has required incision. The tip of the sac is caught in curved dressing forceps and is then passed through the external inguinal ring underneath the aponeurosis of the external oblique up to a point opposite the internal ring. At this point a small incision is made in the aponeurosis, and through it the sac is pulled out (not invaginated), then pulled taut, transfixed, ligated and the distal portion cut away. The stump slips back and the small incision in the aponeurosis is sewed up.

BALL'S METHOD

5. By Ball's method the sac is twisted into a dense cord, so as to throw it into a series of folds, thereby producing a prominence internally rather than an infundibuliform depression. To prevent untwisting, a ligature is tied as high as possible, and it is also transfixed below the ligature by a suture, the ends of which are passed through the abdominal parietes.

Fig. 3.—Pad of knotted sac ribbons after tying of each pair of ribbons, but not yet replaced into the internal abdominal ring.

BARKER'S METHOD

6. In Barker's method, the neck of the sac is isolated, ligatured and divided, the fundus being left in situ, while the suture used in tying the neck is employed as the first suture to close the deep part of the canal. By this means it is hoped that the neck of the sac will be displaced and, hence, more satisfactorily occluded—which I doubt very much.

AUTHOR'S METHOD

The method which I would present I term the "auto sac closure." It consists (when the sac is long enough) in the usual isolation of the sac from the cord and the surrounding structure well into the internal abdominal ring. When the sac is opened at its apex, it is freed from its contents and divided longitudinally into four, six or eight ribbons, which run nearly to the base of the sac. Then each opposite two ribbons are seized with hemostats and tied together with a single knot, each successive knot overlying the preceding one, thus forming a solid pad of the sac itself. To prevent slipping of the knots, a catgut ligature, if desired, can be placed around the entire knot and the ribbon

ends cut off. Then this pad of knotted sac ribbons is reduced into the internal ring, and one proceeds with the usual method of closure of the canal.

The advantages of this method are:

1. The solid bulwark of knotted sac which lies internal to the internal abdominal ring prevents the abdominal contents from again descending into the canal.
2. It does away with the infundibular shape of the sac, which is conducive to recurrence.
3. There is less likelihood of transfixing any of the sac contents by ligature.
4. The introduction of a foreign body like catgut into the peritoneal cavity, especially if it should not be sterile, is eliminated, though this point, to be sure, is of minor importance.

I have used this method in innumerable cases in my service at the Michael Reese Hospital, and have yet to see a recurrence of the hernia.

5 North Wabash Avenue.

EXFOLIATIVE DERMATITIS DUE TO ARSPHENAMIN

REPORT OF A FATAL CASE

JOSEPH R. LATHAM, M.D. (BELHAVEN, N. C.)
First Lieutenant, M. C., U. S. Army
CAMP JACKSON, COLUMBIA, S. C.

It has been recognized for some time that arsphenamin could, when given intravenously, cause arsenical poisoning. At the base hospital at Camp Jackson, there have occurred four cases within the past five months. The most typical of these—a fatal one—is here reported.

This fatal result followed the intravenous administration of 0.8 gm. of arsphenamin. The case, in its general features, bears the stamp of arsenical intoxication. The clinical diagnosis was confirmed by abundant laboratory data.

REPORT OF CASE

J. C., a private, aged 27, in a routine examination of his company gave a positive Wassermann reaction. He had at that time no symptom or sign of syphilis, and denied any venereal history. His physical condition was excellent. An injection of 0.4 gm. of arsphenamin was given intravenously. The reaction following this was so marked that he was kept a week in the hospital. He was then sent to duty, although there was present, at the time of his discharge from the hospital, a peculiar keratoid condition of his face and neck. From his second dose, received one week later, he experienced no apparent reaction. Ten days following the second injection, he was admitted to a medical ward at the base hospital. On admission, he had no complaint other than that the skin of the face and neck was hard and red and knobby to the touch, and was burning and tingling considerably. Even then, the process was showing a tendency to generalize. On the skin of the face and neck, in addition, there were to be found areas of superficial branny desquamation. Where the process was oldest, the skin color had faded to a dull red. In the superficial layers there was a marked amount of a dirty brown pigmentation. The eyes were the seat of a moderate conjunctivitis. The ocular media were normal. The fundus showed no sign of inflammation. The external ear showed, from the first, a distinct seborrheic eczema, which was apparently an independent process, so markedly did it differ from the other skin condition.

There was, at this time, no demonstrable evidence of polyneuritis. Reflexes were equal and well balanced, and there

were no sensory disturbances. It was only after the first week that he showed any laryngobronchial catarrh. At no time during his illness did he have abdominal pain or diarrhea. The urine content and output were within normal limits. Arsenic, while present in easily demonstrable amounts, was productive apparently of no renal irritation. The phenol-sulphonephthalein output at this time was 60 per cent. for the two hour period.

Four days after admission, the patient's skin began to exfoliate. The skin of the face and arms showed this most

being 0.0576 gm. per hundred c.c., whereas one week before it had been 0.056 gm.

As the exfoliation became more and more extreme, mental hebetude gradually deepened. Now, for the first time since his admission to the hospital three weeks before, there was some sensory disturbance, in that the whole left upper extremity became very insensitive to both pain and temperature. The patient suffered very little now, probably because his mental acuity was so much decreased. Fever was constant, and showed a distinct tendency to rise. Stupor deepened into coma, and despite active eliminative therapy, light convulsions began to occur, these being preceded and followed by localized muscular tremors. As a result of hot packs, the mental condition cleared somewhat, and the convulsions disappeared; the general condition, however, remained very poor.

It was only after three weeks in bed that the patient began to waste. This may be explained partly by the fact that he suffered at no time from either vomiting or diarrhea, and partly by the circumstance that up to within a day or two before his death, he partook willingly of large quantities of liquid nourishment. Toward the end, however, his mouth became so sore that feeding by means of the stomach tube had to be resorted to. When wasting did appear, it was very rapid. The body was now devoid of skin, so far as function was concerned. Bleeding fissures extended down and in places through the basement layer. The exposed deeper layers

had none of the pigmentation and induration of the original process.

The blood vessels showed, early in the course of the disease, evidence of severe trophic disturbance, in that the blood would extravasate from the capillaries at the slightest trauma. In the toes, this was apparently spontaneous, but the condition was especially marked in the fingers following needle puncture for blood counts. That this condition was due to blood vessel degeneration, rather than to any fault of the blood itself, is indicated by the fact that the coagulation time remained within normal limits throughout, never going above seven minutes.

The eyes, which had, as noted before, from the first shown

markedly. As the exfoliation became generalized, there appeared a moderate fever. The pigmentation, mentioned before, became extreme; especially was this true in the axillae and on the forearms. At the same time the skin of the face—at first dry and hard—now began to fissure and to weep a viscous fluid, intensely disagreeable in its odor.

Exfoliation became steadily deeper and more extensive, reaching its maximum about thirty days following the first dose of arsphenamin. The scales, which at first had been thin and parchment-like, now began to increase in size and in thickness and to slough away, leaving a basement layer which was soft and bleeding. With the scales disappeared the pigmentation. The mouth became very sore; especially was this true of the tongue, the papillae of which were enormously enlarged, the mucosa being covered by a thick fur.

Even at this time, four weeks after the first dose of arsphenamin, the only evidences of constitutional disturbance were the continuing moderate fever, and a pronounced and peculiar leukocytosis which will later be described in detail. The mind was absolutely clear. The skin inflammation, however, caused so much tingling and discomfort that sleep was impossible except as an effect of opiates.

Prior to the beginning of the deep exfoliation, the heart remained strong and the lungs clear. As the exfoliation progressed, toxic symptoms appeared and ran hand in hand with it. Blood pressure, which had up till then remained at 130 mm. or above, now sank to below 120 mm. The pulse rate mounted steadily, as did the fever. Râles appeared in the lungs; the voice became husky, and a slight, productive cough appeared. The picture was that of the so-called laryngobronchial catarrh, which is so often found in the literature referring to chronic arsenical poisoning.

Coincident with the foregoing, the urine output steadily declined, and there was, for the first time, a trace of albumin. During the seven-day period, the phenolsulphonephthalein excretion index sank from 60 per cent. to 37 per cent. for the two-hour period. The latter reflected accurately the progressive physical and mental deterioration of the patient. During the same seven-day period the nonprotein nitrogen of the blood remained low, at the conclusion of the period

a conjunctivitis, now began to show evidence of involvement of the deeper structures. The globes became sunken and flaccid, and there was decided atrophy of the orbital fat. The media became too cloudy to admit of fundus examination. When last seen, there was no optic atrophy. Cultures made of the conjunctival sac showed only the white staphylococcus.

On the thirty-fourth day after his first dose of arsphenamin, the patient lay in coma. The lung signs, which had been those of a moderate bronchitis, now began to be those of a bronchopneumonia. The heart sounds became almost inaudible except for the sharp snap of the second pulmonic. The

Fig. 1.—Exfoliative dermatitis: marked pigmentation.

Fig. 2.—Exfoliative dermatitis: more advanced stage, with increased pigmentation.

patient's condition progressively deteriorated and he died on the thirty-eighth day following his first dose of arsphenamin.

The laboratory data, especially the blood picture from day to day, constitute the most interesting feature of the case.

TRACES OF ARSENIC IN URINE

The urine showed traces of arsenic as late as the thirty-fourth day. The urine remaining in the bladder after the patient's death also showed presence of arsenic. It was only toward the end that there was evidence of renal irritation. Considering the comparatively small dose of arsphenamin administered, it is rather remarkable that arsenic appeared so long and so consistently. Severi¹ found in repeated experiments that, with a single dose of toxic quantity administered by mouth, the metal was completely eliminated within five days, provided the kidneys were previously normal. Witthaus² considers this to be the usual result. Almqvist and Welander,³ however, found weighable quantities in the urine as late as sixty days after the stoppage of the poison. The latter condition is more apt to occur when repeated small doses have been given. Yet Wood⁴ found arsenic in the urine as late as ninety-three days after a single toxic dose. In the case under discussion, the slight renal irritation which developed toward the end cannot with justice be ascribed to arsenic, when one remembers the extreme impairment of skin function. Certainly, if it had been due to arsenic it might have been expected to exist from the first; but such was not the case.

The total arsenic equivalent of the drug given was a little over 2½ grains. As might have been expected from the smallness of the dose, it was only after large quantities of the exfoliating skin were subjected to test that arsenic was found in it.

The blood showed consistently a high polymorphonuclear leukocytosis. This persisted up to within a day before the patient's death, being closely related in degree to the height of the fever. As exfoliation progressed and skin function began correspondingly to suffer, there appeared a remarkable eosinophilia. The latter condition is found in many skin diseases, notably in pemphigus and dermatitis herpetiformis, psoriasis and eczema. In all of these, however, the percentage runs usually between 5 per cent. and 15 per cent., being seldom above. At the crest of this patient's illness, the eosin cells formed over 40 per cent. of the total leukocytes. At one time there was transient improvement in the patient's condition. It was then that the eosinophils were most numerous. As the man's resistance lowered, they disappeared from the peripheral blood. During the last three days of his life, the percentage sank to zero. The accompanying chart shows graphically the relation between the fever leukocytosis and eosinophilia.

Fig. 3.—Relation between fever, leukocytosis and eosinophilia: Black line, temperature; upper broken line, leukocytes; lower broken line, percentage of eosinophils in the peripheral blood.

NECROPSY FINDINGS

There was everywhere evidence of atrophy of the subcutaneous fat. The tissues themselves were very dry.

The lungs showed an extensive passive congestion, and, at the right base, was an area of bronchopneumonia.

The heart and liver were soft and pulpy to the touch, and in many places showed extensive areas of fatty degeneration. The spleen was large, very soft and pulpy.

The kidneys were somewhat congested, but did not show either macroscopically or microscopically any definite nephritis. The suprarenals were decreased in size, and the medulla was replaced by a waxy white substance, which no longer took the chromic stain.

The mucosa of the stomach and the intestine was covered by an adherent mucus; chemically, it showed the presence of arsenic. It must be remembered that the bowel is a very important pathway for the excretion of the poison from the body.

Analysis of the liver, brain and skin, as well as of the pericardial fluid, revealed the presence of arsenic.

SUMMARY

1. A fatality followed a therapeutic dose of arsphenamin, the equivalent of less than 3 grains of metallic arsenic.

2. Diarrhea and vomiting were absent during all stages of the intoxication.

3. Nephritis was not a marked feature at any time, and appeared only at the end.

4. There was apparently a decided affinity of the poison for the skin or for the trophic nerves supplying it. From first to last all the toxic symptoms may logically be ascribed to impairment of skin function.

5. Arsenic was persistently present in the urine. This was remarkable in the absence of accompanying renal inflammation.

6. There was a high leukocytosis and eosinophilia, the latter related closely to the patient's resistance. The height of the leukocytosis followed that of the fever.

7. Arsenic was found at necropsy in every tissue in which it was sought.

Tuberculosis in Brazil.—According to the statements of Dr. O. Clark in a recent pamphlet on the need of organizing a Service of School Hygiene in Rio de Janeiro, tuberculosis causes an average of 4,000 deaths every year in the urban section of that capital, in spite of the fact that the climate is most mild, the air most pure, that there is plenty of sunshine; no congestion of population, no factories, and no subways; that there is no poverty, nor any alcohol problem; that the temperature changes but little all the year round, and that there are plenty of gardens and parks and miles of beaches. It is estimated that among all the large cities of the world, Rio de Janeiro has proportionally the largest tuberculosis mortality. Taking as a basis the usual proportion between deaths and cases, the above figures mean that there are at Rio de Janeiro all the time 40,000 cases of open tuberculosis.

1. Severi: *Ann. di chim. et di farm.* **18**: 33, 1893.

2. Witthaus: *Manual of Toxicology*, p. 547.

3. Almqvist and Welander, quoted by Heffter: *Ergebn. d. Physiol.* **2**: 115-116, 1903.

4. Wood: *Boston M. & S. J.* **118**: 414, 1893.