tered magnesium is excreted by the kidney, one may conclude that the low urinary excretion of *orally* administered $Mg^{28}$ is due to poor gastrointestinal absorption of this material. The very slight rise in serum magnesium concentration following oral dose of magnesium also suggests poor gastrointestinal absorption, as does the recovery of 83 and 88% of total administered dose in the feces in the 2 medical students (#11 and 12).

*Summary.* A tracer dose of $Mg^{28}$ (10 to 25 $\mu$c contained in 3 to 10 meq of magnesium) was administered orally to 26 subjects, and serial specimens of blood, urine and feces were assayed for radioactivity. Urinary excretion within 72 hours accounted for less than 10% of total dose of $Mg^{28}$; the maximal excretion (6.3%) occurred during the first 24-hour period. Maximal concentration of radioactivity in plasma occurred at 4 hours, but the actual increase in serum magnesium was negligible. Fecal excretion within 120 hours accounted for 60 to 88% of administered dose. Low renal excretion of magnesium is believed due to poor gastrointestinal absorption of this material.

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## Effect of Experimentally Induced Fever on Alimentary Lipemia. (23930)

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The discovery of Hahn(1) that intravenous injection of heparin rapidly clears the turbidity of alimentary lipemia has been amply confirmed. Clearing occurs similarly when the serum of heparinized animals is mixed with lipemic serum and with fat emulsions *in vitro,* but not when heparin is added directly(2). This observation led to the conclusion that heparin does not act as such, but produces a clearing factor in the body. Release of heparin by shock and by histamine injection has a similar clearing effect(3).

The fortuitous observation that plasma of a patient with familial hyperlipemia cleared repeatedly during several bouts of fever caused by idiopathic pericarditis suggested that a rise in body temperature may similarly release the clearing factor. It was, therefore, attempted to investigate the possible effect of experimentally induced fever on alimentary lipemia in the dog.

*Material and method.* A) *In vivo studies.* 105 mongrel dogs of either sex, weighing 10 to 15 kg were fasted for 48 hours. The animals were then fed 10 egg yolks either alone or added to a mixture of small pieces of cooked meat and oat meal. To prevent regurgitation each animal received 50-60 mg of Thorazine intramuscularly immediately after eating. The animals were divided into 6 groups: 1) 25 dogs serving as controls were fed the egg yolk meal as indicated above. 2) 30 dogs received 0.5-0.7 cc of typhoid and paratyphoid vaccine (Parke, Davis and Co.) subcutaneously. 3) 11 animals were given 100 $\gamma$ of Piromen (Travenol Laboratory, Inc.)† intravenously 1½ hours after test meal. 4) 25 dogs were injected with 25 mg of heparin intravenously after similar time interval. 5) 8 dogs received simultaneously injections of 0.5-0.7 cc of typhoid and paratyphoid vaccine subcutaneously and 25 mg

* Deceased.

† Piromen is non-protein, bacterial component, containing mixture of nucleic acids and polysaccharide complex.

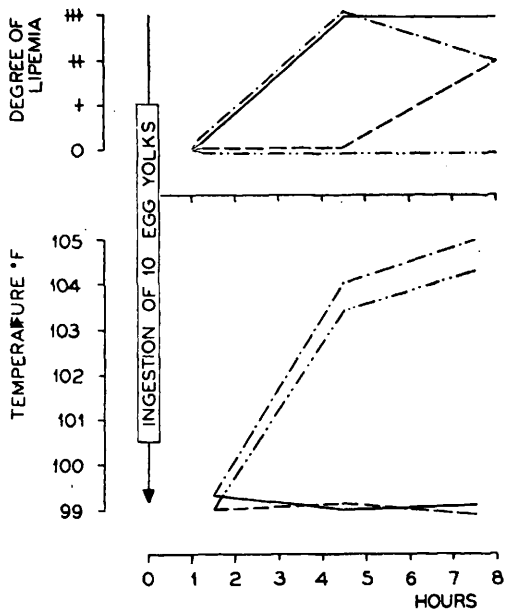


FIG. 1. Effect of vaccine alone or simultaneously with protamine sulfate, and heparin on body temperature and alimentary lipemia of 3 dogs after ingestion of 10 egg yolks. Control animals received no medication after the test meal.
— • • — • • 0.7 cc typhoid and paratyphoid vaccine; — • — • 0.7 cc typhoid and paratyphoid vaccine + 25 mg protamine sulfate; — — — 25 mg heparin; ——— Control.

protamine sulfate intravenously 1½ hours after test meal. 6) 6 dogs received a single subcutaneous injection of 0.7 cc of typhoid and paratyphoid vaccine daily for 8 days. All animals of this group received a test meal on first and last day of experiment 1½ hours prior to injection of the vaccine. Rectal temperature of all dogs was recorded at one-half hour intervals for 6-8 hours after ingestion of egg yolk meal. Blood samples were withdrawn and immediately centrifuged at similar periods. After separation from the clot the turbidity of the sera was estimated and graded from 0-3 +. B) *In vitro studies.* To investigate *in vitro* the effect of temperatures equivalent to those *in vivo* on lipemia, sera of 8 dogs obtained from 1½ to 4 hours after the egg yolk meal were placed in water bath of 105° up to 24 hours.

*Results.* A) *In vivo experiments.* 25 dogs receiving vaccine and 8 receiving Piromen developed a marked rise of temperature within 1½ hours varying from 103 to 107°F which remained significantly elevated to 7 hours after injection. None of the animals of the other 5 groups had increase of temperature.

All dogs developed marked lipemia 1½ hours after ingestion of test meal. Sera of control animals remained turbid throughout. However, lactescence had disappeared when blood was drawn 24 hours later. Sera of those dogs in whom administration of the vaccine or of Piromen produced fever of 103°F or more cleared rapidly and remained so for the following 8 hours (Fig. 1), while no change of lipemia occurred in those animals with temperature below that level. Administration of heparin induced immediate clearing of the alimentary lipemia. After 3 hours, however, the sera became turbid again and remained so until end of experiment (Fig. 1). Administration of protamine sulfate to vaccine-treated dogs prevented clearing of heavy lipemia despite a marked temperature rise. Daily administration of typhoid and paratyphoid vaccine for one week failed to induce significant temperature elevations from 3rd day on. Complete clearing of the lipemic sera occurred after test meal on first day when the temperatures rose above 104°F, while it was not observed on repeating the experiment on 7th day.

B) *In vitro experiments.* Turbidity of the sera obtained from dogs fed egg yolk meal, failed to clear when kept in water bath at 105°F up to 12 consecutive hours.

*Discussion.* Our experiments demonstrate that administration of typhoid and paratyphoid vaccine as well as Piromen caused rapid clearing of alimentary lipemia in dogs. This phenomenon appears to be unspecific and apparently associated with marked rise in temperature since no clearing of the turbidity occurred in animals which remained afebrile or whose temperature elevation never exceeded 103°F. Prevention of clearing of the lactescent serum by injection of protamine sulfate simultaneously with vaccine suggests that the fever, which rose to above 103°F, may cause release of heparin or a heparin-like substance to produce the clearing factor. This hypothesis is based on observations that injection of protamine sulfate, known to combine with and inactivate heparin(4), reverses the clear-

ing of alimentary lipemia in dogs and rats (5). Furthermore, administration of heparin caused also lipemia clearing although of shorter duration. The assumption that not the high body temperature *per se* but the production of the clearing factor by an intermediary mechanism, possibly release of heparin, induced by the fever is necessary for clearing of the lipemic sera is also suggested by the *in vitro* experiments in which lactescent sera of dogs fed the egg yolk meal failed to clear when kept in water bath of 105°F for 24 hours, a temperature approximately equivalent to that obtained in animals after vaccine and Piromen injection.

*Summary.* Administration of typhoid and paratyphoid vaccine as well as Piromen caused rapid clearing of alimentary lipemia in dogs which developed fever of 103°F or more. This phenomenon appears to be unspecific and seemingly due to marked temperature rise induced by these agents, since no change of turbidity occurred in animals with fever below 103°F. Administration of protamine sulfate to vaccine-treated dogs prevented clearing of the lipemic sera despite a marked elevation of temperature. No clearing of turbidity occurred when the lactescent sera were kept in water bath at 105°F for 24 hours. It is suggested that high body temperature in the dog may cause release of heparin or a heparin-like substance to produce a lipemia clearing factor.

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## Increased Serum Glutamic-Oxalacetic Transaminase of Monkeys Following Oral Administration of Staphylococcal Enterotoxin.* (23931)

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The symptoms of staphylococcal food poisoning are nausea, vomiting, retching, abdominal cramping of varying severity, and diarrhea after an incubation period of 1-6 hrs. Marked prostration with hypothermia and hypotension may occur(1). However, no histopathology has been observed in animals fed enterotoxin.

This paper reports on serum glutamic-oxalacetic transaminase (GOT) levels of monkeys following oral administration of staphylococcal enterotoxin. Elevation of serum GOT which occurs in several disease states(2,3) is attributed to release of the enzyme into the blood circulation following death of injured cells or increase in cellular membrane permeability(4). The slight, but distinct, rise in serum GOT of monkeys fed enterotoxin suggests cellular injury.

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*Materials and methods.* Blood samples were obtained from a leg vein just before enterotoxin was given by stomach tube. These prefeeding blood samples were used to establish normal GOT levels of individual animals. Animals were observed for 5 hrs, vomiting being the criterion of the reaction of monkeys to enterotoxin. Sensitivity of monkeys to enterotoxin varied considerably, some being used for the first time, others having developed substantial resistance through previous feedings of enterotoxin(5). Post-feeding GOT levels were determined on blood drawn about 6 hrs after toxin feeding. GOT assays were made on unhemolysed sera by a method essentially identical to Steinberg *et al.*(6). The Beckmann DU spectrophotometer was used; units are corrected to incubation at 25°C. Sera prepared the day of toxin feeding were kept frozen until used. Sera whose GOT values were to be compared were assayed the