jected to a continuous water loading test initiated by giving 25 cc/kg of water while on chlorpropamide in a dose sufficient to cause antidiuresis without hypoglycemia. Water excretion was impaired in all patients. Serum Na fell at least 10 mEq/L in each patient. Serum osmolality fell 25 mosm/kg in 2 patients and fell 20 mosm/kg and 15 mosm/kg in the other 2 patients. All patients gained weight. Chloride spaces increased from 3% to 5% after the water load. CH_2O remained negative throughout the entire test in 2 patients. A third patient developed a CH_2O of +0.9 cc/min/1.73 m^2. This maximum CH_2O occurred 6½ hours after the water load. A fourth patient developed a CH_2O of +2.5 cc/min/1.73 m^2. This maximum CH_2O occurred 2 hours after the water load, subsequently fell to .6 cc/min/1.73 m^2 and never again exceeded +2.1 cc/min. Since published evidence indicates that chlorpropamide acts by potentiating ADH, our data suggests that the subthreshold circulating ADH presumed to be present in our patients is not further suppressed by water loading. Therefore, a potential danger exists for anyone taking chlorpropamide who either requires intravenous therapy, or who may drink a large amount of fluid.

"Transient acetylcholinosis": Cause of Chinese Restaurant syndrome. H. GHADIMI, F. ABAGI, M. RATHI and S. KUMAR. *Downstate Med. Ctr., S.U.N.Y., and Methodist Hosp. of Brooklyn, N. Y.*

Both clinical data and biochemical findings in our studies suggest that the signs and symptoms following monosodium glutamate (MSG) ingestion represent "transient acetylcholinosis." Dose-related reactions occurred in 14 volunteers after MSG ingestion on empty stomach, including numbness of neck, heaviness of eyelids and legs, lacrimation, headache, nausea, urgency of urination and defecation, drowsiness, substernal pressure, abdominal discomfort, palpitations and colicky pain. The protean nature of the symptoms, the mode of appearance and recovery, variations in severity, all were remarkably similar to the diffuse, evanescent action of acetylcholine (ACh). In 4 subjects primed with atropine, there was blockage of symptoms even though MSG dose was doubled. On the other hand, prostigmine (⅓ usual dose) given with MSG markedly exacerbated symptoms in 4 subjects tested. Others have shown that glutamate is a suitable substrate for ACh synthesis. In 10 subjects receiving 150 MSG/kg body wt., cholinesterase (ChE) was measured at 0, 20, 40, 60, 90, 150, and 180 minutes. At 60 min., there was a drop of 30% below baseline. A significantly sharper drop was observed when prostigmine was administered simultaneously with MSG. On the other hand, control tests with histidine showed no fluctuation beyond 5%. Following infusion of ACh into a dog, the pattern of ChE activity was strikingly similar to that observed in man following MSG ingestion. Clinical response to ACh also paralleled human symptoms, except for severity. In 2 infants with Down's Syndrome, ChE changes after MSG also followed the pattern seen in adult volunteers. Judging by this criterion infants do develop Chinese Restaurant Syndrome following MSG ingestion.

Incorporation of heparin-S^{35} by cultured leucocytes as a diagnostic tool in cystic fibrosis (CF). MARK W. STEELE and JOAN B. RODNAN (Intr. by Richard Michaels). *Univ. of Pittsburgh Sch. of Med., Children's Hosp., Pittsburgh, Pa.*

By culturing leucocytes for 5 days in media with Heparin-S^{35} and PHG and then assaying for cellular incorporation of S^{35}, we were able to distinguish homozygous CF from: heterozygous CF and Hurler's; and homozygous normal and Hurler's. We also noted that cells after incorporating higher levels of Heparin-S^{35} disrupted when fixed in Carnoy's mixture; so that after staining with Toluidine Blue O, the slide was covered with heavy amorphous metochromatic debris.

	S*	N†	Mean S.A.‡	Range (S.A.)	Cell Disruption§
Presumed Normals	6	11	27	9–45	2/11
Heterozygous CF	8	15	41	19–83	3/15
Homozygous CF	6	12	129	47–349	12/12
Heterozygous Hurler's	1	2	48	41–55	0/2
Homozygous Hurler's	1	2	39	36–41	0/2

* # of Subjects.
† # of Assays.
‡ DPM/mg. protein.
§ # positive instances/N.

The mean S.A. for homozygous CF was significantly ($p < 0.01$) greater than that for all other subjects. The mean S.A. for heterozygous CF, heterozygous and homozygous Hurler's were all the same and different ($p < 0.01$) from the S.A. for homozygous normal. There was a significant positive ($r = 0.65$, $p < 0.001$) correlation between cell disruption and S.A. We suggest that these two complimentary assay systems could be useful in confirming the diagnosis of CF in questionable cases. Furthermore, contrary to metochromasia, cellular Heparin-S^{35} uptake might differentiate homozygous from heterozygous CF. Hence, if applicable to cultured amniotic fluid cells, the technique could allow detection of homozygous CF in utero.

The response to parathyroid extract (PTE) in infants of diabetic mothers (IDM). REGINALD C. TSANG, LEONARD I. KLEINMAN, IRWIN J. LIGHT, and JAMES M. SUTHERLAND. *Univ. of Cincinnati, Cincinnati, Ohio.*

Neonatal hypocalcemia (NHC) in infants of diabetic mothers (IDM) has been thought to be related to transient hypoparathyroidism or lack of responsiveness to parathyroid hormone. Previous reports of NHC in IDM have not documented its existence when compared to gestation matched infants. A previous study of low birth weight infants demonstrated the importance of early gestation on the incidence of NHC. In the present study 28 IDM were matched with infants of similar age, sex, gestation and perinatal complications. Seven IDM developed NHC compared with one in controls ($p < 0.025$). In IDM mean calcium levels were lower at 12, 24, 48, 60 and 72 hours of age. One IDM (maternal class D) developed temporary hypomagnesemia with NHC. During the first 3 days of life, in all infants tubular reabsorption of P (TRP) fell (93% to 87%), urinary P excretion rose (5 to 40 mg/24 hour) and urinary Ca and Mg remained low (<1 and <0.5 mg/24 hr respectively). In 6 IDM who were given PTE (5 units/kg) at 24 hours and 48 hours of age, 5 responded with temporary elevations of Ca at 12 hours post-injection compared with untreated IDM ($p < 0.05$). There was no significant difference in serum Mg and P levels, TRP and urinary P, Ca and Mg between treated and untreated IDM