then observing the effect of dexamethasone on blood pressure.

THYROID AND IMMUNOLOGICAL STUDIES AFTER CHILDHOOD HEAD AND NECK IRRADIATION. A. Guansing,* J. Arkins,** N. Engbring,** J. Cerletty, R. Holmes, S. Wilson,* T. Hagen, P. Rosenfeld, R. Komorowski* and K. Shetty.* Departments of Medicine, Radiology, Surgery and Pathology, Medical College of Wisconsin, Milwaukee, Wisconsin.

In an attempt to characterize the delayed effects of head and neck irradiation during childhood, thyroid screening procedures and delayed hypersensitivity testing were carried out in a population at risk. Of the first 500 patients examined (209 males, 291 females; age range 21-43 years), 96 (19%) had thyroid abnormalities. Solitary nodules accounted for 43%, diffuse enlargement for 34% and multiple nodules for 4% of cases. 19% had thyroid surgery prior to examination. Technitium ^{99M} Pertechnitate scanning demonstrated 84.2% of the nodular lesions. TSH levels were elevated (31.0 $\pm$ 3.1 uU/ml SEM) in 14 (2.8%) cases. All had normal thyroxine levels. Clinical hypothyroidism was not found. One patient had Graves' disease. Thyroid antibodies were positive at low titers (1:100) in 2.8% of cases. Delayed hypersensitivity tests using Monilia, Trichophyton and Mumps antigens showed 74.2% positive skin reactions (to at least 1 of the 3) in 349 individuals. Those with thyroid abnormalities had 84.6% positive skin reactions. At surgery, 11 of 19 patients (57.9%) had benign adenoma and 8 (42.1%) had thyroid carcinoma (4 follicular, 2 papillary and 2 mixed type). No correlation was found between malignancy and the skin test results.

In conclusion, childhood head and neck irradiation leads to significant thyroid abnormalities with a high malignancy rate. Furthermore, a defect in delayed hypersensitivity response, using skin reactivity to antigens as an indicator, was not demonstrated in this group.

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HYPOGLYCEMIA, A NON-THYROTROPIN RELEASING HORMONE (TRH) MEDIATED STIMULUS TO PROLACTIN (Prl) RELEASE IN RATS. A.R. Guansing,* Y. Leung,* K. Ajlouni,* B. Piacsek* and T.C. Hagen. Department of Medicine, Wood VA Center, Medical College of Wisconsin and Department of Biology, Marquette University, Milwaukee, Wisconsin.

Studies in our laboratory indicate that hypoglycemia is a stimulus to TRH release in rats. To determine whether such an endogenous secretion of TRH could lead to Prl release, and to ascertain whether such a rise in Prl could modulate thyrotropin (TSH) secretion, hypoglycemia was induced by intraperitoneal injection of insulin in adult female Holtzman rats divided into 8 groups of 6. Following rapid decapitation trunk blood was collected and tissues were taken and extracted in cold methanol. Serial measurements of blood sugar (BS), hypothalamic (Hth) TRH, TSH, and serum Prl were performed as shown in the table. In

TIME (min)	0	15	30	45	60	90	120	180
BS (mg%)	120	39+++	60++	55++	83	62++	77	62++
Hth-TRH (ng/Hth)	3.3	1.5+++	5.0++	4.5	4.1	4.4	4.6+	6.2++
Pit-TSH (mcg/pit)	10	8.1	2.7++	6+	2.4+	3.2++	5.5+	9.2
Serum TSH (uU/ml)	42	29	102	141+	53	25	17	16
Serum Prl (ng/ml)	19	28	32	20	29	187+	155+	93

+ $p < 0.05$ ++ $p < 0.02$ +++ $p < 0.01$

association with a fall in blood sugar, Hth-TRH decreased and within 30 minutes a peak serum TSH level was observed. Serum prolactin increased 45 minutes after the peak TSH response. Serum T3 rose from a basal value of 108 ng% to a peak of 712 ng% at 90 minutes. Serum T4 was unchanged.

We conclude that hypoglycemia stimulates delayed Prl release in rats which probably is unrelated to TRH secretion. The data further indicate that Prl is not a factor in the TSH response to hypoglycemia.