vation in total fatty acids, triglycerides, cholesterol, lipid phosphorus, and total lipids. Paper electrophoresis revealed a concomitant increase in β-lipoproteins. Lipid mobilizer (LM) also increased strikingly, as was determined indirectly by testing plasma aliquots for inhibition of heparin activated clearing lipase. On the basis of these and related studies, is is suggested that Triton hyperlipemia is produced, at least in part, through sustained release of increased amounts of LM which mobilize triglycerides to the portal circulation. Furthermore, Triton modifies metabolic activity of the liver so that post-hepatic hyperphospholipidemia and hypercholesteremia ensue.

1. Seifter, J., Baeder, D. H., Proc. Soc. Exp. Biol. & Med., 1954, v86, 701.
2. ———, ibid., 1957, v95, 318.
3. ———, ibid., 1957, v95, 469.
4. Lipovetskii, B. M., Pathol. Fiziol. i Exsperim. Terapiya, 1965, v9, 49.
5. Kadas Von, L., Nagy, D., Endokrinologie, 1965, v48, 1.
6. Zarafonetis, C. J. D., Seifter, J., Baeder, D. H., Kalas, J. P., Am. J. Med. Sci., 1959, v237, 418.
7. ———, J. Lab. & Clin. Med., 1957, v50, 965.
8. ———, Clin. Res., 1958, v6, 265.
9. Seifter, J., Baeder, D. H., Zarafonetis, C. J. D., Kalas, J. P., Ann. N. Y. Acad. Sci., 1959, v72, 1031.
10. Zarafonetis, C. J. D., Bartlett, R. H., Brody, G. L., J. Am. Med. Assn., 1965, v191, 235.
11. Kellner, A., Correll, J. W., Ladd, A. T., J. Exp. Med., 1951, v93, 373.
12. Hirsch, R. L., Kelner, A., ibid., 1956, v104, 1.
13. Friedman, M., Byers, S. O., ibid., 1953, v97, 117.
14. Scannu, A., Page, I. H., J. Clin. Invest., 1962, v41, 495.
15. Seifter, J., Baeder, D. H., Proc. Soc. Exp. Biol. & Med., 1957, v95, 747.
16. Zarafonetis, C. J. D., Seifter, J., Baeder, D. H., Kalas, J. P., Chang, W. Y. M., Am. J. Med. Sci., 1959, v237, 771.

Received February 23, 1967. P.S.E.B.M., 1967, v125.

A Simple and Inexpensive Device for Restraining Rodents.* (32083)

PASQUALE PELLECCHIA (Introduced by V. H. Auerbach)
St. Christopher's Hospital for Children, Temple University School of Medicine, Philadelphia, Pa.

In many experimental procedures involving rats or mice, regularly timed blood specimens must be obtained. The use of the tail veins as a source of this blood necessitates incapacitating the animal in either elaborate restraining devices or wrapping them in toweling. Both techniques are often traumatic to both animal and experimenter. A simple device is described which alleviates this problem.

An ordinary low-density narrow-mouth polyethylene bottle is cut with a razor blade as in Fig. 1. Enough of the bottle is left intact to allow the rat to brace his hind legs. Also an injection port may be cut in the side if necessary. The animal needs very little inducement to enter the bottle and frequently will walk in to it of its own accord if the bottle is merely placed before it. Once the rat is in with its tail still out, a wide piece of adhesive tape is used to close the entrance. The size of the bottle should be commensurate with the size of the rat; an 8 oz bottle will conveniently hold a 200 g rat.

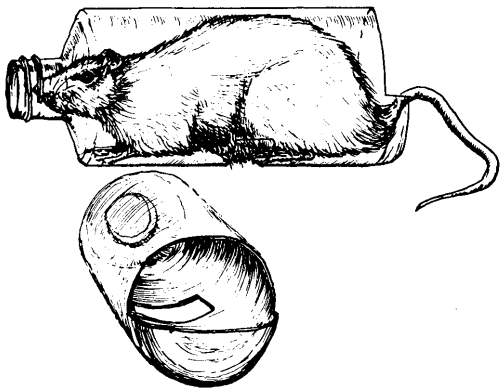

FIG. 1.

* Supported in part by Grant AM-06469 from Nat. Inst. Health, USPHS.

Once within the bottle, the rat cannot turn around, sits comfortably with his nose in the neck of the bottle thus breathing freely, and is handled easily without being agitated or aroused. Intraperitoneal injections can be given easily through a port cut in the side of the bottle. If the experiment is terminal, the bottle cap, containing a piece of cotton saturated with chloroform, may be screwed on, to kill the animal. The device is sufficiently inexpensive so that both rat and bottle may be disposed.

Received February 24, 1967. P.S.E.B.M., 1967, v125.

Granulocytopoiesis in Germfree Mice.* (32084)

D. R. BOGGS,† P. A. CHERVENICK, J. C. MARSH, H. I. PILGRIM, G. E. CARTWRIGHT, AND M. M. WINTROBE
Departments of Medicine and Surgery, University of Utah College of Medicine, Salt Lake City

The only well delineated function of the neutrophilic granulocytic leukocyte is that of a phagocyte serving to prevent and combat infection. Human or canine subjects, in the absence of clinically apparent infection, produce enough neutrophils to replace the total number found in the blood an average of 3 times each day(1,2). The quantitative aspects of the fate of this massive number of neutrophils leaving the blood each day are unknown but neutrophils are routinely demonstrable in the bronchial secretions, in the gut and in the urine(3). Since bacteria form part of the normal ecology of the body orifices and of the gut, the outflow of neutrophils to these areas may play a role in the prevention of invasion by the "normal bacterial flora". The "normal flora" appear to be the source of many infections occurring in neutropenic patients(4).

This study was designed to determine if the absence of micro-organisms in the environment would result in a reduced rate of production of neutrophils. The total number of neutrophils and neutrophil precursors in the marrow of germfree and conventional mice was compared. A similar comparison was made with respect to the mitotic rate of neutrophil precursors and the number of neutrophils in blood. The ability of the neutrophil system of germfree mice to respond in a normal fashion was tested by determining the effect of endotoxin injection upon the rate of release of neutrophils from the marrow.

Materials and methods. Mice were males of the C3H/Pi strain, 7-10 weeks of age at the time of removal from the germfree environment. Germfree mice weighed an average of 20 g as compared to 25 g for control conventional mice of the same age, maintained on the same diet.

Germfree mice were reared and maintained in aluminum isolators(5), 10 mice per isolator and conventional controls were placed in isolators at 3 to 5 weeks of age. Both groups were given an identical diet which was autoclaved for 50 minutes and consisted of ground liver (750 g), corn oil (190 g), instant powdered milk (85 g), wheat germ (100 g), red beans (830 g), iodized salt (18 g), calcium propionate (4 g), water (1,550 ml), thiamin (30 mg), riboflavin (30 mg), pyridoxine hydrochloride (15 mg), nicotinamide (150 mg), pantothenic acid (75 mg), vitamin B_{12} (3 μg), ascorbic acid (450 mg) and desiccated liver (0.9 g). The food for the conventional animals was kept refrigerated to prevent spoilage, and was fed to the animals as needed. The food for the germfree mice was kept in the isolator at room temperature.

Mice removed from the germfree environment were then maintained 5 to 10 per cage in close proximity to a colony of normal mice. During this time they were given food

* This investigation was supported by a research grant (AM-04489) and a graduate training grant (2A-5098) from Nat. Inst. of Arthritis & Metab. Dis. and by a research grant (CA-05771) from Nat. Cancer Inst. Nat. Inst. Health, Bethesda, Md.

† Faculty Research Associate of Am. Cancer Soc.