cally and biochemically had primary hyperparathyroidism. All six patients had initial negative parathyroid images with technetium-99m/thallium-201 dual isotope scintigraphy using the technique described by Gordon and Carr (*3*).

A blood sample was drawn for serum calcium, serum phosphate, and PTH levels. Our measurement of PTH was by radioimmunoassay using the PTH mid-molecular portion. A normal range was 0–85 pm/l with a normal serum calcium. The patients were then given Fleets Phospho-Soda, 1/2 teaspoon three times daily for 7 days, which provided ~1 g/day of phosphorous. Upon return to our laboratory, a second blood sample was drawn for the above parameters, and the patients were re-imaged.

The results revealed that in all six patients serum calcium and phosphate levels decreased while the PTH level slightly increased in two, decreased in three, and was not available in one patient. Only in one patient was the second image read as being positive, and in this patient the PTH was not available. In this patient, the presence or absence of an abnormality on the first image reading was debated, but officially it was read as negative. To date, none of these six patients has undergone surgical exploration to definitively identify the presence or absence of parathyroid disease.

An underlying purpose in our using phosphate intervention was to evaluate its possible use as a routine premedication for the dual isotope scintigraphy. However, since the results of the images were not significantly altered, we do not feel oral phosphate therapy to be beneficial; nor should it be used routinely.

References

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3. Gordon L, Carr D: Preoperative localization of parathyroid adenomas using thallium-201 technetium-99m subtraction scintigraphy. *So Med J*: in press

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REPLY: We read with interest the letter of Carr et al. about their experience with the use of oral phosphate to enhance parathyroid adenoma detectability on dual isotope scintigraphy. Limitation of resolution is a major problem with scanning parathyroid adenomas, with 0.250 g being the lower limit in our experience (*1*). It is possible that small adenomas in the cases of Carr et al. may have been below the resolution limits. Of course, one would also have to take into account the possibility of hypercalcemia being due to causes other than primary hyperparathyroidism in their patients, especially since they all had negative initial images.

The dose and duration of phosphate therapy is another important factor which may influence the results. We found in two patients the use of oral phosphate, 1 g/day for three weeks resulted in elevation of PTH with enhancement of already visualized adenomas. In one patient however, oral phosphate 1 g/day for only 10 days did not influence the calcium, PTH, or the scan. The use of phosphate for only 7 days in their cases was therefore probably not sufficient as was seen in their results where only two out of six patients had slight increase in PTH level.

In conclusion, therefore, the subjects studied by Carr et al. were not strictly comparable to our cases because of having negative initial images and a much shorter course of phosphate administration. Our cases with enhanced images also had definite PTH elevation after phosphate administration.

We agree with Carr et al. that routine use of phosphate is not indicated at the present time but also believe that this maneuver does provide a way to enhance imaging in appropriate patients where initial images may have been equivocal. One might be able to judge whether re-imaging is worthwhile following phosphate therapy by assessing PTH level in comparison with a baseline study.

References

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Correction: Cardiovascular Nuclear Medicine-Training for the Future

In an Editorial appearing in *J Nucl Med* 27:1642–1643, 1986, Reference *1* should read as follows:

1. McPhee SJ, Garnick DW: Imaging the heart:Cardiac scintigraphy and echocardiography in U.S. hospitals. *J Nucl Med* 27:1635–1641, 1986