Because of similarities in the kinetics of enzymatic reactions and of active intestinal transport, the effect of actinomycin D on the latter was investigated. Observations were made of the active transport of amino acids (L-leucine and cycloleucine), sugar (3-0-methyl-D-glucose) and inorganic sulfate in the small intestine of the rat utilizing the everted gut sac technique of WILSON and WISEMAN. Transport by intestinal segments obtained from actinomycin D treated animals (single injection of 66 μg/100 gms subcut.) was compared with transport by intestinal segments obtained from matched controls. Studies were carried out at time intervals ranging from 1 to 48 h following injection. No significant effect of actinomycin D on transport was noted before 24 h. However, active transport of each of the substances studied was markedly increased (2 to 4 fold increase in concentration gradients) in the animals who received actinomycin D 36 to 48 hours previously.

Although commonly used to inhibit enzyme activity, actinomycin D has been shown to induce several enzymes in rat liver 24 to 48 hours after the administration of a single dose (ROSEN: Science 146–661 [1964]). This delayed effect is similar to that observed in our study of intestinal transport and suggests a possible relationship between the two. The mechanism by which actinomycin D stimulates intestinal transport is unknown although it possibly acts by inhibiting a substance that normally limits the rate of transport. (SPR)

81 *Split Products of Fibrinogen in Cord Serum.* E. RICHARD STIEHM*, Univ. Wisc. Med. Sch. Madison, Wisconsin (introduced by C.C. Lobeck).

The presence of split products of fibrinogen (SPF) in serum is an indicator of abnormal fibrinogen deposition or breakdown (e.g. intravascular coagulation). Normal serum (53 samples) does not have material reactive with antiserum to human fibrinogen. 66 umbilical cord serums were studied for SPF by a semiquantitative precipitin tube technique using rabbit antiserum to human fibrinogen. 44 of 66 (67 %) of cord serums had significant levels of SPF, including 26 (39 %) with high levels (> 2 % of tube length). 13 of 32 (41 %) maternal serums obtained immediately post partum had SPF. Serial determinations of SPF in several mothers and infants showed that these products generally disappear rapidly, usually be the second day after birth. No relationship existed between maternal and cord serum levels of SPF in 32 paires specimens; the cord level exceeded the maternal level in 20 instances, the maternal level exceeded the cord level in 7 instances, and in 5 instances neither maternal nor cord serum had SPF. Since gel filtration on Sephadex G-200 indicated that the immunologically active material was a large molecule (MW > 160,000), it is unlikely that SPF in cord serum represents transplacental passage from the maternal circulation. Among randomly selected infants without SPF in their umbilical cord serum, 1 of 16 (6 %) had a respiratory problem or illness during the first days of life. In contrast, 14 of 28 infants (50 %) with SPF greater than 1.0 % had a difficult birth or neonatal illness. This included 2 prematures, 2 neonatal deaths, 7 infants with Apgar scores below 7, 4 infants with respiratory problems and 2 infants with cephalo-hematomas. These data suggest that newborn infants with high and/or persistent levels of SPF may be a high risk group. (SPR)

82 *Temperature, THAM, Glucose and Asphyxia in Newborn Puppies.* JAMES A. MILLER, Jr.*, MICKEY VIA*, EL SAYED H.H. HEGAB* and FAITH S. MILLER*, Tulane Univ. Sch. of Med., New Orleans, La. (introduced by Margaret H.D. Smith).

The time of last gasp (TLG) of asphyxiated neonatal puppies increases from 4.3 minutes at 42° C body temperature to 105 minutes at 15° C. Below 15° C survival times decrease. This is true protection; all 15° animals recovered from exposures up to 1 hour (4X TLG of warm littermates) and 50 % of those exposed 9X or 10X TLG recovered without assistance. In warm animals artificial respiration with 100 % O_2 was only partially successful for animals exposed for $1^1/_4$X to 2X TLG and most showed behavioral deficits indicating brain damage. By contrast, cold animals which were exposed to 10X TLG of warm littermates and which recovered spontaneously showed no evidence of brain damage. During asphyxia, blood glucose, potassium, lactate and hydrogen ions increased and pyruvate decreased. In puppies at 15° all of these changes took place at a slower rate than in warm littermates. In coenothermic animals (37°) controlling pH by infusion of Trishydroxyaminomethane (THAM) increased survival times by 18 %, infusion of glucose by 19 %, and combining the two by 68 %. Cooling to 15° C colonic temperature gave survivals of 655 %. Combining THAM and glucose with hypothermia (15° C) gave an average of 1104 %. THAM-glucose infusions also permitted recoveries from exposures which were lethal for non-infused animals at the same temperature. Thus, the combination has been the most effective of any tried to date. (SPR)

83 *Comparison of Rapid Versus Gradual Correction of Acidosis in RDS of Prematurity. A Sequential Study.* ROBERT USHER, Royal Victoria Hospital, McGill University, Montreal, Canada.

This therapeutic trial of rapid alkali therapy immediately after birth was initiated because of 1. the observation that prognosis in respiratory distress syndrome (RDS) is worse the longer that acidosis persists, as well as 2. the recently proposed hypothesis that the pathogenesis of RDS may be pulmonary hypoperfusion caused by hypoxemia and acidosis. From 1960–1964, and again in 1966, consecutively delivered infants with RDS were treated with slow drip infusions of sodium bicarbonate (5–15 mEq/100 ml 10 % G/W, at 65 ml/kg/day until capillary pH rose to 7.35 (Pediat. Clin. N. Amer. *8:* 525 [1961]). In 1965 a trial of rapid correction of acidosis was made in which pH was corrected up to 7.35 within 3 h of birth, using a solution of 20 mEq Na bicarb/100 ml 10 % G/W. The rates of flow ranged from 10–50 ml/kg/3 h. Other than rapidity of alkali administration, therapy was similar to that used before and after.

	Mortality rate	
	Slow	Rapid
1001–1500 g	35/82–45 %	7/13–54 %
1501–2000	18/87–21 %	6/16–38 %
2001–2500	8/88–9 %	3/12–25 %

Mortality in RDS infants weighing 1001–2500 g was 61 out of 257 (23.7 %) with slow correction, and 16 out of 41 infants (39.0 %) with rapid correction ($P < 0.05$). (SPR)

84 *UDP-Glucuronyl Transferase.* ELIAS HALAC* and PAMELA WEISS*, New York Univ. Sch. of Med. New York, N.Y. (introduced by Joseph Dancis).