N. Allen, P. S. Spencer, D. O. Carpenter, *Neuroscience* **54**, 5677 (1993).
8. P. S. Spencer *et al.*, *Science* **237**, 517 (1987).
9. J. D. Rothstein, L. Jin, M. Dykes-Hoberg, R. W. Kuncl, *Proc. Natl. Acad. Sci. U.S.A.*, in press.
10. P. Couratier, J. Hugon, P. Sindou, J. M. Vallat, M. Dumas, *Lancet* **341**, 265 (1993).
11. D. K. Dastur, *Fed. Proc.* **23**, 1368 (1964); ______, R. S. Palkar, D. K. Manghani, in *ALS: New Advances in Toxicology and Epidemiology*, F. C. Ross and F. H. Norris, Eds. (Smith-Gordon, London, 1990), pp. 129–144.
12. R. M. Garruto, R. Yanagihara, D. C. Gajdusek, *Lancet* **ii**, 1079 (1988); R. M. Garruto, M. J. Strong, R. Yanagihara, *Adv. Neurol.* **56**, 327 (1991).

There has been a long-standing but unsuccessful search for an environmental cause of ALS-PDC not only on Guam but also in two other places with a high incidence of ALS-PDC, the Hobara and Kozagawa regions of the Kii Peninsula on Honshu Island in Japan and the Auyu and Jakai villages of West Irian in New Guinea. ALS-PDC appeared in these culturally different and relatively isolated regions at about the same time as it did in Guam, and its incidence then steadily declined. The pathology of ALS-PDC shows that it is distinctly different from classical ALS and Alzheimer's disease. Neurofibrillary tangles are not present in ALS, and while they are present in Alzheimer's disease, their distribution is different from that in ALS-PDC (*1*). Senile plaques, sparing of the globus pallidus, and clinical differences (*2*) also distinguish Alzheimer's disease from ALS-PDC. However, the clinical features and pathology of ALS-PDC strongly resemble postencephalitic parkinsonism–ALS, a sequel of encephalitis lethargica first described by Constantin von Economo in 1917 (*3*).

Encephalitis lethargica accompanied the swine influenza pandemic in the 1920s, peaking in 1920 and 1924, and disappeared at the end of the decade. Because of their coincidence they were believed to be related, but this remained unproved. However, postencephalitic parkinsonism–ALS continued to appear over the next three decades. Guamanian ALS-PDC was first recognized in 1947 and was initially thought to be a late sequel of encephalitis lethargica, but attention soon focused on environmental toxins. It is probable that small epidemics of encephalitis lethargica have recurred for centuries, each having been referred to with different names, such as "febrilis comatosa" and "La Nona" (*4*). We may now be observing the decline of a postinfective illness that has finally run its course on Guam, West Irian, and the Kii Peninsula. The genetic isolation of island, valley, and tribal peoples may have produced a marked susceptibility to disease that has far more genetic importance than we realize.

Arthur J. Hudson
University Hospital,
339 Windermere Road,
London, Ontario, N6A 5A5,
Canada

References

1. A. J. Hudson and G. P. A. Rice, *Can. J. Neurol. Sci.* **17**, 427 (1990).
2. A. J. Hudson, *ibid.* **18**, 387 (1991).
3. C. von Economo, *Encephalitis: Its Sequelae and Treatment* (Oxford Univ. Press, London, 1931).
4. *Brit. Med. J.* **1**, 748 (29 March 1890).

Stone's article on Guam disease concentrates on the search for a toxic agent or agents as a cause for ALS-PDC, but there is suggestive evidence—epidemiological, pathological, and clinical—that Guam disease may be due to a transmissible agent, probably a virus. There are many affinities between Guam disease and postencephalitic syndromes. Encephalitis lethargica, after raging for some years, also vanished and apparently died out completely around 1927. It too could produce neurological syndromes many years or decades after the original infection—the longest such interval in my experience was 45 years. And these syndromes could be extremely variable—some patients presented ALS-like syndromes, some amnesia, and a great many parkinsonism, all of which are common in Guam disease. Moreover, as I saw myself during a recent visit to Guam, ALS-PDC can also present as catatonia, ticcing, or arousal syndromes intensely sensitive to the "awakening" effects of L-dopa, precisely as in postencephalitic patients.

It is difficult to imagine a toxin having effects so varied, so unpredictable, so delayed; it is much more plausible to conceive of an infectious agent, with perhaps an animal reservoir or vector that was destroyed or altered around the time of World War II.

It is crucially important, as Stone points out, that this unique disease be cracked before it disappears, for even if its etiology is different, it could cast a flood of light on every sort of neurodegenerative disease and process. And it is especially important that the current research on Guam itself be properly funded and encouraged, for this is where we will find the answer.

Oliver W. Sacks
Department of Neurology,
Albert Einstein College of Medicine,
c/o 299 West 12th Street, #14C,
New York, NY 10014

■

Quantum Mechanics: Not Mysterious

I write to disagree fundamentally with the 12 March article by David H. Freedman (Research News, p. 1542). Leaving aside the discomfort I feel with language like "mysterious," "queerness," and "somehow [collapsing] into an ordinary particle . . . ," I focus on a proposed experiment using a "Stern-Gerlach (SG) apparatus" (*1*).

"[T]he SG experiment epitomizes the quantum mechanical description of microscopic phenomena" (*2*). The famous SG experiment featured a beam of magnetic atoms (spin 1/2) running through a transverse, inhomogeneous magnetic field, resulting in the splitting of the beam into two components. When the field inhomogeneity is sufficiently strong that the two emerging components do not overlap, one has made a measurement of the transverse component of magnetic moment. With weakening field gradient, from the onset of overlap to undetectable broadening of the beam width, there is no act of measurement.

Consider feeding a single beam from one SG apparatus into a second one that has the direction of the inhomogeneity rotated by the angle θ relative to the first one. Geometrical intuition correctly suggests that the average spin emerging from the second apparatus is provided by projection of the initial output, as expressed by the factor $\cos\theta$. Then the fractions that emerge in the two beams are

$$\frac{1}{2}(1 + \cos\theta) = \left(\cos\frac{1}{2}\theta\right)^2$$

and

$$\frac{1}{2}(1 - \cos\theta) = \left(\sin\frac{1}{2}\theta\right)^2$$

These fractions are consistently interchanged when the directions of the beams are interchanged through the substitution $\theta \leftarrow\rightarrow \pi - \theta$.

One sees the emergence of fractions—probabilities—as squares of more fundamental quantities: probability amplitudes. I use this designation, rather than "wave" or wave function, because the latter invite misleading analogies with classical waves and classical determinism. In contrast, probability amplitude makes no reference to individual acts, which are generally unpredictable, but recognizes that microscopic phenomena are statistically predictable.

According to Freedman's account, in the proposed SG experiment, the inhomogeneity of the transverse "magnetic field is kept weak." I interpret this to mean that no SG measurement is performed, but a detectable broadening of the beam can occur. Then comes the new ingredient: a strong homogeneous field along a different direction. That certainly acts to suppress the effect of the inhomogeneous field, conceivably to the point that no beam broadening remains. Yet the claim is made that a slight deflection of the whole beam should be observable. I prefer to leave that as a moot point, because it is irrelevant.

Omitted from this description is a much more important phenomenon: the longitudinal SG effect (*2*). When a magnetic atom enters a region of homogeneous magnetic field, thereby acquiring an additional positive, or negative, potential energy, its kinetic energy correspondingly decreases, or increases, to maintain the total energy.

This slowing down, or speeding up, of individual atoms produces a splitting of the beam in time, rather than space. A magnetic moment measurement is realized when the ratio of magnetic energy to kinetic energy is sufficiently large that no overlap of the outgoing beams occurs. The extreme example appears when one type of atom is brought to rest and sent into retreat, while the second type emerges from the other side. Here, indeed, is the ultimate clash between the two concepts. Tenets anyone?

Julian Schwinger
University of California,
405 Hilgard Avenue,
Los Angeles, CA 90024

References

1. Y. Aharonov, J. Anandan, L. Vaidman, *Phys. Rev. A*. 47, 4616 (1993).
2. J. Schwinger *et al.*, *Z. Phys.* **D10**, 135 (1988).

Response: Schwinger's disagreement with Freedman's article in the preceding letter is irrelevant to the proposed "protective" Stern-Gerlach (SG) experiment (*1*). The inhomogeneous magnetic field here is the Stern-Gerlach field with the usual strength, and not a weak field such that "no SG measurement is performed," as stated by Schwinger. This ordinarily splits the wave function of a neutral spin-1/2 atom into two wave functions with opposite spins. But if the spin state is "protected" by a homogeneous magnetic field (which is large *compared* with the inhomogeneous magnetic field) in the unknown direction of the spin, then the wave function would not split. Even though the inhomogeneous field is small compared with the homogeneous field, it is large enough to cause a shift in the position of the center of mass of the particle, which is larger than its quantum spread. By observing this shift for three such experiments, one can determine the spin state for a single particle, instead of having to use an ensemble.

We also studied (*1*) the longitudinal SG experiment that Schwinger mentions. Here also when the spin state is protected the wave function does not split, as ordinarily happens. By observing the displacements of the wave packet for three such experiments, one can reconstruct the spin state. In both experiments the spin state would be determined in a nonstatistical way for a single particle.

These proposed experiments are special cases of a general scheme to measure the wave function of a *single* particle "protectively," as shown (*1*). And this *is* allowed by the laws of quantum theory. But it provides a new meaning to the wave function that seems to go beyond the usual meaning of the "probability amplitude" used by Schwinger.

Y. Aharonov
J. Anandan
Department of Physics,
University of South Carolina,
Columbia SC 29208

References

1. Y. Aharonov, J. Anandan, L. Vaidman, *Phys. Rev. A* 47, 4616 (1993).

Security of E-Mail

Users of e-mail as well as publishers of articles or data in bulletin boards should be aware that the information they are transmitting is not secure. This can affect, indeed destroy for most of the countries of the world, the rights of the transmitter (and of the transmitter's employer) to obtain patent protection. Indeed, the transmitter, if transmitting without the knowing consent of the employer to the specific information being transmitted (as for any publication), may be breaching provisions that appear in a great number of employment contracts. This is true of university employment contracts to a somewhat lesser extent than industry employment contracts. The e-mail user may not be risking as much as the bulletin board user because an e-mail message, while it is no doubt stored at least temporarily at each station from which it is forwarded, may not be accessible to the public. However, there have been no decisions of which I am aware in any country on this point.

Hubert E. Dubb
Chair,
Committee on Patents and Related Matters,
American Chemical Society,
c/o Four Embarcadero Center, Suite 400,
San Francisco, CA 94111–4156

Corrections and Clarifications

In Robert K. Englund's review of *Before Writing* by Denise Schmandt-Besserat (University of Texas Press, Austin, 1992) in the 11 June issue (p. 1670), two cuneiform characters in columns 1 and 2 on page 1671 were misrepresented. The correct characters are as follows.