ciable, it requires aggressive surgery and outcome is poor (only 57% were salvaged in our series).

To address some of Dr. Clark's questions:

Patients that have less than a total thyroidectomy require ablation by either surgery or radioactive iodine before metastatic thyroid cancer can be effectively treated by I^{131}. Indeed, if there is only a small remnant of normal thyroid tissue left, then ablation by iodine is most reasonable. In patients where an entire lobe remains, then it is reasonable to complete the thyroidectomy before starting the iodine I^{131} because of the large amount of iodine required to ablate the normal gland. Approximately, 100 μCi are needed to ablate a remaining normal gland, which may compromise the amount of I^{131} that can be used safely to aggressively treat the metastases. The maximum tolerated dose of I^{131} given as a single fraction is approximately 250 to 300 μCi, although if one treats recurrence over a protracted period up to 600 to 700 μCi can be given, usually in 100- to 150-μCi fractions. Thus, it is prudent to limit the use of radioiodine to treat the metastases only rather than compromise the treatment by having to use I^{131} to ablate a large amount of residual thyroid tissue.

Differentiating radioiodine uptake in the thyroid bed from recurrence in the central neck may be difficult, as is obvious in the question posed by Dr. Clark. It is well recognized that post-total thyroidectomy scanning of the neck will show some remnant uptake in the bed which may represent a small focus of occult thyroid capsule and which can be ablated easily. Postoperative activity in the side of the ipsilateral lobe is assumed to be cancer; activity in the contralateral gland is considered normal tissue unless it occurs in an area previously free of uptake (or if there was evidence of multifocality at initial surgery).

In the patient who has been treated adequately for a primary thyroid cancer as confirmed by a postoperative nuclear scan and has a follow-up study showing uptake in the thyroid bed, this is tantamount to a diagnosis of recurrent disease. A computed tomography scan is required to determine whether there is a mass at the site of radioiodine uptake. If one demonstrates minimal disease, *i.e.*, less than 1 cm, then radioiodine ablation would be our first choice and in most cases, should be curative. If there is bulk disease, we would favor doing a re-exploration with resection of the thyroid bed and a regional node dissection. If there is any residual disease, this microscopic cancer could be treated with additional I^{131}. In our study, most of the distant metastases were treated in the lung, which as Dr. Clark pointed out, respond to I^{131} better than disease in the bone. Although metastases in a bony site can be treated by I^{131}, external beam radiation also can be used to supplement therapy. Regarding thyroglobulin levels, our database did not have sufficient patients with thyroglobulin levels performed and we do not have the data to answer Dr. Clark's final question. Elevated thyroglobulin levels should return to normal levels after treatment of recurrence. If there is clinical evidence of recurrence *versus* a radionuclear scan evidence, this more likely would be associated with an elevated thyroglobulin level reflecting the extent of disease. As is stated in the article, the salvage rate is obviously higher in patients with limited recurrence (as documented by scan only) and is only about half that in patients with clinically documented recurrences (who probably would generally have higher serum thyroglobulin levels).

Thyroglobulin levels are a useful monitoring technique in high-risk thyroid cancer patients after therapy. Our personal data suggest that routine measurement may be used in place of follow-up scans, avoiding the thyroid hormone withdrawal sequence. A rise in thyroglobulin levels, however, would prompt evaluation by scan to localize recurrence.

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April 11, 1995

Dear Editor:

We read with interest the paper by Xu and others.[1] The authors have demonstrated that prostaglandin E_1 treatment increases survival with extended anhepatic phase during rat liver transplantation, but the mechanism of action remains purely speculative.

The products of the arachidonic acid cascade have diverse effects on different organ systems. The effects of prostaglandin E_1, as the authors have discussed, are numerous, and its cytoprotective, vasolidator, and platelet aggregation and thromboxane generation-inhibiting properties are well established. The same effects can be attributed to prostacyclin and prostaglandin E_2[-]. It also has been demonstrated that the effects of the products of the arachidonic acid cascade are interrelated.

We recently conducted a similar study on liver transplantation in pigs based on the assumption that the liver glycogen content plays an important role on post-transplantation liver functions. During liver harvesting from the donor animal in the first group (n = 3), a 20% dextrose solution containing 20 mEq potassium chloride and 100 units of insulin was infused from the splenic vein at a rate of 5 mL/kg/hour. Into the same solution 5 μg/mL of Iloprost (Schering, Germany; ZK 36374), a prostacyclin analogue, was added and transfused at the same rate to the second group (n = 4) and the harvested livers were transplanted to recipient animals. Liver biopsies for glycogen content determination were obtained from both groups at the beginning and end of the harvesting procedure, at the end of the cold ischemia period when the portal and caval clamps were removed, and 60 minutes after reperfusion. The liver glycogen contents were 124 ± 8.6 mg, 165 ± 71 mg, 61 ± 27 mg, and 20 ± 8.6 mg per 100 g tissue respectively in the first group, whereas the same values were 136 ± 9 mg, 773 ± 176 mg, 646 ± 259 mg, and 533 ± 239 mg per 100 g tissue in the second group. (The values are given as mean ± standard error of the mean.)

As can clearly be seen, intraportal Iloprost infusion was very effective in increasing and preserving the liver glycogen content during the harvesting procedure, cold ischemia period, and after transplantation during reperfusion. The glycogen content has been found to be an important prognostic indicator in liver transplantation.[2]

Previously, we demonstrated the beneficial effect of iloprost on hepatic ischemia reperfusion injury[3] and on isolated, transplanted pancreatic islet cells.[4]

Therefore, our data provide further support to the authors' findings. Although prostacyclin has a different metabolic path-

way than prostaglandin E_2 and prostaglandin E_1, all of them have similar physiologic properties. The beneficial effect of prostaglandin E_1 in increasing the survival with extended anhepatic phase may be further attributed to the increase in the liver glycogen content.

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May 11, 1995

Dear Editor:

We are grateful for the interest shown by Drs. Yegen and colleagues in our published paper on prostaglandin E_1 and liver transplantation.[1]

Liver glycogen content in the graft has a close relation with the liver viability after long-term cold preservation[2] and is an important prognostic indicator[3] in the liver-transplanted recipient. Dr. Yegen and his colleagues found significant higher glycogen content in pig's donor livers perfused by a solution with Iloprost (ZK 36374) and prostacyclin analogue than the control. We agree the inference from their study that the beneficial effect of prostaglandin E_1 in increasing the survival with extended anhepatic phase may be attributed to the increase in liver glycogen content.

We appreciate their data providing further support to our findings.

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May 25, 1995

Dear Editor:

I read with great interest the article by Drs. Jordan and Thornby on perforated pyloroduodenal ulcers.[1]

This article and its discussion, as well as another recent communication by the same authors[2] offer an apparently clear message: parietal cell vagotomy (PCV) should be restricted to patients who have real duodenal ulcers and, because PCV for pyloric and prepyloric ulcers are associated with an unacceptable recurrence rate, an antrectomy should be added for these types of ulcers.

I would like to point out, however, that the addition of gastric resection is not mandatory in the definitive management of complicated and uncomplicated pyloric and prepyloric ulcers, nor should it be considered the procedure of choice. PCV with a drainage procedure (PCV+D) represents an excellent alternative. First performed in the laboratory of Griffith and Harkins[3] it subsequently was performed by Holle's group in Munich in a few thousand patients.[4] After a prolonged follow-up, the ulcer recurrence rate was below 5%, and notably, excellent results were achieved in all types of ulcers, including pyloric and Johnson's type I, II (prepyloric), and III gastric ulcers. In a prospective, randomized trial from Sweden, PCV+D was shown to be superior to PCV in all types of ulcers.[5] PCV+D also proved superb in patients with obstructing duodenal ulcers.[6,7]

The technique of drainage added to the PCV varies among authors to include Finney's or Heineke-Mikulicz pyloroplasty, Jaboulay's gastroduodenostomy or Holle's pyloroplasty (anterior hemipylorectomy). The pyloroplasty incision may include or transverse the anterior perforations of the duodenal, pyloric, or prepyloric ulcers and through it, posterior bleeding ulcers can be oversewn. When local circumstances for pyloroplasty were unfavorable because of excessive inflammation, we have added a posterior gastroenterostomy instead.[8]

It is unclear as yet why PCV+D succeeds where PCV alone fails. It may be that the addition of drainage prevents gastric stasis-associated hypergastrinemia and that the pyloroplasty increases the completeness of vagotomy.[9] Another commonly asked question is why should PCV+D be better than the "simple-old" truncal vagotomy-pyloroplasty or selective vagotomy-antrectomy? Clearly, as opposed to the other options, PCV+D preserves both the antrum and its innervation, a factor that may account for the improved functional results.

Surgeons should be aware that PCV+D is a good operative option for complicated and uncomplicated pyloric and prepyloric ulcers.

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