence of the lacrimal swellings to cause difficulty in seeing, and the patient was readmitted. An interesting feature on this admission was the presence of considerable swellings in both lower eyelids, presumably consisting of lymphoid tissue. These swellings were some distance from the free margin of the lids and appeared to be close under the skin; they were certainly not connected with the tarsal glands. Parotid glands were visible and palpable but not grossly enlarged. The submandibular glands could not be felt. Lymph glands generally were still enlarged and the spleen now reached 6 in. (15 cm.) below the costal margin. Otherwise there was no change and the patient felt and looked well. Blood examination showed values for haemoglobin, red cells, and total white cells to be still within normal limits, though the differential white count showed a persistence of the lymphocytosis and neutropenia that had been a feature of previous examinations. A further sternal-marrow film showed the presence of large numbers of lymphocytes, comprising some 95% of the total nucleated cells.

Further local x-ray therapy resulted in rapid regression of the swellings around the eyes, but the lymphocytosis persisted. It may be remarked here that the only occasion on which this latter feature was modified by treatment was during the initial course of x-ray therapy, when the whole body was irradiated.

The patient was discharged to his home on Aug. 26, and thereafter his general condition seems to have deteriorated fairly rapidly. He was confined to bed from mid-September and died on Sept. 27. His practitioner, Dr. W. D. McLellan, wrote to say that the lacrimal and parotid glands remained of normal size, but there was a rapid and enormous increase in the size of the spleen, which became very tender. Cough and a blood-stained sputum became troublesome symptoms, and there were signs of an increase in size of the mediastinal glands, with collapse of the left lung and a pleural effusion. Blood examination could not be made and a post-mortem examination was not possible; it cannot therefore be said with any certainty that he developed a terminal leukaemic phase, but such an event seems not improbable.

Discussion

Various classifications of Mikulicz's syndrome have been proposed, notably those of Howard (1909), Thursfield (1914), and Schaffer and Jacobsen (1927). Howard's classification, based on four personal and 51 reported cases, has the merit of simplicity, all cases being divided into three groups: (1) Mikulicz's disease proper, (2) pseudo-leukaemia, and (3) frank leukaemia. Thursfield, who described only one case, made an intensive study of the literature and put forward a scheme differing somewhat from Howard's and including tuberculosis, syphilis, and gout as aetiological agents. Schaffer and Jacobsen, with personal experience of ten cases, proposed a classification into Mikulicz's disease (familial and acquired) and Mikulicz's syndrome (due to leukaemia, tuberculosis, syphilis, lymphosarcoma, toxic agents, gout, and other diseases).

The study of these and numerous other papers reporting solitary examples leads to the conclusion that Mikulicz's syndrome is a manifestation of a general involvement of lymphoid tissue rather than a disease of the lacrimal and salivary glands themselves. The involvement may be due to lymphatic leukaemia, but in many cases, as in the present one, there has been only a moderate lymphocytosis in the blood or no deviation at all from normal. It would appear likely that many of these will eventually develop the full picture of chronic lymphatic leukaemia. In the present case, despite the development of a grossly abnormal marrow picture, the peripheral blood showed only a slight absolute lymphocytosis even after a course of at least two years.

Many, if not all, examples of this syndrome described in the past as being due to tuberculosis (e.g., Garland and Thompson, 1933) would probably now be classified as examples of sarcoidosis, while a study of the clinical features of those ascribed to syphilis leaves the impression that in many the syphilitic state may have been purely coincidental. Why Mikulicz's syndrome should be so rare when the pathological states which give rise to it are relatively so common does not appear to have been explained.

Summary

A record is presented of the findings in a case of Mikulicz's syndrome (enlargement of lacrimal and salivary glands) followed from the initial hospital admission until death 14 months later. Many of the features of chronic lymphatic leukaemia, including lymphocytic infiltration of the sternal marrow, were present, but the total leucocyte count was not raised during the period of hospital observation. Swelling of the lacrimal and salivary glands was treated by x-ray therapy with satisfactory though temporary results.

Brief reference is made to the more important literature on the subject.

I should like to express my thanks to Professor J. W. McNee, in whose wards the patient was investigated and treated, for permission to publish this account, to Professor D. F. Cappell for permission to include his report on the gland section, and to Miss M. M. Todd for the photographs.

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A SIMPLE EMERGENCY METHOD FOR ESTIMATION OF BLOOD SUGAR*

BY

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A simple and accurate method for estimating blood sugar, either in the ward or in the patient's home, has long been required. The technique of Haslewood and Strookman (1939) as described by King (1946), when used in conjunction with some simple apparatus we have designed, would seem to satisfy these criteria. This apparatus can easily be made by any competent glass-blower. No colorimeter or photometer is necessary.

Apparatus

The following apparatus is required: (1) a number of clean and dry teat-end pipettes; (2) one blood pipette calibrated to contain 0.1 ml., for measuring the blood sample; (3) one pipette calibrated to contain 0.2 ml., for measuring the solution of 10% w/v sodium tungstate; (4) a 1-in. (2.5-cm.) glass funnel and a supply of 5.5-cm. Whatman No. 41 filter papers; and (5) four tubes as shown in the accompanying diagram.

Tube A has an internal diameter of approximately 10 mm. and is calibrated to contain 3.7 ml. It is fitted with a rubber bung.

Tube B has an internal diameter of approximately 13 mm. and is calibrated to contain 7 ml. at the lower mark and 14 ml. at the upper. It is fitted with a rubber bung.

Tube C has a narrow portion approximately 5 cm. long with an internal diameter of approximately 8 mm., and a wider portion approximately 16 cm. long with an internal diameter of approximately 13 mm. It is calibrated by ring markings to contain 1, 2, 5, 10, and 20 ml. The length between the 5- and 20-ml. calibrations is further subdivided into 24 equal portions by alternate short and long etched lines. The long lines from below upwards are marked 100,

The four tubes required. Figures shown at the sides of the tubes are etched on the reverse faces of the tubes.

120, 140, and so on up to 320, which coincides with the 20 ml. mark. These markings represent mg. of glucose per 100 ml. of blood.

Tube D is similar to tube C and of identical measurements. It also has ring calibrations at 1, 2, 5, and 10 ml., the length between its last two markings being subdivided into four portions. These are determined by adding calculated volumes of water, namely:

At calibration	70	the tube contains a total volume of	5.7 ml.
"	"	60	" " " " 6.7 ml.
"	"	50	" " " " 8 ml.
"	"	40	" " " " 10 ml.

Solutions Required.—These are exactly as described by King (1946). The standard solution contains 2 mg. of glucose per 100 ml. and is prepared fortnightly by suitably diluting the stock solution containing 0.1 g. of glucose per 100 ml. with saturated benzoic acid.

Method

Tube A is filled to the mark with the isotonic sodium-sulphate-copper-sulphate mixture by means of a teat-end pipette. Blood obtained by capillary puncture is collected in a tube of internal diameter about 8 mm., which contains a small quantity of neutral potassium oxalate to prevent coagulation.