blankets may be the replacement of woollen blankets by blankets made of some material which can withstand boiling and other rigorous laundry procedures.

When considering laundry hygiene, attention should not be directed solely to the process of washing. It is clearly important to see that freshly washed articles are not recontaminated.

In sorting dirty articles of clothing in a laundry it is usual for air contamination to reach high levels. Again, large laundries have powerful appliances which by design or accident draw in large volumes of air and then expel dust or aerosols into the environment. Obviously it is important that this contaminated air should not have access to regions of the laundry where freshly laundered articles are exposed.

By very simple methods—exposing plates to the air during normal working conditions, producing artificial contamination with aerosols of an easily recognizable organism, and studying the prevailing air currents with smoke—it is possible to detect these sources of contamination.

We cannot generalize on the steps which should be taken to remove such sources of contamination, since these will largely depend on the layout of individual laundries. However, it seems likely that quite simple alterations in ventilation or the resiting of machinery or sorting-rooms and storerooms will usually improve the hygiene of laundries where these defects still exist.

#### Summary

Hospital blankets are often contaminated with pathogenic organisms which are not removed by accepted methods of laundering.

A simple method of laundering blankets is described which, though not ideal, appears to give better results than many of the methods in current use.

In planning methods of laundry procedure it is important to guard against air-borne re-contamination of freshly laundered articles.

We acknowledge the help received in the course of this investigation from Mr. H. Besser, the laundry manager at Guy's Hospital.

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# **Medical Memorandum**

# Agranulocytosis with Thrombocytopenia following Chlorothiazide Therapy

Chlorothiazide (6-chloro-7-sulphamyl-1,2.4-benzothiadiazine-1.1-dioxide), an orally effective non-mercurial diuretic agent, has been employed extensively both in hospital and in general practice during the past year. This drug is believed to be free of toxicity, and the only side-effects observed occasionally have been mild gastro-intestinal disturbances. One case with a maculopapular rash and pruritus has been noted by C. K. V. van Dommalen (1958, personal communication) following the use of chlorothiazide, but no other toxic manifestations have been published. It is therefore worth recording a case of fatal bone-marrow dysplasia following treatment with chlorothiazide.

# CASE REPORT

A man aged 70 was admitted to hospital with a history of having collapsed in the street four weeks previously. He had been treated with digoxin and chlorothiazide at home for acute congestive cardiac failure due to hypertensive heart disease. The total amount of chlorothiazide administered Weisfuse, L., Spear, P. W., and Sass, M. (1954). Amer. J. Med., 17, 414.

was 25 g. over a period of 13 days. He had received no other drugs, and there was no history of occupational exposure to chemicals or radiation. He was transferred to hospital after four weeks' treatment because of the development of delirium and incontinence over the previous two days. On admission he was found to be mildly confused, with a generalized maculopapular and purpuric rash over the face, trunk, and extremities. A pyrexia of 100.4° F. (38° C.), mild congestive cardiac failure, left hydrothorax, and a strongly positive Hess test were noted. There was no enlargement of lymph nodes or spleen. Tendon jerks in the lower limbs were absent and proprioception and deep sensation lost. Next day his condition deteriorated, he became mildly jaundiced, and there was further extension of the rash. He died six days after admission.

Investigations.—On his admission there was a normocytic normochromic anaemia with haemoglobin of 10.1 g. per 100 ml., and 3,000 white cells per c.mm. (lymphocytes 97%, monocytes 3%). Examination of two blood films revealed only a single eosinophil polymorph, and, although a few of the lymphocytes were atypical, no primitive forms were seen. The Paul-Bunnell test and W.R. and Kahn tests were negative. Two days later the white cells numbered 900 per c.mm. (neutrophil polymorphs 14%, lymphocytes 73%, monocytes 10%, atypical cells 3%). The neutrophil polymorphs stained histochemically showed a high alkaline phosphatase content. Platelets were 76,000 per c.mm., and E.S.R. 44 mm. in one hour (Westergren). Blood urea was 140 mg. per 100 ml., serum bilirubin 1.8 mg. per 100 ml., and thymol turbidity 4 units.

The direct Coombs test and Schumm's test were negative. Electrophoresis of the plasma proteins showed an increase in  $\alpha_2$  globulin. Serum electrolytes were: sodium 133 mEq/1., chloride 94 mEq/l., and potassium 3.9 mEq/l. Examination of the pleural fluid showed a simple transudate containing fresh blood.

The cause of the antecedent heart failure was confirmed at the post-mortem examination as being due to dilatation of a hypertrophic left ventricle. There was extensive purpura in the skin, the white matter of the brain, and the intestine. The bone marrow was replaced macroscopically by fibro-gelatinous tissue, and histological examination of that obtained from the sternum, lumbar vertebral body, and shaft of the femur showed it to be very hypoplastic. Myelocytes and some metamyelocytes were seen, but mature granulocytes were scanty. Erythropoiesis was normal or only slightly depressed. There was a relative increase of plasma and reticulum cells and a great increase in free iron. The number of megakaryocytes present in the marrow had also strikingly increased.

## COMMENT

It is impossible to avoid the conclusion that the marrow disturbance in this case was due to an idiosyncratic reaction to chlorothiazide. The dosage of chlorothiazide was high but not excessive. The maculopapular element of the rash had the familiar characteristics of a drug eruption, and the purpura was linked with a severe thrombocytopenia. The combination of a low platelet count in the peripheral blood with large numbers of megakaryocytes in the marrow is the antithetical phenomenon that has been noted by Weisfuse et al. (1954) in the case of quinidine. The agranulocytosis, which was almost complete at one stage, is a frequent finding in this type of reaction, and the patient had not been exposed to any other potentially toxic material.

We thank Dr. H. E. S. Pearson who was in charge of this case, and Dr. P. Darby for the histological reports.

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