help to indicate the diagnosis, and finally, the localization of the larvæ with the formation of a punctate opening in the skin and eventually the recovery of the characteristic larva, will confirm the diagnosis.

While hypodermal myiasis may cause a great deal of discomfort and pain, the prognosis is usually excellent. A fatal case has been reported by Hamilton,[3] but the history would indicate that death was due to a concomitant bacterial infection. The only treatment is surgical removal of the larvæ when they can be localized. If the air-hole has been cut through the larvæ can usually be expressed by gentle digital pressure, or if that fails, the opening can be enlarged by incision and the larvæ removed. Crushing of the larvæ *in situ* should be avoided because of possible anaphylactic shock. While this has never been reported in man, it has occasionally occurred in cattle where the larvæ have been crushed while still in the host tissue.

The term hypodermal myiasis has been used here to indicate specifically an infestation by larvæ of the ox-warble flies, *Hypoderma* spp. In the past this condition has been referred to as creeping cutaneous myiasis, cutaneous myiasis, and creeping eruption. The term "creeping eruption" should be retained for skin infestation by larvæ of certain nematode species (*Ancylostoma braziliense*, etc.), while "creeping cutaneous myiasis" is not sufficiently specific and usually refers to infestation by larvæ of the horse bot fly (*Gastrophilus* spp.). Cutaneous myiasis is purely a descriptive term and refers to the finding of any fly larva under the skin.

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OVERDOSAGE WITH DEXEDRINE

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No instances of a toxic effect of dexedrine on blood-forming organs have been found in medical literature. There are references to benzedrine's successful use in suicidal attempts, but in these latter incidents death followed within several hours, without evidence of damage to blood elements. The case to be reported refers to a nurse-in-training, who consumed massive doses of dexedrine over a prolonged but indeterminate period.

On February 26, 1949, the patient, a female nurse, aged 21 years, complained of tiredness and a sore throat. Previous medical history disclosed only childhood contagious diseases. For the past month there was increasing tiredness. The temperature was 102° F., pallor was obvious; there were enlarged cervical lymph nodes, and the tonsillar surface presented discrete follicular exudate. There were petechial hæmorrhages in the soft palate. Inguinal lymph nodes were enlarged; the spleen was not palpable; generalized petechial hæmorrhages appeared in the skin on February 27, and followed at irregular intervals until death. One dose of sulfadiazine was given (15 grains) but was not repeated; on February 26, penicillin in a dosage of 40,000 units every 3 hours was given and continued for four days. On March 1, the temperature was normal and did not rise above 99° until March 10, when it rose to 101° F. Thereafter it remained high (101 to 105) until death. Epistaxis occurred at intervals but was controlled by nasal packing. On March 13, menstruation began (it was two weeks delayed, having been previously regular, except that the preceding period was described as profuse and lasting 12 days). Bleeding *per vaginam* continued, until death. On March 18, hæmatemesis occurred, and hæmatemesis and melæna thereafter, with vaginal bleeding, gradually exsanguinated the patient in spite of the administration of large quantities of whole blood, a total of 17 litres being given during hospitalization. The Wassermann was negative. Urinalysis was negative except for a few red cells. Several blood cultures were negative. X-rays of the chest were normal. Death occurred March 22. There was no autopsy.

BLOOD STUDIES

February 26.—Hgb., 8.3 gm. White blood cells, 1,500. Neutrophils, segmented 180, juvenile 110; lymphocytes 1,050.

February 28.—Hgb. 9.3 gm. Red blood cells 3.4 million, reticulocytes 0.0%, mean corpuscular Hgb. 27.5 γγ. Platelets 10,000. White blood count 1,300. Neutrophils, segmented 0, juvenile 117, myelocytes 26; eosinophils 13, lymphocytes 1,144.

Bleeding time, 3 minutes; clotting time, 22 minutes; prothrombin, 100%; no abnormal cells seen.

March 1.—Bone marrow, sternal aspiration: nucleated cell count 39.0 thousand (normal, over 50.0); megakaryocytes, none present on smears; erythroid series, maturation arrested at erythroblast level; very few normoblasts present.

Granulocytes.—Predominant cells are myelocytes and metamyelocytes, maturation showing no progression to segmented forms. Eosinophils are present. Lymphocytes are present in normal members.

Opinion.—Maturation arrest involving erythrocyte, granulocyte and megakaryocyte series. Typical of pancytopenia due to drug intoxication.

March 3.—Hgb. 9.1 gm. Red blood cells 3 million, mean corpuscular hgb. 30 γγ.

March 8.—Hgb. 7.6 gm. Red blood cells 2.6 million; reticulocytes 0.0%; mean corpuscular hgb. 30 γγ; white blood cells 3,100; neutrophils, segmented 0, juvenile 124; platelets 30,000.

March 10.—Hgb. 7.3 gm. White blood cells 2,000.

March 15.—Hgb. 9.5 gm. Red blood cells 3 million, mean corpuscular hgb. 31.5 γγ, reticulocytes 0.2%, white blood cells 600. No granulocytes present on smear. Platelets 50.0 thousand. Bleeding time 3 minutes. Clotting time 17 minutes.

March 19.—Hgb. 9.0 gm. Red blood cells 3.1 million, mean corpuscular hgb. 28.5 γγ, reticulocytes 0.0%, white blood cells 900. No granulocytes present on smear. Platelets 40,000.

The blood and bone marrow examinations carried out at the time of admission to hospital demonstrated a well-advanced state of pancytopenia, there being no evidence of megakaryocyte platelet production, the maturation of the erythrocytes being arrested at the erythroblast level, granulocytes showing poor maturation beyond the myelocyte stage. There was no apparent involvement of the lymphocyte or reticulum cell series.

Acute panhæmatocytopenia, associated with generalized hypoplasia of the bone marrow may be associated with a number of conditions, but drug intoxication, especially due to compounds having a benzene-ring structure are the most frequent and most serious offenders; benzol, sulfonamides, barbiturates, and pyramidon are well known in this respect. The exact mechanism of poisoning is not clear but individual idiosyncrasy or hypersensitivity appears to play some part in the sequence of events.

Progressive anæmia, granulocytopenia and thrombocytopenia are the presenting hæmatological manifestations. Infection of the mucous membranes often associated with ulceration, and hæmorrhagic phenomena, petechiæ, ecchymoses and mucous membrane hæmorrhages are the outstanding clinical manifestations.

The prognosis is very serious, exsanguination or hæmorrhage into vital organs being the usual cause of death. Spontaneous recovery of hæmopoiesis may avert death, especially in the post-infectious form and in sulfonamide poisoning. No known therapeutic measure will ensure stimulation of blood formation, although numerous drugs have received enthusiastic support. Pyridoxine, pentnucleotide, transfusions of fresh blood have had some success. The control of mucous membrane infection and attendant ulceration, formerly with sulfonamide poisoning) and more recently with penicillin, and the transfusion of large quantities of fresh blood may preserve life until spontaneous recovery of the bone marrow occurs. In certain cases the severity of hæmorrhage may be reduced by the administration of toluidine blue to combat heparin-like anticoagulant substances.

When the possibility of drug intoxication or idiosyncrasy was entertained in this case, she informed us that she had been taking dexedrine for nine to twelve months, to reduce her weight; and that the last dose had been about a month previously. Following her death a pharmacist, who had refilled a prescription found in her personal effects, produced the information that in the period February 2 to February 28, she had been issued 250 five mgm. dexedrine tablets, (1,250 mgm.); indirect information indicated that some prescriptions had been refilled at an additional pharmacist's establishment. Besides, dexedrine was available at times, in her occupation as nurse. Two prescriptions of 50 five mgm. tablets had been obtained *after* she was admitted to hospital and while the cause of her anæmia was under discussion.

Also discovered in her effects were three tablets of a capsule containing 1½ grains each of seconal, and amytal. There is no information relative to the quantity of barbiturate or duration of exposure, she may have endured.

Large doses of benzedrine have been consumed in other reported cases and the effects of benzedrine on blood-forming organs is apparently negligible. A patient with Parkinsonism consumed 160 mgm. a day (benzedrine) for three weeks without reported ill effects. Another patient consumed 15 to 30 mgm. a day (benzedrine) for nine years with no ill effects. Two cases of narcolepsy taking 70 mgm. a day (benzedrine) for two years and eight months and another taking the same dose for one year and eight months are reported without ill effect.

Benzedrine is taken in these large reputed doses chiefly for its stimulating effect. Dexedrine having a much less noticeable exhilarating effect, is less often (apparently) used for this purpose. It is possible therefore that large doses of dexedrine have an effect on blood-forming tissue that benzedrine has not demonstrated.

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I come from a state that raises corn and cotton and cockleburs and Democrats, and frothy eloquence neither convinces nor satisfies me. I am from Missouri. You have got to show me.—Willard Duncan Vandiver.