stance represents a new element, to which we propose to give the name RADIUM.

We have determined the atomic weight of our active barium in proportion to the chlorine in the anhydrous chloride. We have found numbers which differ very little from those obtained in a like manner using inactive barium; however, the numbers for active barium are always a little heavier, but the difference is of the order of magnitude of the experimental error.

The new radioactive substance contains, of course, a very high proportion of barium; in spite of this the radioactivity is considerable. The radioactivity of radium must therefore be enormous.

Uranium, thorium, polonium, radium and their compounds cause air to conduct electricity and act photographically on sensitive plates. From these two standpoints, polonium and radium are considerably more active than uranium and thorium. On photographic plates one obtains good exposures with radium and polonium in one-half minute; it takes several hours of exposure to obtain the same results with uranium or thorium.

The rays emitted by the compounds of polonium and radium cause barium platinocyanide to fluoresce; their action from this standpoint is analogous to Röntgen rays but considerably weaker. To perform the experiment, one places on the active substance, a very thin sheet of aluminum upon which is spread a thin layer of barium platinocyanide; in the dark, the platinocyanide appears faintly luminous.

A source of light is thus achieved, and although, in truth, it is a very feeble light, it functions without any source of energy. This is a contradiction of the principle of Carnot, or at least it appears to be.

Uranium and thorium do not give off light under these conditions, their action probably being too weak.

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INCREASE IN PBI CAUSED BY LT_3

I am concerned that the literature should contain the statement that the administration of LT_3 increases the PBI. In the article by Dr. Cuarón entitled "Determination of Serum Thyroxin by Saturation Analysis of Thyroxin-Binding Protein" (*J. Nucl. Med.* **10**:532, 1969) there is a statement and a figure to support that statement which must be challenged.

On page 536 there appears, "Daily administration of 100 μg of L triiodothyronine for 10 days increased the PBI concentration in the serum from every subject studied but had quite a different effect on T_4 iodine level". Figure 3 on page 537 graphically illustrates these findings.

It should be noted that this finding is entirely different from many other reports in the literature and textbooks. These indicate that the PBI is reduced in euthyroid individuals receiving L triiodothyronine. Indeed, this is the basis of the T_3 suppression test. It would be a great disservice if this error (from whatever cause) should remain in the literature unchallenged.

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THE AUTHOR'S REPLY

I do appreciate the observations made by Dr. Jackson concerning our paper on serum thyroxine estimation. His comment has in itself the accreditation of rightness.

The fact that T_3 administration decreases thyroid function in euthyroid subjects but fails to do so in thyrotoxic patients is well known. Last June we sent to the *Journal of Nuclear Medicine* a paper designed to study the effect of T_3 medication on T_4 concentration in sera from euthyroid and thyrotoxic individuals and to assess the value of this procedure in the diagnosis of borderline thyrotoxicosis in iodine-contaminated patients. A clear-cut distinction between both groups was obtained, mainly when the inhibition of serum thyroxine concentration was described as a percent fraction of the basal pretreatment value.

In regard to his pertinent observation, the only obvious explanation for the unusual increase of protein-bound iodine concentration in sera from those subjects receiving T_3 medication is a presumable contamination of the hormone we were using at that time with other iodinated compounds.

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