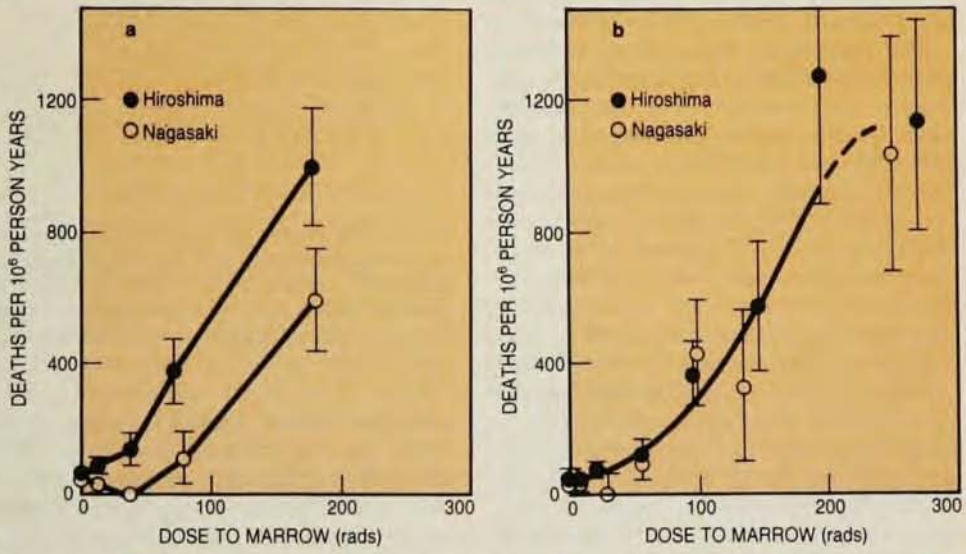
Studies revise dose estimates of A-bomb survivors

We know very little about the long-term health hazards of human exposure to neutron irradiation—certainly less than we thought we knew a year ago. The painstaking follow-up of the Hiroshima and Nagaski survivors has provided our only extensive data on human neutron exposure. A joint Japanese–American effort has for more than three decades attempted to keep track of the medical history of every person within a few kilometers of Ground Zero who survived the cataclysm and its immediate aftermath in these two ill-fated cities.

The interpretation of the epidemiological data—mostly the occurrence and death rates for various kinds of cancer—in terms of absorbed neutron and gamma doses, has until recently been based on a 1965 estimate of the radiation doses as a function of distance from Ground Zero in the two cities. The dose–response curves derived from this generally accepted "T-65 dosimetry" (tentative 1965 dose estimates, producd by John Auxier at Oak Ridge) showed a striking difference between the two cities. Leukemia mortality rates in response to a given dose, for example, appeared to be much higher in Hiroshima than in Nagasaki. The difference was generally attributed to the much higher neutron component in the T-65 estimate of the Hiroshima radiation field, resulting from the different detonation mechanisms and casings of the two atomic bombs.

Last fall, however, William Loewe and Edgar Mendelson at Livermore made public a sharply revised estimate of the A-bomb dosimetries[1] that has thrown all conclusions based on T-65 into turmoil. Their transport calculations, starting from a 1976 Los Alamos calculation of the gamma and neutron spectra emerging from the bombs, conclude that the neutron doses at Hiroshima were an order of magnitude lower than previously believed. Gamma dose estimates for Hiroshima, on the other hand, are increased markedly in the Livermore paper.

Replotting the leukemia mortality dose–response curves, Loewe and Mendelsohn now find no difference between Hiroshima and Nagasaki. The new neutron dose estimates are now so low in both cities, they conclude, that one can derive only very limited information (from the leukemia data) about the relative biological effectiveness of neutron and gamma irradiation.



Leukemia mortality dose-response curves for Hiroshima and Nagasaki, **(a)** using the old T-65 dose estimates (adapted from Rossi and Mays, 1978), and **(b)** using the new Livermore dosimetry (Straume and Dobson, 1981). Striking difference between cities in **(a)** had been attributed to the higher neutron component at Hiroshima and very high relative biological effectiveness of neutrons. With the lower neutron dose estimates in **(b)**, the difference between cities appears to go away. Both fit well to a single linear-quadratic function out to 200 rads.

Tore Straume and Lowrie Dobson, also at Livermore, have however drawn some conclusions[2] about the carcinogenic effectiveness of neutrons by applying the new Loewe–Mendelsohn dose estimates to a more extensive body of cancer data for the Japanese survivors. Their conclusion, that neutrons may be orders of magnitude more carcinogenic than gammas at low doses for some forms of cancer, is disputed by those who contend that one can no longer draw any plausible inferences from so small a neutron component.

At a Munich symposium on neutron dosimetry, held in June, George Kerr reported[3] the results of his work with Joseph Pace at Oak Ridge, also reestimating the Hiroshima–Nagasaki dosimetry. Their technique and conclusions are quite similar to those of Loewe and Mendelsohn, but they argue that the Livermore group has overestimated somewhat the gamma radiation field at Hiroshima. This conclusion, based on a recent recalculation for gammas from the post-explosion fission clouds and on a disagreement about the total yield of the Hiroshima bomb, would spoil to some extent the identity of the dose–response curves for the two cities that Loewe and Mendelsohn had found for leukemia.

A week earlier Kerr had given a preliminary version of his results at a Minneapolis meeting sponsored by the National Council for Radiation Protection and the Radiation Research Society, at which Loewe and Auxier also spoke. Warren Sinclair, president of NCRP and chairman of the meeting, told us that most participants were agreed that it was unwise to set about revising radiation risk estimates before the new dosimetry was well tied down. "We've lost information about neutrons," he told us, "but we've gained information about the gamma risks, because the reduction of the neutron doses has brought the two cities into better agreement."

Last year the Biological Effects of Ionizing Radiation (BEIR) committee of the National Academy of Sciences fi-

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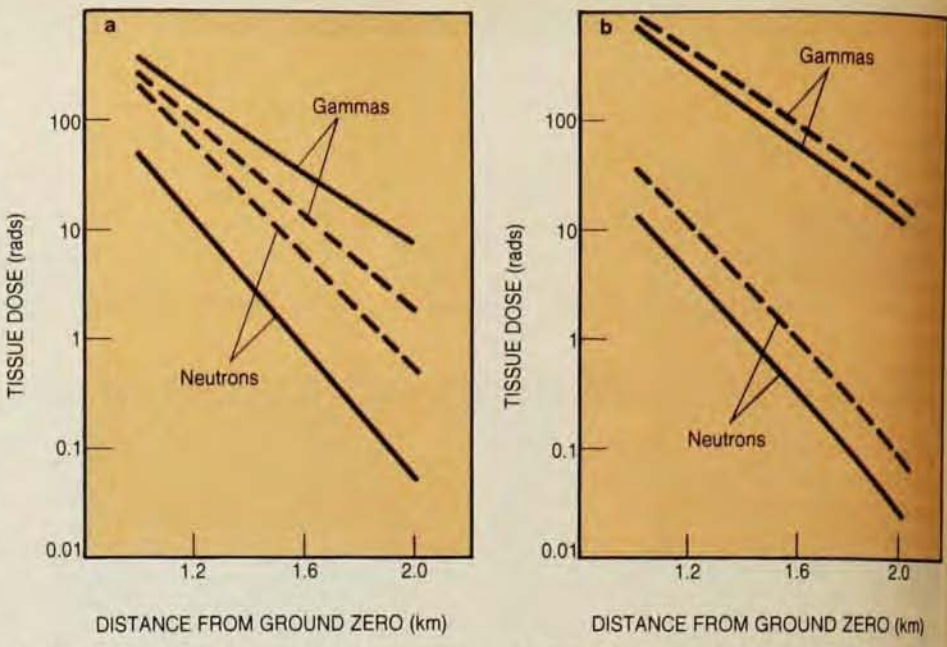
nally released a much disputed report (BEIR III) on the health risks of gamma radiation (PHYSICS TODAY, July 1979, page 78). The BEIR III committee did not have at its disposal the new A-bomb dosimetry estimates from Livermore or Oak Ridge. Although these new calculations have not yet laid to rest the hot contention over low-gamma-dose risks that split the BEIR III committee into three factions, it is widely agreed that the new gamma dose estimates have now brought the A-bomb data into better agreement with gamma exposure data from epidemiological studies of medically irradiated patients.

The Hiroshima bomb, whose unlikely code name "Little Boy" is still in use, was a uranium device whose detonation system was a massive steel gun assembly; a subcritical mass of U^{235} was fired down a gun barrel into another subcritical mass to initiate the fission blast. The Nagasaki plutonium bomb, code-named "Fat Man," was implosively detonated by a spherically symmetrical surrounding shell of high explosive. The high hydrogen content of the Fat Man implosion shell served to attenuate the fission neutron flux to such an extent that very few neutrons managed to emerge from the bomb. In Little Boy, by contrast, the fission neutron spectrum was softened (shifted to lower energy) by the steel assembly, but many more neutrons were able to escape with sufficient energy to survive more than a kilometer in air.

For purposes of studying long-term effects on survivors, one is concerned primarily with gamma and neutron fluxes at distances between one and two kilometers from the blast. Those unfortunate enough to be much closer in did not in general survive the *prompt* effects of the bomb; for distances beyond about two kilometers, long-term effects, if any, are difficult to ascertain. When Auxier was preparing his dose estimates for Hiroshima and Nagasaki in the early 1960's, computer techniques were not yet available to give reasonably reliable estimates of the neutron and gamma spectra emerging from the bombs. A number of devices of the Fat Man variety had been test-fired in the Nevada desert, but Little Boy was the only bomb of its type ever detonated.

Unlike the recent dosimetry estimates from Livermore and Oak Ridge, Auxier's empirical 1965 estimates were based neither on calculations of the bomb outputs nor transport calculations of the resulting radiation fields at distant points. In addition to limited weapons-test data, Auxier used a reactor mockup of Little Boy at Los Alamos to simulate the Hiroshima bomb's output, and he determined attenuation in air by measuring the distant radiation fields emanating from an unshielded uranium metal reactor sitting on a tower at the Nevada Test Site. Because the bare reactor output spectrum was not softened by a massive steel enclosure, Loewe told us, the Nevada measurements overestimated the fraction of neutrons surviving to large distances. Furthermore, neutrons traversing a kilometer or two of dry Nevada air suffer considerably less degradation than those passing through the humid atmosphere of the Japanese cities. These, Loewe contends, are the principal reasons for the overestimate of neutron doses for Hiroshima in the T-65 dosimetry.



Revised estimates of neutron and gamma doses absorbed by unshielded persons in Hiroshima **(a)** and Nagasaki **(b)**, as a function of distance from Ground Zero at the moment of the A-bomb explosion. Solid lines are 1981 estimates calculated by Livermore group. Dashed lines are previous standard (T-65) estimates produced by John Auxier in 1965. The most striking changes are reduced neutron estimate and increased gamma estimate for Hiroshima.

The immediate impetus for the Livermore reevaluation of the Hiroshima and Nagaski dosimetries was a disquieting 1978 paper by Harald Rossi (Columbia) and Charles Mays (University of Utah). Using the T-65 dose estimates, corrected for structural and body shielding to give radiation doses absorbed by bone marrow, Rossi and Mays produced dose–response curves for leukemia mortality among the survivors in both cities. (A dose–response curve is a plot of the rate of occurrence of some biological effect as a function of absorbed radiation dose). From the striking difference between the Hiroshima and Nagasaki curves thus produced, they sought to deduce the relative biological effectiveness (RBE) of neutrons as a function of dose. Particles such as neutrons and alphas, which produce densely ionizing tracks in tissue, can cause more cell damage than gammas, and the neutron RBE coefficient is the neutron dose divided *into* the gamma dose that would be required to elicit the same biological response.

Assuming that the small Nagasaki neutron component had a negligible effect on leukemia mortality, Rossi and Mays calculated from the difference between the two dose–response curves that the neutron RBE was as high as *sixty* at low doses. That is, a 1-rad dose of neutrons would cause as much leukemia as 60 rads of gamma irradiation. Although this result was consistent with the "dual action theory" of radiation effects developed by Rossi and Albrecht Kellerer in the early 1970's, it was the first human epidemiological study to present evidence for such enormous neutron RBEs.

As far as environmental risks to the general public are concerned, gamma radiation is much more important than neutrons. But the high RBE estimates of Rossi and Mays were troubling for people working around reactors or nuclear-weapons production facilities. Because of Livermore's involvement with the weapons program, Loewe and Mendelsohn were asked to review the T-65 dosimetry on which the Rossi–Mays work had been based. Although Kerr and Pace at Oak Ridge had begun a similar review in 1977, shortly after the new Los Alamos bomb-output calculations became available, neither group was aware of the other's effort until shortly before the results of Loewe and Mendelsohn were made public a year ago.

Transport calculations. Both groups took the Los Alamos output spectra for Fat Man and Little Boy as the starting points for their transport calculations. Only about a tenth of the gammas encountered a kilometer or so from Ground Zero came directly from the

bombs. The bulk of the gammas come in roughly equal measure from neutron capture reactions in air and from the decay of fission products in the fireball rising from the blast. Loewe and Mendelsohn used a Monte Carlo computer code to follow individual hypothetical neutrons and prompt and neutron-capture gammas as they were scattered and attenuated in the air and ground. Because of the high attenuation rate, the Monte Carlo calculation yields good statistics out to only about a kilometer if one starts with a manageable number of hypothetical particles at Ground Zero. To solve the Boltzmann transport equation for distances beyond 1 km, the Livermore group used a "discrete-ordinate transport" (DOT) program developed at Oak Ridge. The DOT calculations treat the transported flux in discrete bins of energy and angle, instead of following each individual particle through thousands of scatterings. The good agreement between their Monte Carlo and DOT results, Loewe told us, contributed much to his confidence in the correctness of the Livermore results when it became clear they were in strong disagreement with the standard T-65 dosimetry.

Having concluded that the results of their transport calculations were also in good agreement with neutron-activation and thermoluminescence measurements of building materials from the Japanese cities, and with recent dose measurements in liquid air using a neutron source at Livermore, Loewe and Mendelsohn had sufficient confidence in their surprising new dosimetry to make it public last September. The results of the Oak Ridge transport calculations, which used only the DOT program, were confined to private reports (to DOE and the National Council on Radiation Protection) until Kerr's presentations in Minneapolis and Munich a few months ago.

The new dosimetry arrived at by both groups gives only about one-tenth the neutron dose estimated by T-65 at 2 km from the Hiroshima Ground Zero. At 1 km, it deflates the T-65 neutron dose by a factor of only five. For Nagasaki, the new estimates reduce the already much smaller neutron doses by about a factor of three. Thus the only two nuclear weapons ever fired in earnest no longer appear to be so generous a source of information about the long-term health hazards of neutron irradiation.

Livermore and Oak Ridge are in agreement that Auxier's estimate of the Nagasaki gamma doses must be reduced by about 30%—"not a very significant change," Loewe told us. A bigger revision is the Livermore quadrupling of the T-65 gamma dose estimates for Hiroshima at 2 km; at 1 km it is only doubled. Although Kerr agrees that Auxier's Hiroshima gamma doses were much too low, he contends that Livermore has now gone too far—overestimating the gamma doses by about 25%.

Both groups have relied on outside sources for estimates of the very significant decay gamma component from the fission fireball. The gamma flux from this maelstrom of radioactive fission products rising out of the blast is very difficult to calculate. Livermore used a 1966 phenomenological model (based on earlier work of Loewe) confined to classified Defense Department manuals. Kerr's lower gamma dose estimate results from a recent calculation of the fireball component by William Scott of Science Applications Inc (San Diego), and from a lower estimate of Little Boy's total yield. Scott is continuing to refine this fireball calculation, and Loewe agrees that a thorough new look at the fireball output has been long overdue.

Radiation risk implications. The new Livermore dosimetry wipes out the difference between the Hiroshima and Nagasaki dose–response curves for leukemia mortality—previously attributed to a significant neutron component at Hiroshima, with very high neutron RBE at low doses. The best estimate of Loewe and Mendelsohn for the neutron RBE (for leukemia) is close to unity, but with a large uncertainty because the new neutron dose estimates are so small. With their somewhat lower estimates for gammas at Hiroshima, the Oak Ridge group does not find that the leukemia data for the two cities fall so nicely on a single dose–response curve. Kerr argues that it is too early to speculate on neutron RBEs from the still tentative data.

A serious three-way dispute arose in the BEIR III committee over the form of the dose–response curves for cancer risk at low gamma doses. A linear response curve without threshold, as advocated by Chairman Edward Radford, a University of Pittsburgh epidemiologist, enhances the risk estimate for low gamma exposure. Rossi argued for a quadratic fit to the existing dose–response data (prior to the new Livermore and Oak Ridge dosimetries), implying smaller low-dose risk. The final BEIR III report last year, from which both Radford and Rossi dissented, used a compromise linear–quadratic fit (of the form $ax^2 + bx + c$).

From the new Livermore dosimetry, Dobson and Straume have now produced new dose–response curves for leukemia, breast cancer, total cancer mortality and chromosomal aberrations. The result is a somewhat perplexing mixed bag. For leukemia and breast cancer mortality, they find that the data for the two cities coincide, fitting well to linear–quadratic dose–response curves that assume no threshold "safe" dosages. Because the Hiroshima neutron doses, albeit small, are still much larger than those for Nagasaki, the coincidence of the dose–response curves for the two cities lead Dobson and Straume to conclude that neutron RBEs for leukemia and breast cancer are close to unity.

For total cancer mortality and chromosome aberrations, on the other hand, Dobson and Straume find that the Hiroshima responses lie well above those for Nagasaki at a given dose level. If one ascribes the remaining differences between the two cities to neutrons, they calculate, much as Rossi and Mays had done earlier, that neutron RBEs would be of the order of 100 for chromosome aberrations and malignancies other than leukemia and breast cancer at low neutron dose levels. Radford argues that one is not justified in calculating RBEs from the low neutron doses and death rates involved here; there were only 20 Nagasaki leukemia deaths in the low-dose bins, he stresses. Nonetheless, the human RBE estimates produced by Dobson and Straume do in fact agree well with a number of studies of animals and human tissue (*in vitro*) irradiated with neutrons.

Mortimer Mendelsohn (no relation to Edgar), associate director of Livermore for environmental and biomedical research, told us that Dobson and Straume have suggested a possible explanation for the striking differences observed for different malignancies. There is evidence, he points out, that leukemia and breast cancer are of viral origin. For virally induced cancers, it is plausible that malignancies are triggered by a "one-hit mechanism," the interaction of a single ionizing particle with a virus. For such a mechanism, he explained, a given dose of gammas and neutrons would be about equally effective—giving an RBE near unity. On the other hand, forms of cancer induced by chromosomal aberrations would require a number of adjacent hits. It is known that single ionizing lesions do not break chromosomes; both DNA strands must be severed. Whereas this would require several gamma hits, a single neutron, generating multiple ionizing tracks, could do the trick by itself. Thus cancers produced by radiation-induced chromosome damage could be expected to exhibit very high neutron RBEs at low dose levels, he argues.

There is a considerable body of data on human cancer induction by gamma and x-ray sources other than the atomic bombs—mostly from studies of patients exposed in various medical procedures. With the old Auxier dosimetry, Mendelsohn told us, the response rates of the Japanese survivors had appeared anomalously low. The

new dosimetries appear to have closed the gap between the two data sets. Risk coefficients (death rates per rad) calculated by Dobson and Straume for gamma-induced leukemia and breast cancer are consistent at low doses with those promulgated in 1977 by the International Commission on Radiological Protection. At high gamma doses, however, the leukemia risk coefficients calculated at Livermore are four times as large as the dose-independent ICRP risk factor. For total cancer mortality, on the other hand, the new gamma radiation risk coefficients are lower than the ICRP value.

It is generally agreed that more work needs to be done before the new atomic-bomb dosimetries can provide a firm basis for a revised set of radiation-risk standards. Uncertainties remain with respect to the total yield of the Hiroshima bomb and the gamma doses coming from fission products in the fireballs. New estimates of structural and body shielding are yet to be calculated for the revised radiation spectra.

Dissenting from this cautious consensus, Radford argues that too much emphasis has been put on the leukemia and other mortality data, leading to underestimated gamma risks. The "more reliable" data on total cancer incidence (as distinguished from mortality), he contends, remove all differences between the two cities (without reference to neutron RBEs) and lend strong support to his belief that the dose–response functions are indeed linear. —BMS

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Reversed-field pinch stable 8 msec

Tokamaks are certainly in the vanguard of the quest for plasma ignition in magnetic-confinement fusion experiments. Nevertheless, the persistent concern that technological difficulties may ultimately render tokamaks of present-day design impractical as power reactors has encouraged a broad search for alternative magnetic-confinement concepts. One such alternative, the reversed field pinch (RFP), is an axisymmetric toroidal scheme very much like the tokamak. But the differences between the two could eventually prove to be of considerable practical significance.

A few months ago, the ZT-40 reversed-field pinch machine at Los Alamos experienced a dramatic hundredfold increase of its period of plasma stability immediately after its ceramic vacuum liner was replaced by a metallic liner. With this change, suggested by the earlier successes of the smaller "Eta–Beta II" machine in Padua (Italy), the ZT-40 has now achieved quiescent stability periods of 8 milliseconds—a world's record for RFPs. During these quiescent periods, the magnetic field at the edge of the plasma appears to maintain itself in the characteristic "reversed" configuration upon which stability depends in such devices.

With the successes achieved at Los Alamos, and similarly encouraging results coming from RFP experiments at Padua, Culham (England) and Tokyo, plans are afoot for the next generation RFP experiment. In July, the DOE Office of Fusion Energy convened a technical review panel to consider the proposal that the US become a full participant in the British RFX experiment. The RFX, currently being designed at Culham in collaboration with the Los Alamos and Padua groups, will be a reversed-field-pinch torus with three times the linear dimensions and plasma-current capacity of the ZT-40. The DOE would not at present consider an RFP of such size—estimated to cost about $40 million—as a purely American undertaking. Among the alternatives to tokamaks and mirrors considered by the DOE Alternate Concept Review Committee two years ago, only the Elmo Bumpy Torus was recommended for funding at the level of a proof-of-principle experiment (PHYSICS TODAY, October 1979, page 18). The RFP was however the Committee's clear second choice among nine alternative concepts.

The principal differences between the tokamak and the RFP are the magnitude and profile of the toroidal component of the magnetic field and the magnitude of the plasma current. The toroidal field component B_ϕ, running the long way around the torus, is primarily generated in either machine by the current in the field coils wrapped poloidally (the short way) around the torus. The poloidal field component B_θ in both schemes is not generated directly by external coils, but rather by a toroidal current introduced in the plasma itself by transformer action. Because the plasma current in the RFP is much stronger than in a comparable tokamak, it also contributes significantly to the toroidal field, producing a strong variation of B_ϕ with distance from the center of the plasma.

The tokamak achieves plasma stability by having B_ϕ so large relative to B_θ that magnetohydrodynamic kink instabilities cannot fit into the torus. The larger the poloidal field component becomes relative to the toroidal component, the more helically twisted are the magnetic field lines as they travel around the torus. The requirement of tokamak stability imposes a strict limit on the degree of helical twisting; the field lines must traverse the torus at least once the long way before they complete a twist the short way. This is expressed by the Kruskal–Shafranov constraint: $q > 1$, where $q = (B_\phi/B_\theta)/A$, and A is the "aspect ratio," the major radius of the torus divided by its minor radius.

Thus, for a given toroidal field strength the Kruskal–Shafranov constraint severely limits the magnitude of the plasma current that generates the poloidal field. This is perhaps unfortunate, because the ohmic heating generated by the plasma current would be the simplest way of heating the plasma to ignition temperature. The higher the plasma current, however, the greater is the toroidal field required to satisfy the Kruskal-Shafronov limit. But the larger the toroidal field, the greater is the cost of the machine and the magnetic stress on the coils.

The RFP simply ignores the Kruskal-Shafranov limit and the problems it presents. Not only does it operate stably with q less than one, q actually goes *negative* at the plasma edge. That is what's meant by field reversal: B_ϕ goes through zero and changes direction as one goes out from the center to the edges of the plasma. This field reversal is one of the two tricks that lets the RFP get away with violating the Kruskal–Shafranov limit. It turns out that the field-reversed configuration is a minimum-energy state of the plasma-field system, stable against localized MHD instabilities. The other trick is the presence of a thick conducting shell placed just outside the vacuum liner. Image currents generated in this shell stabilize the plasma against larger-scale MHD modes. A tokamak, operating at $q > 1$, does not require such a conducting shell.

Circumventing the Kruskal–Shafranov limit, the RFP operates with a very modest toroidal field and very high plasma current. The strong poloidal field generated by the plasma current plus the plasma-generated variations in the toroidal field result in a field configuration of very high "shear." That is to say, the pitch of the helical field lines increases very rapidly as one goes from the center to the surface of the plasma. It is this high shear, culminating in the actual reversal of the pitch (hence the reversal of