

without biochemical confirmation of catecholamine excess, but I would not discount the possibility we might do it in a patient in whom all the affected members of the kindred have pheochromocytoma. We have not found adrenal scans, arteriography, or caval catheterization to be of much value in adrenal medullary hyperplasia. We think it is important to rule out this diagnosis because it is potentially lethal. Four of the sixteen patients who had pheochromocytomas in our group of patients died as a direct consequence of their disease, three in hypertensive crisis, one with pulmonary metastases. We also have tried autotransplantation of diced adrenal cortical tissue in much the same way as the Ann Arbor group has done, and with the same lack of success.

Concerning the medullary thyroid cancer: not all of these affected patients have elevated basal calcitonin levels; 30% of our group did not. We have likewise found pentagastrin stimulation to be more reliable than the calcium infusion test.

As to how young patients might be when one begins screening, we have had ten affected patients under 10 years. The youngest was age 1½ years.

What about the success of surgery for medullary thyroid cancer? Dr. Freier emphasized that a number of their patients were not cured by total thyroidectomy. That has also been our experience. Forty percent of the group having total thyroidectomy with or without vein dissection have not been cured. Dr. Block emphasized that interesting group of patients who have no clinical evidence of the disease but who have elevated calcitonin levels after operation. We have seen this also.

Finally, as far as the parathyroid disease is concerned, about half of the group are eucalcemic with normal parathyroid hormone levels, but they are found to have hyperplastic glands at the time of operation. We do not know what to do with these patients, and I would like to ask the Ann Arbor group what they do. With some of the patients in this group we have done a subtotal parathyroidectomy and in others we have simply biopsied the glands and tagged them.

We do not know the natural history of this disease yet. The other half of the patients are truly hypercalcemic and have bona fide hyperparathyroidism; we treat them by a subtotal parathyroidectomy.

I want to congratulate the authors for shedding some light on this rather complex area of surgical interest.

Dr. Duane Freier (closing). The first question of Dr. Block's concerning what to do after you have done a thyroidectomy and the patient continues to have hypercalcitonemia is very pertinent. If a palpable lesion develops during close follow-up, we excise it. If not, we simply follow the patient along. We do not know what the natural history is. We have had some elderly folks who have very high levels, who had their operations 20 years before, and do not show any symptoms or physical evidence of their disease.

The medullary hyperplasia localization by serum sample that Dr. Edis mentioned has not been helpful for them and not for us. It has been frustrating.

Autotransplantation of the adrenal cortex is difficult. There are reported cases of cortical transplantations that have functioned, but those that have been successful have consisted of huge slices of cortex put into large muscular areas. However, if you have a lot of medullary hyperplasia you cannot get much cortex, and you do not want to transplant the hyperplastic medulla.

The MEA IIb patients can be obvious within a year of life, but I do not think the MEA IIa patients have shown their genetic transmission until later.

Concerning the hyperparathyroid dilemma: What if you do a thyroidectomy on a patient who has normal calciums and notice there is a hyperplastic gland? We remove the grossly enlarged glands and leave the rest of them behind. We follow Dr. Block's admonition that you treat these conservatively, because the parathyroid hyperplasia in MEA II has not been a significant clinical problem.

ERRATUM

In the article "L. R. D.—recollections and reminiscences" (*SURGERY* 81:442, 1977) by John H. Landor, the date of death of Lester Reynold Dragstedt inadvertently was printed as having occurred on July 16, 1976. Dr. Dragstedt died on July 16, 1975.