preciable quantity of glucogenic amino acids, it is also possible that rapid conversion of these amino acids to glucose by the liver could account for the changes that occurred in NEFA.

Glucose C-V differences and venous glucose levels were measured concurrently with plasma NEFA in our study. The results show that C-V glucose differences increased significantly during administration of amino acid mixture with concomitant fall in venous glucose. Capillary glucose levels remained virtually unchanged; thus it is difficult to attribute NEFA changes to amino acid-induced hyperglycemia. Moreover, Gordon(5) reported fall in NEFA when nonglucogenic amino acid, leucine, was fed. During administration of the glucogenic amino acid, arginine, C-V glucose differences also increased, but this time, associated with a rise in capillary and venous glucose levels. This observation is in agreement with reports that response of blood glucose to amino acids differs with individual amino acids administered(14). Since blood flow through the forearm was not measured during experiments, the conclusion that rate of glucose uptake by tissues of forearm increased, must remain tentative. However, when added to observation that venous blood sugar level fell significantly during administration of amino acid mixture, these results suggest that at least some of the NEFA-lowering effects of parenterally administered amino acids occurred because of an induced increase in rate of glucose utilization.

Summary. (1) The effect of infusing an amino acid mixture (protein hydrolysate) on plasma NEFA, venous glucose level, and capillary-venous glucose difference, was studied in 6 healthy volunteer subjects. In every instance, plasma NEFA levels fell precipitously during amino acid infusion, and capillary-venous glucose differences increased concurrently. Similar results were obtained when a single amino acid, L-arginine, was infused in 2 normal subjects. (2) The results suggest that NEFA-lowering effects of parenterally administered amino acids may have been mediated entirely or in part *via* an induced increase in rate of glucose utilization.

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Absence of Intestinal Viral Flora in Infants During Epidemic of *Escherichia coli* Gastroenteritis. (24703)

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A large number of instances have been recorded in which specific strains of *Escherichia coli* have been isolated from most or all cases in outbreaks of infant diarrhea(1-6). Several

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TABLE I. Inoculations Carried Out with Preparations #1, 2 and 3.

Host and description	No.	Type of inoculation	No. of passages	Further inoculations
1- to 2-day-old suckling mice	1 litter	IC and IP	1 to several	None
10 to 12 g mice	6 to 8	*Idem*	*Idem*	"
Idem	*Idem*	Fed 0.25 ml via stomach tube	1	"
16 g hamsters	2	IC and IP	1	"
7- to 9-day-old embryonated eggs	6 to 8	CAS	1 to several	"
Monkey kidney Roller tubes	4 to 8	0.2 ml	3	Final harvest of 3rd passage into 1 litter suckling mice
HeLa cell Roller tubes	*Idem*	*Idem*	3	None

IC = Intracerebral inoc. IP = Intraperitoneal inoc. CAS = Allantoic sac inoc.

investigators suggested the possibility that such diarrheas are the result of interaction between the pathogenic bacterium and a virus (7-9). The following study presents an outbreak of infant diarrhea characterized by almost universal presence of *E. coli* 0-111 in fecal specimens(9) and in which intensive efforts for virus isolation gave entirely negative results.

Materials and methods. One or more fecal samples (including 6 autopsy stools) were collected from 25 infants. Two infants in this study were premature at time of onset of disease and the others were within the first 10 days after full term delivery. Time of collection ranged from day of onset to 23 days later during relapses. Two throat washings and one vomitus were also studied. Samples were frozen with dry ice and, within 4 weeks of their collection, 10% suspensions were prepared in 0.85% NaCl and centrifuged at 3,000 rpm at 4°C for ½ hour. Three inocula were prepared from each supernate. Preparation #1: a portion of supernate passed through ultrafine (UF) sintered glass filter. Preparation #2: supernate from a portion of original recentrifuged at 10,000 rpm for ½ hour at 4°C. Preparation #3: a portion of second inoculum centrifuged at 30,000 rpm for 1 hour at 4°C. The supernate was discarded and the sediment, resuspended in 0.85% NaCl with a few ml of the overlay, used for inoculation. One thousand units of penicillin and 50 μg of streptomycin/ml were added to the 2 latter preparations. Inoculations were carried out as shown in Table I. Each of the 3 preparations were inoculated into the 7 hosts listed. In addition, preparations from 7 specimens were inoculated into the yolk sac and amniotic sac as well as onto the chorioallantoic membrane of 6 to 8 embryonated hen's eggs each. Further, several specimens were inoculated intraperitoneally and intracerebrally into suckling rabbits and passaged intratesticularly through 3 to 4 adult male rabbits. Monkey kidney cultures were maintained in medium #199 plus 2% calf serum and HeLa cell cultures in a medium composed of 85% Hanks' solution, 10% chicken serum and 5% chick embryo extract ultra-filtrate.

Results. Numerous *Escherichia coli* 0-111:B_4 organisms were present in fecal samples obtained from all but 2 out of 25 patients and were also isolated from one specimen of vomitus. No *Salmonellae, Shigellae* or pathogenic protozoa were found.

All attempts to establish a transmissible virus from stool filtrates and ultracentrifugates were unsuccessful. In some instances samples were toxic for monkey kidney and HeLa cell cultures and for embryonated eggs, but repeated passages gave entirely negative results. All tissue cultures were obtained from a commercial source (Microbiological Assoc., Bethesda, Md.), and tubes from these same lots were currently being used in other laboratories for study of poliomyelitis and other agents. The same workers who carried out our studies, using the same technics and toward the end of the studies the same lots of

cells, were successful in isolating Polio, Coxsacki and ECHO viruses from fecal samples collected from infants and young children from Puerto Rico and Washington, D. C.

Inability to isolate any virus from this group of severe diarrheal cases is noteworthy in view of the numbers of viruses which have since been isolated by various investigators from fecal samples of infants and young children using monkey kidney tissue cultures alone (10-14). However, Stulberg, *et al.*(15) were unable to isolate any cytopathogenic agents from a group of newborn infants with diarrhea caused by *E. coli* 0-127. Absence of viral agents from our cases, as detectable by methods employed, does not preclude the possibility of a virus actually being present, but the likelihood of any viral agent known to date causing this outbreak, either alone or in combination with *E. coli* 0-111:B_4, appears to be remote. It should be added that sera from 2 of these patients, which had been kept frozen at −20°C for the intervening period, were tested for presence of circulating antigen of *E. coli* 0-111:B_4 by means of hemagglutination-inhibition test(16) and both gave a strongly positive reaction. This result, together with negative viral findings, lends further weight to the conclusion that *Escherichia coli* 0-111:B_4 is by itself the true causative agent in some cases of highly infectious fulminating infant diarrhea.

Summary. Systematic viral isolation attempts were made from an outbreak of infant gastroenteritis in which *Escherichia coli* 0-111:B_4 was found. Treated stool samples collected from 25 infants were inoculated into monkey kidney and HeLa cell tissue cultures, embryonated eggs and a variety of young animals by various routes. No viral agent could be detected. It is concluded that this outbreak of gastroenteritis was caused by *Escherichia coli* 0-111:B_4, uncomplicated by other microbial agents.

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Effects of Total Hypophysectomy, Adenohypophysectomy, and Neurohypophysectomy on Brain Excitability. (24704)

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It is generally accepted that the endocrine system exerts a definite influence on brain excitability, using the minimal electroshock seizure thresholds (EST) for determination. Age(3), ACTH(2,4,7), STH(2,6), adrenocortical steroids(2,3,7) and pitressin(6) also affect EST. The role of the pituitary gland in altering threshold values has been demonstrated(1,2,4,5,7) by measuring the changes induced by complete hypophysectomy. In view of the fact that the pituitary gland is composed of 2 lobes and that the hormones