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President—E. D. D. DAVIS, F.R.C.S.

Discussion on Throat and Nose Manifestations of Blood Diseases

By W. M. MOLLISON

THE diseases chosen for consideration are : (1) Bleeding of the newborn. (2) Essential thrombocytopenia or purpura hæmorrhagica. (3) Polycythæmia vera. (4) Hæmophilia. (5) Hereditary telangiectasia or Osler's disease. (6) Agranulocytosis. (7) Leukæmia. (8) Achlorhydric anæmia. (9) Avitaminosis. (10) Glandular fever. Though the first three of these diseases exhibit bleeding from the nose and gums, in the absence of any local cause the pædiatrician takes over the case.

(4) *Hæmophilia* has generally declared itself in the boy before the laryngologist is consulted ; all operations are to be avoided. In suspected cases full examination of the blood must be carried out before any operation. Even a normal clotting and bleeding time and normal platelets do not always save the surgeon from disaster.

(5) *Osler's disease* is rare but embarrassing ; bleeding occurs spontaneously from the skin and mucous membranes ; several members of the family are similarly afflicted. The following case gives a typical picture : A man aged 40 gave a long history of severe epistaxis. This occurred daily for three weeks, resisting all accepted treatment, but the resulting anæmia may have caused cessation of the bleeding. On recovery from anæmia bleeding again started ; this cycle had recurred over a period of years. Since cauterization had failed it was decided to use surgical diathermy under an anæsthetic ; bleeding was extremely difficult to stop and there was no lasting effect. His father, an uncle, brother and a sister suffered from epistaxis.

(6) *Agranulocytosis*, as its name indicates, is a blood disease in which there is absence or great diminution of the granular cells in the blood. It occurs after the ingestion or injection of certain

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drugs—pyramidon, sulphanilamides (though not till 30 gm. have been taken), sanocrysin, arsenical compounds and benzol. It also is seen after exposure to deep X-rays. Owing to the inflammation and ulceration of the tonsils, palate and gums the disease is called agranulocytotic angina. The disease has a high mortality. The patient is very ill with a high temperature often out of proportion to the condition of the pharyngeal lesions; death may take place in a few days. There is, too, a chronic form in which treatment leads to temporary improvement in the blood picture; cases have lived for two years or more. Even slight operations are apt to produce serious relapses with extensive ulceration in the mouth; in one case extensive ulceration of the palate, gums and tonsil followed extraction of a tooth. Alcohol increases the symptoms; in some cases symptoms are connected with menstruation; a case has been seen as a complication of enteric fever. Rigors produced in the course of the disease lead to an enormous leucocytosis with cure. In a case recently published by Cross a leucocytosis of 40,000 followed a third blood transfusion for agranulocytosis and cure was immediate and permanent. One case of pemphigus has been seen in which prontosil caused disappearance of the skin lesions but was followed by agranulocytosis and on withdrawal of the prontosil the pemphigus recurred with fatal results.

(7) *Leukæmia* produces severe ulceration of the tonsil and is only distinguished from agranulocytosis and other tonsil ulcerations by a blood examination. Prognosis is fatal.

(8) *Achlorhydric anæmia.* Professor Witts wrote an admirable paper on this subject analysing fifty cases; the symptom of dysphagia was one of the least frequent exhibited. The disease is a fruitful cause of chronic invalidism in women; its age-incidence is 30 to 50 (compared with 45 to 60 in Addison's anæmia). Most of the cases occur in the asthenic type of woman, her hair prematurely grey contrasting with dark eyebrows; the skin is pigmented but not icteric; anæmia is not obvious till a blood-count is done; the patients are often nervous and difficult; some have never felt well since girlhood, others date the onset from influenza or pregnancy; in 44 of the 50 cases achlorhydria was complete while in the remaining six faint traces only of free acid were present. Treatment consists in giving large doses of iron (iron and ammonium citrate) up to 240 grains a day, and is successful, but return of hydrochloric acid in the gastric juice is never seen. The tongue may be bald towards the tip and dorsum. The dysphagia was only slight and transient in four cases, severe and the dominant symptom in two. In my experience complete removal of the teeth has been carried out in almost all cases.

In 1919 Brown-Kelly described a symptom-complex in women complaining of dysphagia: spasm at the entrance to the

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œsophagus ; smooth tongue ; fissures at the corners of the mouth and pale pharyngeal mucous membrane. Oesophagoscopy showed a small circular lumen which could be opened easily with much relief. Similarly Paterson described a clinical type of dysphagia with spasmodic dysphagia and/or superficial glossitis ; he noted also changes in the mucous membrane which tended to make it crack on the passage of a tube. The narrow lumen just below the cricoid appeared to be due to spasm of the crico-pharyngeus muscle and this led to the suggestion that this muscle was unable to relax.

There seems to be some tendency towards the development of malignant disease at the site of the narrowed lumen or perhaps on the posterior wall of the cricoid. Authorities are by no means in agreement about the frequency of this complication. Some speak of it as frequent but Vinson and Lamb consider it rare, and I incline to this view.

(9) *Avitaminosis* supplies two diseases with nose and throat manifestations—scurvy and rickets. Scurvy occurs usually between the ages of 6 months and 3 years; it causes hæmorrhages under the periosteum ; when the swelling occurred over the frontal bone a mistaken diagnosis was made of acute osteomyelitis ; incision revealed no pus and some lemon juice cured the patient. Laryngismus stridulus is a manifestation of rickets and may be confused with other causes of stridor.

(10) *Glandular fever* is characterized by sore throat and enlarged glands at first cervical but later epitrochlear, axillary, inguinal and mediastinal ; it is the last group that produces cough and perhaps stridor. The blood shows a leucocytosis up to 60,000 of which 80 per cent. are lymphocytes.

By R. G. MACFARLANE

I HAVE subdivided the buccal manifestations of the blood disorders morphologically into hæmorrhagic, infiltrative, atrophic and ulcerative.

The hæmorrhagic lesions are usually manifestations of one or other of the hæmorrhagic states, associated with a vascular defect, or with faulty blood coagulation. In the hæmorrhagic purpuras there is usually a demonstrable vascular defect, revealed by decreased capillary resistance or a prolonged bleeding time which is often, but not always, accompanied by thrombocytopenia. These purpuras may be idiopathic, or secondary to toxæmia, aplastic anæmia, or leukæmia, and the type of bleeding they produce is characteristic. There is often persistent oozing from apparently intact mucous membranes, particularly of the nose and gums, which may in fact be the only symptom of the underlying disease. Bleeding into the tissues is in the form of petechial hæmorrhages or superficial ecchymoses, and deep hæmorrhages are rare. Hæmorrhagic

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telangiectasia is an example of a localized vascular defect. Lesions in the nose may produce such persistent epistaxis that a severe anæmia develops, but in the mouth they do not often bleed unless injured.

The other type of hæmorrhagic diathesis is due to defective blood coagulation, and includes hæmophilia and hypoprothrombinæmia. In these states uncontrollable bleeding as a rule follows injury and there is a liability to form extensive hæmatomata. I have seen a hæmophilic boy die of asphyxia following hæmorrhage into the base of the tongue before anything could be done to save him, and another who on several occasions developed hæmorrhage into the tissues of the throat, and required intubation to prevent the impending asphyxia.

Infiltrative lesions are the result of deposition or proliferation of abnormal cells. In the leucoses, the cell type affected may infiltrate the mucous membranes of any part of the nose, throat, mouth or larynx. This infiltration may be diffuse or localized. Ulceration of the surface follows such invasion of the mucous membranes, usually in the acute leucoses, and particularly in the monocytic type in which angina and gingivitis may be the presenting signs. The lesions are complicated by the hæmorrhagic purpura that is frequently the terminal event in these diseases. There is no clearly defined boundary between the leucoses and frankly malignant states with a leukæmic blood picture. Leukæmic plasmocytoma may arise from any of the bones of the skull; leucosarcoma, involving the tonsils, may have a leukæmic blood picture; and chloroma, with the blood picture of myeloblastic leukæmia, may invade any part of the skull and upper respiratory tract.

The origin of the mouth and throat lesions of glandular fever is more obscure, as autopsy material is rare. They are probably due to infiltration by the abnormal cells present in the blood, and Glanzmann (1930) considered that lymphocytic infiltration of the mucous membranes in some of his cases are responsible for them. Throat lesions occur in about 80 per cent. of cases and angina is not uncommon. The throat lesions of acute leukæmia, agranulocytosis, glandular fever and acute infection may be very similar. Examination of the blood, or if necessary of the bone-marrow, will, usually, provide a certain differentiation.

The atrophic lesions include the glossitis, stomatitis, and pharyngitis that are associated with the deficiency anæmias, in which there is typically an atrophy of the surface epithelium; sometimes of underlying muscle accompanied by chronic inflammation and occasionally ulceration. There are many points of similarity between pellagra, sprue, steatorrhœa, pernicious anæmia, the tropical macrosytic anæmias, and the anæmia associated with gastro-intestinal operations and diseases. In all these there is a

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tendency to develop glossitis, stomatitis, and disorders of the gastro-intestinal tract, changes in the nervous system, pigmentation and lesions of the skin, anæmia, usually macrocytic, leucopenia and thrombocytopenia. Moreover all these conditions have been reported to respond favourably to treatment with crude liver, and their manifestations may be produced experimentally by withholding the factors contained in autolysed yeast known collectively as vitamin B. While pernicious anæmia will respond to a highly purified liver extract, the other conditions often fail to do so, though responding to crude liver or autolysed yeast.

It may be supposed, therefore, that these diseases are the result of a deficiency of the factors contained in crude liver, and derived from autolysed yeast, of which at least one, the anti-pernicious anæmia factor, requires the collaboration of Castle's intrinsic factor for its synthesis.

In pernicious anæmia the deficiency is conditioned by the absence of the intrinsic factor, and may be limited to the anti-pernicious anæmia factor as represented for the moment by anahæmin. The failure of other conditions to respond to anhæmin shows that these manifestations result from the lack of some other, or more than one, factor.

The group of factors required probably includes nicotinic acid, riboflavine, the so-called extrinsic factor and others as yet unidentified. The lesions produced by their deficiency are almost certainly the result of the lack of these substances in the enzyme systems on which the living cell depends for its metabolism. The oxidation reduction processes, from which the energy for vital activity and growth is derived, are the result of such enzyme chain reactions. The first link of this chain is dehydrogenase, specific for each particular substrate, followed by co-enzymes 1 and 2, flavine enzyme and cytochrome. The co-enzymes contain nicotinic acid, flavine enzyme contains riboflavine, and cytochrome contains iron. The absence of any one of the links of this chain would bring the whole vital process to a standstill, and the partial deficiency of any one is likely to impair functional activity and growth.

If such a deficiency exists, evidence of it might be expected in those parts of the body where there is normally great activity, particularly in the production of new cells. The most obvious example of such tissue is the bone-marrow. Here there is a continual production of relatively enormous numbers of cells, roughly 50 gm. of red cells and leucocytes, and an unknown quantity of platelets, every day. Deficiency of one or more of the factors required for growth and maturation results in a partial or complete breakdown in the process of production with consequent anæmia or leucopenia. Bone-marrow studies in the nutritional macrocytic anæmias reveal a very characteristic picture, that of arrested maturation. The

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marrow is full of primitive cells, most of which seem unable to develop beyond a certain point. The process of maturation when it does occur is distorted, resulting in the production of abnormal red cells and leucocytes. The disorder affects the red cell, white cell, and platelet precursors equally and in the peripheral blood the leucopenia and thrombocytopenia are usually as pronounced as the anæmia. If cells so dissimilar are all affected by lack of one or more of the maturation factors it is probable that other types of cell undergoing rapid production and maturation will be affected also. Witts (1932) has already compared the development of the cells of the bone-marrow to the development of the cells of the skin. Undoubtedly the skin and mucous membranes must provide a continual supply of new cells to replace those lost from the surface. Atrophic skin changes occur in the diseases we are considering, being most pronounced in pellagra, and least in pernicious anæmia, but the skin, being protected by its horny layer, is less susceptible to injury than the mucous membranes.

The epithelium of the mouth has probably to contend with more injury, chronic irritation, gross infection, violent changes in temperature and the application of noxious substances than any other part of the body. It would not be surprising therefore that any retardation of the continual production and maturation of new cells were followed by soreness, atrophy, or even ulceration. The changes found in the deficiency anæmias may be reasonably explained on this basis of general arrest of maturation. That each of the manifestations may occur separately, and will respond to treatment with crude liver suggests that each is the direct result of deficiency and that they are not interdependent. Whether it is the skin, gastro-intestinal tract, bone-marrow or nervous system that is mainly affected depends probably on variations in diet, absorptive and digestive efficiency, and the requirements of the tissues concerned.

Iron deficiency may be accompanied by more specific changes resulting in a reduction of the hæmoglobin content of the red cell, and disordered development of the nails, both of which can be corrected by treatment with iron. Iron is also a factor in general metabolism, as it has already been shown that it is a constituent of cytochrome. The glossitis, atrophy of the lingual papillae and pharyngitis may all be manifestations of this particular deficiency. The dysphagia that completes the Paterson-Kelly syndrome occurs in about 15 per cent. of iron deficiency anæmias. It has been reported in a few cases of pernicious anæmia, and is of importance because the atrophic changes and chronic irritation may predispose to carcinoma, particularly of the post-cricoid area.

Ulcerative lesions of the mouth and throat may be merely an exaggeration of the atrophic changes just described. I have recently been able to observe a case of nutritional anæmia with acute ulceration

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of the mouth, leading to perforation of the lower lip. The patient was a girl aged 14 years, with a previous history of cœliac disease. On admission she was desperately ill, with fever, and severe ulceration of the mouth, there was profound anæmia, the red cells numbering 1·6 million per c.mm., hæmoglobin 26 per cent., and only 600 neutrophils per c.mm. The platelets were 50,000 per c.mm. Bone-marrow examination showed a cellular marrow, with almost complete arrest of erythrogenetic and leucogenetic elements. The primitive red cells were definitely megaloblastic, and the whole picture resembled that found in pernicious anæmia. She was transfused and given liver and marmite, and in seven days the neutrophils rose to over 5,000 per c.mm., the platelets to 300,000 per c.mm., and the ulcers, one of which had perforated, were beginning to heal. She left hospital perfectly well in a month. A second case very similar to this was seen shortly afterwards, this time in a woman of 32 who had suffered from rickets and chronic diarrhœa. The ulceration was not so severe, but again there was an extreme degree of anæmia and leucopenia, and a megaloblastic hypercellular marrow; a rapid improvement followed blood transfusion and a course of liver and marmite. A similar condition has been described by Bowen-Davies (1934).

These cases introduce the problem of agranulocytosis, and the question whether the mouth lesions were due primarily to the maturation factor deficiency, or to the absence of granulocytes. Undoubtedly the granulocytes play an important part in limiting the spread of infection, once a break in the surface has occurred. Agranulocytosis superimposed on the atrophic stomatitis of the deficiency anæmias might be expected to result in severe angina. The granulocytes may be greatly reduced, however, without the occurrence of angina. I have seen cases which neutrophils numbered 100 per c.mm. or less as a result of aplastic anæmia, X-ray irradiation and Felty's syndrome, without mouth lesions. Yet in toxic agranulocytosis there may be acute angina with 2,000 or more granulocytes per c.mm. (Plum, 1937).

Many cases of toxic agranulocytosis have followed the use of amidopyrine and related compounds, including the sulphanilamide derivatives. These drugs may produce in sensitive persons arrest of maturation or even disappearance of the granulocyte precursors in the bone-marrow. The red cells are usually stated to be unaffected, but in my experience fatal cases following the use of sulphanilimide developed a profound anæmia and thrombocytopenia before death.

The possible mechanism of the toxic action of sulphanilamide in this respect might be considered more closely, because the effects of the drug on living cells have been carefully studied. Sulphanilamide not only inhibits the growth of leucocytes in certain people,

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it inhibits the growth of certain bacteria. Investigation of the latter action has shown that sulphanilamide blocks the metabolic process of the organism by interfering with a growth factor identified as para-aminobenzoic acid. This latter substance has a very similar structure to the sulphanilamide, and it has been suggested that the inactive sulphanilamide is incorporated, in mistake, so to speak, for the similar, but active para-aminobenzoic acid in an essential enzyme chain reaction that is thus brought to a standstill. The action can be inhibited by excess of para-aminobenzoic acid.

Dr. R. H. Mole has pointed out that sulphanilamide may have a similar action on developing leucocytes, and has suggested the use of para-aminobenzoic acid in the treatment of agranulocytosis. There is some evidence to favour the view that some at least of the toxic effects of sulphanilamide may be due to interference with the enzyme systems in which the vitamin B factors are concerned. Thus abnormal excretion of porphyrins occurs both in pellagra and following the use of sulphanilamide. Sulphanilamide produces a neuritis in animals that may be prevented by giving vitamin B_1, and it also produces skin lesions that may resemble those of pellagra. Vitamin B deficiency has already been shown to produce leucopenia and stomatitis, and it is possible that toxic agranulocytosis following sulphanilamide may be an extreme form of the same process. If this be so, therapy should be directed towards the supply of the deficient factor.

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The PRESIDENT said that hæmophilia was probably the commonest blood disease encountered in routine work. Hæmophilia seemed to vary; at one period there might be very little bleeding, and at another time the patient was very susceptible. He had a child patient whom he knew to be a hæmophiliac and who had otorrhœa following measles and all the classical signs of an acute mastoid. He resolved, with a little misgiving, to operate on the child. About three-quarters of an hour before operation a hypodermic injection of atropine was given. When the child was ready for operation he was still bleeding from the needle puncture, and therefore the operation was not done. The mastoid cleared up and the child recovered, but died later from hæmophilia.

Another type of case was acute leukæmia. During his long experience at the Royal Dental Hospital he had seen perhaps three of these cases. Attention was drawn to them by reason of a septic

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condition of the mouth and gums. Teeth were extracted, but bleeding continued, and the patient was admitted to hospital very ill, with a temperature of 104° or 105°, and ultimately died in about five days, of septicæmia. Blood examination proved these to be cases of acute leukæmia.

Another condition which was seen frequently was an ulceration in the mouth or on the tonsil of which the diagnosis was uncertain. The Wassermann reaction was negative. It might be an agranulocytosis or some other obscure blood condition. Some cases of acute tuberculosis which were doing badly, with a painful superficial ulceration of the pharynx, epiglottis and larynx, might be mistaken for agranulocytic angina. It would be very easy to confuse a case of acute tuberculous ulceration of the pharynx with one of agranulocytosis. The white cell count in some cases of pulmonary tuberculosis was very low.

R. L. FLETT said that caution was necessary in removing tonsils and adenoids in children with lymphatic leukæmia. In one case of enlarged tonsils and glands in the neck in which he himself was not actually concerned the child died on the table and was found *post mortem* to have lymphatic leukæmia. In certain cases of leukæmia he had seen numerous ulcers down the pharynx and on the epiglottis, and there was some blood staining around each ulcer. He had also seen a Hodgkin's deposit on a vocal cord.

With regard to secondary anæmias where it was necessary to dilate the upper end of the œsophagus, he thought they did the cases some good, but a large number of those with atrophic mucous membrane began to form carcinomas afterwards. Recently he had dilated one of these strictures, secured a very good dilatation, and got the œsophagoscope to go past the stenosed area. A fortnight later he was called to see this patient because she was unable to swallow and was very ill. He thought he must have produced mediastinitis from his over-extensive dilatation, but when the patient died about a fortnight afterwards, a carcinoma was found about 3 in. from the cardia. The stenosed area was shown to be a healing area with a diamond-shaped space where it had been broken.

R. G. MACBETH, in expressing his appreciation of the papers, said that it was interesting to learn that avitaminosis perhaps took such an important part in blood diseases. Had Dr. Macfarlane noticed any increased tendency to bleeding from minor operations, such as tonsils and adenoids or removal of teeth, also had they noticed more cases of epistaxis and so forth associated with lowered vitamin intake? He himself could not quote figures, but he thought he had been having more trouble during the last twelve months from reactionary hæmorrhage, and the dental surgeons had told him that they were encountering the same difficulty.

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They were all accustomed to meet cases of, what he called, " normal abnormalities " such as the child admitted for tonsils and adenoids whose mother would say that, " of course, he always bled excessively if he cut his finger " ; but in the majority of these cases there was an entirely normal bleeding time and clotting time. Occasionally these cases seemed to lose rather more blood at operation, but it was a clinical matter and could not be ascertained in absolute terms.

The question of female bleeders, otherwise healthy, also suggested itself. They were told that hæmophilia was transmitted on the female side but not inherited by the female. One occasionally found women who bled a good deal, and the nursing staff at his hospital were convinced that red-haired women always bled, and the anæsthetists sometimes gave their authority to the same story. A final question was : Should tonsil operations be avoided during the menstrual period ?

J. F. SIMPSON said that it was quite true that one of his colleagues at a Royal Air Force hospital was disturbed during a short period by an unusual number of tonsillar hæmorrhages. He carried out a series of investigations on the vitamin content of the blood and put a number of the patients on to ascorbic acid treatment. The investigations did not show anything conclusive.

I. G. ROBIN said that recently a man aged 60 who had been treated at many hospitals for epistaxis from telangiectases, came in very ill with hæmoglobin under 25 per cent. He had gradually improved on big doses of iron, vitamin K and nasal ionization and liquid paraffin drops. The mucous membrane of the nose was very dry and he bled from many points in the nose. The speaker was not sure how long he should go on ionizing ; he was sceptical of the true value of the vitamin therapy.

Recently he diagnosed the Paterson-Kelly syndrome in a woman aged 50 who had had attacks of dysphagia several times previously and had been admitted to another hospital on a previous occasion. Before doing œsophagoscopy he got a physician to see her and he diagnosed pellagra ; the condition cleared up completely on vitamin B. She had a low hydrochloric acid stomach content but no skin changes. The only changes were in the mouth, a very atrophic tongue and marked dysphagia.

J. C. HOGG said that there was a condition known as reticulosis which was characterized by ulceration in the upper air passages. It was apparently a disease of the reticulo-endothelial system, but perhaps Dr. Macfarlane could tell them whether it was, strictly speaking, a blood disease or not. The first case of the kind in his experience had Vincent's organism and looked like a typical Vincent's infection of the tonsils, though every investigation proved negative. His blood was examined by different pathologists,

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biopsies were taken of glands in the neck, but all were negative, and only when the patient died six months afterwards was the diagnosis established. He had ulceration of his larynx, pharynx, œsophagus, and intestines, and he believed of his stomach as well. Such cases were rare, but he had lately seen a similar case.

The PRESIDENT asked whether it was true that in cases of hæmophilia the clotting time was normal. The family history was very important.

Mr. Robin's case resembled multiple telangiectases of the septum. In one or two cases he had found a 5 per cent. solution of quinine and urethane injected between the cartilage and the mucoperiosteum prove successful.

This treatment was recorded by G. H. O'Kane (*J.A.M.A.*, 1938, cxi, 242).

R. G. MACFARLANE (in reply) said that in glandular fever sometimes the hæmatologist could not help very much in the diagnosis. The Paul-Bunnell reaction might be negative in 30 to 40 per cent. of cases and in these diagnosis could only be made on the abnormal type of lymphocyte occurring in the blood, which might not be present in very large numbers.

Several speakers had raised the question of the diagnosis of hæmophilia. In true hæmophilia he would have said the diagnosis was comparatively easy, because in almost all cases (about 98 per cent.) over the age of 5 or 6 years hæmorrhage into the joints occurred and was a characteristic feature of the condition. There was considerable variation in severity from time to time, there might be a normal coagulation time by the ordinary technique, but it was most important that the investigation be done with extreme care. If venipuncture were bungled there might be a normal coagulation time in hæmophilia, whereas if it had been done properly, the time would be four or five times the normal.

True hæmophilia, he believed, never occurred in females, but there was undoubtedly a hæmorrhagic tendency in some of the females through whom hæmophilia was inherited. He had seen one case of pseudo-hæmophilia in a woman in the sense that he could make no distinction between the condition in her case and that of true hæmophilia, but this was very rare indeed.

Unfortunately the tendency to bleed was not limited to the typical hæmiphiliac. There were undoubtedly people who bled idiopathically, people in whom it was not possible to demonstrate any change whatever in the hæmostatic mechanism, but who were liable to bleed after operation or injury. In one woman who bled for six weeks after a small cut of the finger it was not possible to demonstrate any change in the hæmostatic mechanism.

Menstruation might be regarded as a hæmorrhagic diathesis. At the menstrual period there was a depression in the number of

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platelets and sometimes a prolongation of bleeding time, and although it was of no practical significance in the general run of cases it might be advisable to avoid the menstrual period for operation if possible.

Reticulosis was a collective term covering a large number of conditions, including Hodgkin's disease, so-called "atypical Hodgkin's disease", the leukæmias, the chloro-leukæmias, and the leuko-sarcomas, all of which might have a leukæmic blood picture. Reticulosis might not always have a typical blood picture. Hæmatologists could not as a rule diagnose reticulosis unless there were characteristic cells in the blood-stream.

ABSTRACTS

MISCELLANEOUS

The Masticator Space. COLBY HALL and FRANCES MORRIS. (*Ann. Otol.* (St. Louis), 1941, l., 1123.)

The writers describe a space in which inflammatory processes occur in connection with infection of and operations on the molar teeth. These inflammatory processes have been described as pharyngo-maxillary but the pus is recovered not from the pharyngo-maxillary space, but from the subperiosteal spaces of the mandible.

The masticatory space is a fascial sling containing the muscles of mastication and the ramus of the mandible. It lies between the parotid gland laterally and the pharyngo-maxillary region medially. It encloses the masseter, the external and internal pterygoid muscles and also the temporal muscle. It is limited above by the attachment of the temporal fascia to the skull and below by the attachment of the fascia to the periosteum on the inferior margin of the mandible. The temporal muscle divides the space into a superficial and a deep pouch.

Inflammation in this space usually comes from the lower molar region. It may be more marked either on the inner or outer aspects of the mandible. The swellings in the region so commonly met in the presence of apical abscesses and after molar extractions consist of inflammatory reactions in the contents of the space. When, however, the condition has developed into an abscess the signs and symptoms are augmented and progressive. There may be swellings on both lateral and medial aspects, in only one case out of twenty was there absence of the external swelling. There is trismus, fever

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