none was observed in parietal cells. Some epithelial cells in colon and stomach incorporate thymidine but do not proliferate rapidly, and are retained in the mucosa for prolonged periods. These findings correspond closely to kinetics of proliferation previously observed in the mouse and rat and provide a basis for study of disease states in man.

The Effects of Alcohol in Experimental Infections in Mice. DONALD B. LOURIA, New York, N. Y. (introduced by Robert F. Watson).

Certain bacterial infections occur with inordinate frequency in alcoholics. In the present studies, mice were given 0.5 ml of ethyl alcohol subcutaneously one-half hour prior to infection. After intravenous inoculation with staphylococci, 18 of 81 controls (22%) died in 14 days, as did 93 of 142 (65%) which became ataxic but not stuporous after alcohol, and 88 of 112 (81%) of those developing coma. Kidney staphylococcal populations were similar in all groups at 2, 4, and 6 hours but were significantly higher in alcohol-treated comatose or ataxic mice 24 hours after infection. Peritoneal clearance of *Escherichia coli* and staphylococci was normal in alcohol-treated mice if small numbers of microorganisms were introduced intraperitoneally, but with larger inocula, clearance was markedly reduced. Populations were similar in all groups during the first hour, but 2 to 6 hours after infection the microbial census fell over 95% in 74% of controls, in 35% of ataxic mice, and in 13% of those that were stuporous. Quantitative measurement of the polymorphonuclear response showed that 2 hours after infection only small numbers of polymorphonuclear leukocytes entered the peritoneal cavity of alcohol-treated mice and 4 hours after infection, polymorphonuclear counts averaged 58% less in ataxic animals, and 88% less in comatose mice, than in controls. Since results were similar in ataxic and comatose animals, coma, hypotension, and shock cannot be implicated in these experiments. If peritoneal inflammation was produced by injection of starch-aleuronat prior to infection, alcohol administration did not alter phagocytosis or intracellular killing significantly, suggesting that the enhancement of infection observed in these studies was related primarily to delay in polymorphonuclear mobilization.

The Use of Synchronized Direct-current Countershock in the Treatment of Cardiac Arrhythmias. BERNARD LOWN, RAGHAVAN AMARASINGHAM, JOSE NEUMAN AND BAROUH BERKOVITZ, Boston, Mass. (introduced by Samuel A. Levine).

Some cardiac arrhythmias are refractory to all forms of treatment. In principle, external electric countershock that can induce complete depolarization of the heart and permit resumption of sinus activity would be an ideal form of treatment if it were safe. Experiments were conducted to determine what form of electrical stimulation would be most safe and effective. Nineteen dogs in sinus rhythm received a total of 438 alternating-current countershocks (AC) across the closed chest, with voltages effective for ventricular defibrillation and with lower levels. There was a high incidence of diverse arrhythmias including 78 episodes of ventricular fibrillation (18%). Seven animals died during the first week after countershock. AC countershock was therefore considered unsafe for elective use in patients. A direct-current defibrillator (DC) delivering a brief monophasic discharge was developed. In 30 dogs receiving 858 DC countershocks there was a 1.0% incidence of ventricular fibrillation and no deaths. This technique was consistently effective in defibrillating the heart even when AC failed. In the use of DC, ventricular fibrillation occurred only when the discharge fell during a predictable brief vulnerable period of the ventricular cycle. To prevent this arrhythmia entirely, a synchronizer was developed which permitted the shock to be delivered outside this zone. Thus in 2,600 synchronized shocks, ventricular fibrillation never resulted unless the shock was triggered within the vulnerable period. Single synchronized DC shocks were applied across the closed chest in two patients with ventricular tachycardia resistant to all therapy and four patients with long-standing atrial fibrillation. Reversion was instantly achieved in all except one with atrial fibrillation. There were no complications. This extensive experimental and limited clinical experience suggests that a new simple and effective method of controlling some serious arrhythmias may be available.

Hyponatremia in Acute Porphyria Probably Due to Inappropriate Secretion of Antidiuretic Hormone. GEORGE D. LUDWIG* AND MARTIN GOLDBERG, Philadelphia, Pa.

In the past, it has been noted that hyponatremia frequently accompanies acute attacks of porphyria, but the cause has never been adequately explained. Eight patients displaying the syndrome of hyponatremia, serum hypo-osmolality, natriuresis, and urine hypertonicity were studied. Serum sodium and osmolality were as low as 100 mEq per L and 220 mOsm per kg, respectively, in some cases. Edema, azotemia, and dehydration were absent. The syndrome probably results from inappropriate secretion of ADH. Adrenal insufficiency, chronic renal disease, and "true" sodium depletion were excluded. Central neurological involvement, such as coma, convulsions, or acute psychosis, was correlated with the most severe changes in serum sodium and osmolality. In selected patients, restriction of fluids to 800 to 1,000 ml per day resulted in dramatic clinical improvement with reversal of serum sodium and osmolality toward normal. In one of these patients, increasing fluid intake to 3,000 ml per day produced a sharp fall in serum sodium and osmolality that was accompanied by a convulsive seizure. With subsidence of acute attacks in these patients, the initial greatly increased excretion of porphyrins and porphyrin-precursors decreased as normal values for serum sodium and osmolality were achieved, and the urine became hypotonic. Oral water loading then failed to reproduce the hyponatremic syndrome. This syn-