Re: The Auger Effect, Internal Conversion, and Isomeric Transitions

There seems to be a tendency in nuclear medicine textbooks (*1-3*) to describe the Auger effect as the emission of an x-ray followed by a photoelectric absorption and resulting ejection of an electron. Internal conversion, a nuclear Auger effect, is often similarly described as due to emission of a gamma photon from the nucleus with photoelectric absorption and ejection of an electron. At least one textbook (*4*) also states that internal conversion (IC) and isomeric transition (IT) are synonomous.

While a better understanding of these processes may have little immediate impact on the everyday practice of nuclear medicine, it is still, I hope, worth while to prevent the propagation of these misconceptions into yet another generation of textbooks and students.

It has been known for many years that the Auger effect and its nuclear equivalent, internal conversion, are "radiationless" transfers of energy from the atomic or nuclear exicted state *directly* to the ejected electron. To quote Max Born (*5*), "It is important that this process should *not* (his emphasis) be regarded as one in which the nucleus emits a gamma photon, which then knocks out an electron."

Indeed, a significant portion of theoretical nuclear physics is based on this premise, and many physicist-hours, including some of my own, have been spent in support of it.

The transfer of energy in the atomic and nuclear Auger phenomenon is best understood in terms of the wave-mechanical description of the atom, whereby the wave functions describing the electrons and the nucleus may overlap. Thus, put differently, a K-shell electron, for example, has a finite chance of being found in, or penetrating, the nucleus.

A transfer of energy, or a "quantum-mechanical coupling," may therefore, occur without the necessity of postulating a gamma-ray (or x-ray) "carrier" of energy.

This "nuclear penetration," incidentally, may also be invoked to describe the process of electron capture.

The term *nuclear isomer* denotes different energy states of the same nucleus, and an *isomeric transition* is a transition between two such states. Gamma emission and internal conversion are but two mechanisms, albeit the most common, by which an isomeric transition may occur.

In support of the above statements on the Auger effect, three pieces of evidence:

1. To take the circumstances in which Auger first discovered his effect (*6*), which involved the x-ray irradiation of argon gas in a Wilson cloud chamber, it was noted that the Auger electrons always originated from the same point (atom) as the original photoelectron that causes the vacancy leading to the Auger effect. If the Auger effect were due to the two-step process, the x-ray emitted in the first step should sometimes travel to other parts of the gas and generate "Auger" electrons there. This does not happen. Furthermore, the absorption coefficient for argon and its K x-rays is known. It can be calculated that only one in a million x-rays should produce an "Auger" electron by photoelectric interaction, whereas Auger observed electrons in 93% of his excited atoms (*6*).

2. Gamma transitions abide by a set of well-defined "selection rules" involving the spins and parities of the initial and final energy states. For an initial and final state both having net spin = 0, single-quantum emission is *absolutely forbidden.* Thus there are nuclides where the first step of a two-step process is impossible, yet these transitions still occur by internal conversion.

3. Internal conversion provides an additional mechanism by which an exicted nucleus may lose energy. If it is "frustrated" from decaying by gamma emission by the above-mentioned selection rules, it is more likely to resort to internal conversion. Thus internal conversion can have a measurable effect on the rate of decay of a nuclide.

Since the modern theory of IC involves the wave functions of the atomic electrons, it should be possible to change the rate of decay of such a nuclide by altering its electronic wave functions. Such modification can be achieved by incorporating the atom into different molecular compounds. Goldhaber and Wilson (*5*) performed such an experiment in 1957 and confirmed this effect.

Which nuclide did they use? Well, they needed a nuclide with a convenient half-life, capable of incorporation into a variety of complex molecules where gamma emission would be hindered by the selection rules and IC thus a significant mode of decay. They chose a metastable isomer of Tc-99 with a 6 hr half-life!

We may note as a finale that Tc-99m has such a long half-life because of the spin and parity changes involved in going from the 1/2-level at 142.63 keV to the 7/2+ level at 140.51 keV or the 9/2+ ground-state level (*7*), and the fact that the selection rules penalize such large changes of parity and spin.

One might say that quantum mechanics is more important to nuclear medicine than we had realized. One might also say that we have been making good use of nuclear spin for many years, before the current exciting possibilities of NMR developed.

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Gallium Scintigraphy in Toxic Shock Syndrome

Toxic shock syndrome (TSS) is a multisystem disease, described in 1978 by Todd and coworkers, having a variety of physical and laboratory manifestations and having well-defined epidemiologic and clinical criteria for diagnosis (*1*). The syndrome has been associated with infection or colonization with a strain of *Staphylococcus aureus* that produces a unique epidermal toxin (*2*). We wish to add toxic shock syndrome to the list of causes of abnormal diffuse soft-tissue uptake of gallium-67, which to our knowledge has not been described previously.

A 63-yr-old man with an undefined lymphoproliferative disorder was admitted to our hospital with fevers to 40°C, severe myalgias that limited motion, and diffuse maculopapular erythematous rash with induration of both thighs and forearms. A diagnosis of toxic shock syndrome was made, with the following supporting physical and laboratory findings: Temperature to 39.2°C; an erythematous rash involving thighs and forearms, which after one week was followed by desquamation in his right upper extremity (a biopsy of the involved skin in the right thigh showed nonspecific inflam-

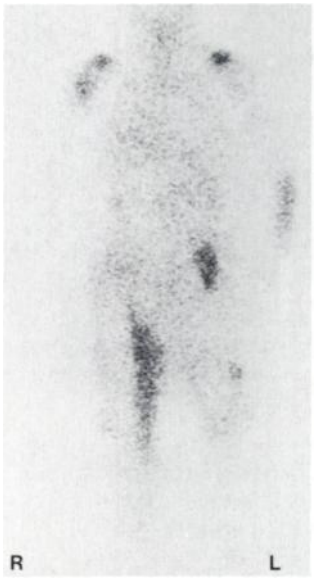

FIG. 1. Tomographic plane at 12.5 cm below anterior chest wall shows multiple regions of uptake of Ga-67 in soft tissues of all four extremities and left lower abdominal wall.

matory reaction with negative Gram stain and culture). Postural hypotension. Involvement of seven organ systems: renal deterioration, with serum urea nitrogen of 94 mg/dl and serum creatinine of 5.4 mg/dl; hepatic deterioration with total bilirubin of 7.6 mg/dl (normal, 0.2–1.2); serum lactic dehydrogenase of 380 U/l (normal, 80–200), and serum glutamic oxalacetic transaminase of 135 U/l (normal, 0–41); muscular involvement, with creatinine phosphokinase of 281 IU/l and 335 IU/l (normal, 0–108); gastrointestinal involvement, with symptoms of dyspepsia and erosive gastritis; hematologic manifestations, with the development of disseminated intravascular coagulation; mucous-membrane involvement, with pharyngitis (throat culture positive with coagulase-positive *Staphylococcus aureus*) and a "beefy-red strawberry tongue." Central nervous system deterioriation, with eventual

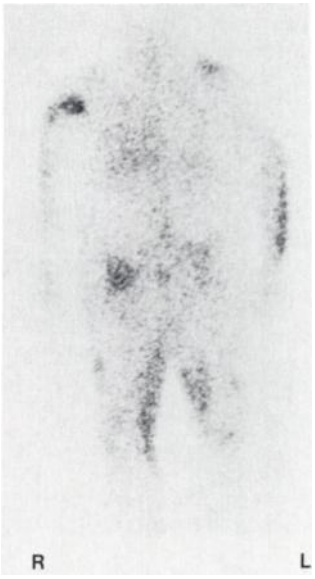

FIG. 2. Tomographic plane 20.0 cm below anterior chest wall again shows uptake in soft tissues of extremities, with additional uptake in right gluteal region.

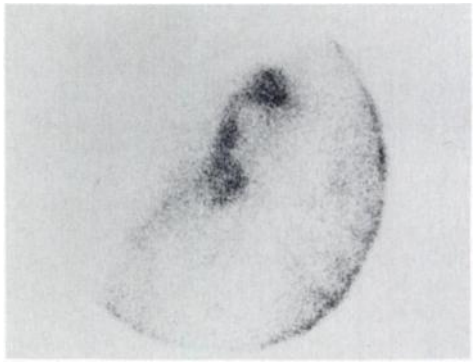

FIG. 3. Gallium-67 scintiphoto of right anterior shoulder shows activity in muscle, but no epidermal or bone involvement.

development of coma. There were other metabolic aberrations often found in toxic shock syndrome: specifically hypokalemia (serum potassium, 1.3 mmole/l), hypocalcemia (serum calcium, 6.8 mg/ml), and a persistent metabolic acidosis. Blood cultures grew coagulase-positive *S. aureus*. Total-body tomographic images were obtained* after injection of 5 mCi of gallium-67 citrate, and these showed multiple soft-tissue concentrations in all four extremities as well as in the left inferior abdominal wall and the right gluteal region—areas not associated with skin lesions (Fig. 1 and 2). Figure 3 is a spot image of the right shoulder, showing activity in the soft tissues but not in bone. With continued deterioration and lack of response to appropriate antibiotics, the patient died approximately two weeks after admission.

This case demonstrates accumulation of gallium-67 in the skin, subcutaneous tissue, and muscle, which are regions of known involvement in toxic shock syndrome. The pathogenesis of this is unknown, but one or more staphylococcal exotoxins may be absorbed into the circulation from soft-tissue colonization or infection. Furthermore, the syndrome has been described with *Staphylococcus aureus* bacteremia (*3*). The exotoxin(s) in toxic shock syndrome presumably cause a diffuse inflammatory reaction in the involved soft tissues, which would explain the nonspecific pattern on gallium scintigraphy. It is different from that in staphylococcal abscess formation where, because of direct bacterial involvement, the gallium uptake is usually discrete (*4*). The more extensive and deeper involvement of the subcutaneous tissues in toxic shock syndrome may also make the gallium distribution different from that in a cellulitis.

There are two situations, however, where the abnormalities noted on gallium scintigraphy during staphylococcal infection may suggest toxic shock syndrome. In necrotizing fasciitis the elaboration of an erythrogenic toxin causes widespread necrosis of superficial fascia with extensive undermining of contiguous tissue (*5*). Likewise, subcutaneous abscesses experimentally induced in rats with *S. aureus* have shown extensive collections of pus extending into the underlying muscle without discrete wall formation. Necrotic-appearing muscle tissue surrounding the pus showed increased gallium uptake, more than the purulent material itself (*6*). These conditions are quite distinct from the multisystem manifestations of toxic shock syndrome and should be distinguished on clinical grounds.

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FOOTNOTE

* Searle PhoCon multiplane tomographic scanner.

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Osteomyelitis of the Ilium: Presentation as an Abdominal Syndrome

Acute osteomyelitis of the ilium is an uncommon condition that should be included in the differential diagnosis of prolonged, unexplained, lower abdominal pain. Clinical signs and symptoms imitate those of acute appendicitis on the right side, or a paracolic abscess on the left. In a recent case we found a gallium scan and a confirmatory bone scan diagnostically valuable.

A 28-yr-old black male with a long history of intravenous drug abuse was admitted with a three-week history of left lower-quadrant pain radiating to his groin. One day before admission, he had a shaking chill, nausea, and vomiting. On admission, physical examination was normal except for left lower-quadrant tenderness. The WBC count was 11,900, with 85% polymorphs. The erythrocyte sedimentation rate was 30 mm/hr (normal 0-15).

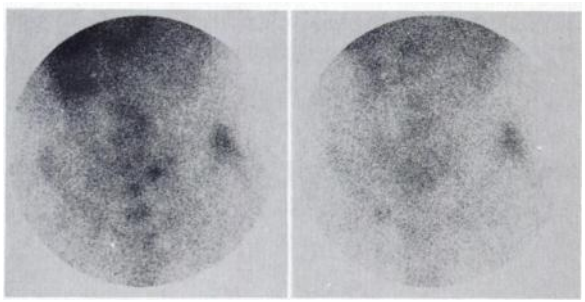

FIG. 1. Gallium-67 scan of anterior pelvis. At 24 hr, (left) asymmetric uptake is visible in left lower quadrant overlying area of iliac crest. There is also colonic activity. At 48 hr, (right) activity over left iliac crest persists; colonic activity has diminished.

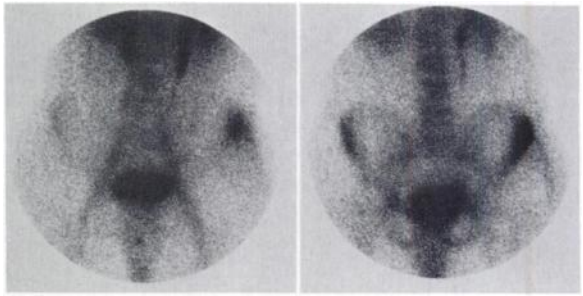

FIG. 2. Tc-99m MDP anterior scan of pelvis. At 5 min after injection, (left) there is significant but poorly defined increased uptake at area of iliac crest. At 2 hr, (right) local increased uptake in left iliac crest is due to osteomyelitis.

Other laboratory studies were all normal, including blood cultures, stool guaiacs, urinalysis, chest, and abdominal radiographs. He was presumed to have an intra-abdominal abscess.

Images taken 24 and 48 hr after injection of gallium-67 citrate (3.0 mCi) revealed increased uptake in the left lower quadrant, overlying the area of the left iliac crest (Fig. 1). At 72 hr after dose, a bone scan with 20.0 mCi of Tc-99m methylene diphosphonate showed markedly increased uptake in the left iliac crest (Fig. 2), from which *Pseudomonas aeruginosa* was obtained by needle aspiration. Radiographically, subtle rarefaction and erosion were evident on the early films; some remineralization could be seen after therapy had begun.

Hematogenous osteomyelitis of any site is now uncommon, and the reported incidence in the ilium is 2-5% of all osteomyelitis cases (*1,2*). If the inner cortex of the ilium is penetrated, inflammation spreads to the iliac fossa, and an abdominal syndrome may develop, with the patient referred to the general surgeon, as in our case. Six reported cases of acute osteomyelitis of the right ilium have caused removal of a normal appendix (*1-4*).

The presence of previously injected gallium does not preclude use of a bone scan to facilitate the diagnosis of osteomyelitis. In our patient, decay and excretion had reduced skeletal gallium activity to ~0.25 mCi (assuming 24% distributed to the skeleton) by the time of the bone scan (*5*). Allowing for physical decay and biological excretion, if 50% of the Tc-99m MDP was adsorbed to the skeleton (*6*), there were 8 mCi of technetium in the skeleton, giving a 32-fold predominance over the Ga-67 and minimizing the influence of gallium scatter. In institutions where higher doses of gallium are routinely used, Compton scatter may be more of a problem. This effect can be minimized with computer subtraction of the gallium background image of the suspicious area before the injection of the technetium agent. Prolonged diagnostic evaluation, unnecessary surgical intervention, and progressive osteomyelitis can be avoided with this approach.

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