The technique of counting eosinophils used in our laboratory and the statistical treatment of the data obtained have been described in detail elsewhere (*2–5*). In the investigation reported here each absolute eosinophil count was obtained by counting cells taken up into 2 standard leucocyte pipettes and delivered into 4 double Levy counting chambers. The data obtained were highly significant for each of the bloods studied, as well as for difference of means, P being less than 0.001.

TABLE 1

EFFECT OF INCUBATING DEFIBRINATED HUMAN BLOOD*

Statistical data	Fall of absolute eosinophil counts in defibrinated human blood after 4 hr incubation (%)		
	Saline	Cortisone acetate	Heparin plus cortisone acetate
Mean	15.2	44.9	11.4
S. E.	4.4	3.6	2.7
Fiducial limits ($P = 0.05$)	5.1–25.3	37.2–52.6	5.5–17.3
Mean $\pm \sigma$	1.9–28.5	30.8–59.0	2.1–20.7

* Blood from 15 patients with eosinophilia (490–3000 cells/mm³) was incubated for 4 hr at 37.5° C. Percentage fall is in absolute eosinophil counts from zero time, when saline, cortisone acetate, or heparin, followed by cortisone acetate, was added to the blood.

Thrombosis is a common complication of therapy with ACTH and cortisone acetate (*6,7*), and it also develops after surgical procedures, when adrenal hyperactivity, eosinopenia (*8*), and reduced blood heparin tolerances (*9*) are found. Therefore, on the basis of our findings, it is tempting to speculate that in man adrenocortical hormones interact with heparin as they do with other polysaccharides, such as joint hyaluronic acid (*10*). However, the concentrations of cortisone and heparin used in the *in vitro* investigations reported here were very much larger than obtain in clinical practice.

The morphological changes in the eosinophils which appear after *in vitro* incubation with cortisone, and the effects of combining heparin and adrenal steroid therapy in man, will be reported elsewhere.

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An Implantor for Forming and Accurate Placing of Small Pellets

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Previously reported techniques for implanting solids into animals (*1–3*) were for large, preformed pellets. In the course of experiments requiring accurate placing of a known small amount of compact, sterile solid, an instrument was designed and constructed which fulfilled these requirements.

The device (Fig. 1), made entirely of stainless steel,

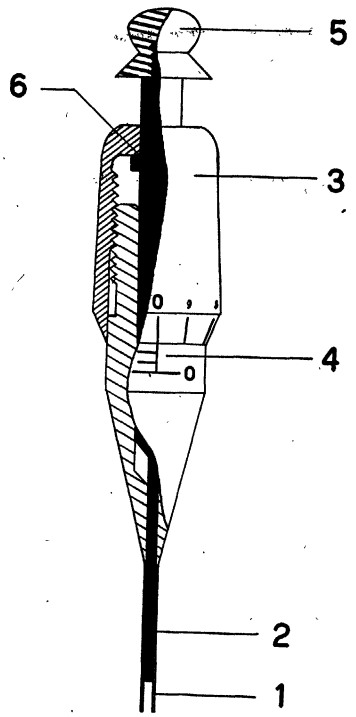

FIG. 1.

consists of a thin-walled tube (1), with a close-fitting plunger (2). When preparing a pellet of material for implantation, the plunger is set to the desired length by turning the body (3) to the setting on scale (4). The plunger head (5) is withdrawn to collar check (6). The body of the device is held firmly, and the tube tamped into a small dish containing the substance (fine crystals or powder) to be used for study.

A sharp needle approximately the size of the pellet is used to make an entry for the tip of the instrument, after which the solid which has been compacted in the end of the tube is ejected *in situ*. Sterile precautions in preparation of the pellets consisted of using sterilized glassware and implantor, with care to avoid contamination, and were sufficient to avoid infecting experimental animals.

The number of taps required for compacting a

pellet of satisfactory hardness depends on the length of the pellet, the diameter of the tube, and the material.

Calibration data are presented for two implantors of each size (Table 1); pellets made of cholesterol were weighed individually on a microbalance.

TABLE 1

Implantor No.	Implantor Diam (mm)	Pellet length (mm)	Tampings (No.)	Sample size (*n*)	Mean wt (mg)	S.D.
1	0.85	1.0	10	10	0.534	± 0.03
		2.0	20	30	0.984	± .05
2	0.835	1.0	10	10	0.520	± .02
		2.0	20	20	0.877	± .06
1	2.505	1.0	10	10	4.896	± .4
		2.0	20	10	8.345	± .26
		3.0	25	10	12.585	± .5
2	2.48	1.0	10	10	4.483	± .4
		2.0	20	10	8.705	± .5
		3.0	25	10	12.117	± 0.4

By tapering the tube end to a cutting edge, the device can be used as a punch and implantor for thin slices of sterile tissue.

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Bactericidal Effects of Propylene and Triethylene Glycol Vapors on Airborne *Escherichia coli*[1]

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In 1941 it was initially reported from this laboratory that the vapors of certain glycols had marked bactericidal effects upon airborne microorganisms (*1, 2*), and these reports were shortly followed by others describing the phenomenon in more detail (*3–9*). In these early reports it was stated that the killing of airborne bacteria was most rapid in the presence of concentrations of either propylene or triethylene glycol vapor that most nearly approached the saturation levels for these substances in the air and that at concentrations below this range the bactericidal effects were less apparent. Furthermore, it was noted that the lethal action of glycol vapors was less marked at relative humidities above 70%. A wide variety of microorganisms, dispersed into the air from broth cultures, was found to be susceptible to the germicidal activity of the glycol vapors. Included among these were *Staphylococcus albus* and *aureus*; *Streptococcus hemolyticus,* Groups A and C; *Str. viridans*; Types I and III pneumococci; *Bacillus subtilis*; *Hemophilus influenzae* and *pertussis*; Friedlander's bacillus; and *Escherichia coli.* Subsequent investigations reported from this laboratory dealing with the mechanism (*10*), rate of bactericidal action of glycol vapors (*11*), and the effects of desiccation (*12*) have utilized Group C hemolytic streptococci, pneumococci, and staphylococci because these organisms were considered to be more typically representative of the respiratory pathogens. From this work has arisen a better understanding of the paramount significance of relative humidity and glycol vapor concentration in determining the bactericidal efficacy of these compounds in the air.

Independent investigators have reported data confirming the susceptibility of some of the previously listed organisms to the glycol vapors (*13*) and, in addition, have found that *Serratia marcescens* and *Salmonella pullorum* (*14*), *Penicillium notatum* (*15*), and tubercle bacilli (*16*) were killed when atomized into saturated atmospheres of these substances. In no instances were airborne vegetative microorganisms, atomized from broth cultures, found to be resistant to the germicidal action of the glycol vapors.

A recent report by Nagy and Mouromseff (*17*) has questioned the validity of these data and thrown doubt upon the lethal effect of propylene and triethylene glycol vapor upon airborne *E. coli.* Using this organism atomized from broth cultures into air containing unmeasured concentrations of either propylene or triethylene glycol, they were unable to demonstrate any bactericidal activity. From their data these authors concluded that the primary effect of the glycol vapors was to increase the sedimentation velocity of the airborne bacterial particles by condensing upon them and hence mechanically removing them from the air. Furthermore, they suggested that the organisms in the sedimented bacterial particles were not necessarily killed but, following evaporation of the condensed glycol, might be redispersed into the air and again be capable of initiating infection. Because their conclusions are so widely at variance with those of other workers in the field, as well as with our own, we considered it essential to report our data, which we believe demonstrate that, under the prescribed physical conditions, propylene and triethylene glycol vapors in adequate concentrations in the air are bactericidal for *E. coli* atomized from liquid cultures, as they are for other microorganisms.

The experiments included in this report were done in the 640-cu ft chamber which has been previously described in detail (*18*). A 17-in. fan, rotating at 500 rpm, was centrally located on the floor and the air stream directed against the ceiling, thus providing adequate turbulence for uniform mixing of the chamber air. Temperature and humidity were pre-

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[2] With the technical assistance of Mabel Smith.