Correction. In the article "Nucleotide sequence and the encoded amino acids of human serum albumin mRNA" by Achilles Dugaiczyk, Simon W. Law, and Olivia E. Dennison, which appeared in the January 1982 issue of *Proc. Natl. Acad. Sci. USA* (**79**, 71–75), the first paragraph of *Discussion* was garbled by a printer's error. The correct paragraph is printed below.

Determining the complete nucleotide sequence of the cDNA has permitted us to identify the pre- and the propeptides of human serum albumin. Actually, the amino acid sequence of the propeptide has been reported for carriers of an abnormal albumin, Christchurch (18), which is longer than normal human albumin by six amino acids at the NH_2 terminus. This additional hexapeptide has the sequence Arg-Gly-Val-Phe-Arg-Gln (18) and differs only in the terminal position from the sequence we are presently reporting for the apparently normal protein, Arg-Gly-Val-Phe-Arg-Arg-albumin (Fig. 3). Thus, carriers of albumin Christchurch must be carriers of a CGA to CAA mutation, which changes the codon for Arg to Gln in the last position of the propeptide. The altered protein consequently ceases to be a substrate for the specific protease that removes propeptides from secretory proteins. It is interesting to note, however, that failure to remove such a propeptide does not prevent the protein from being secreted; at least this is true about albumin Christchurch, which has reached the bloodstream of its carriers (18).

Correction. In the article "A proton gradient controls a calcium-release channel in sarcoplasmic reticulum" by Varda Shoshan, David H. MacLennan, and Donald S. Wood, which appeared in the August 1981 issue of *Proc. Natl. Acad. Sci. USA* (**78**, 4828–4832), the authors request that the following correction be noted. In the experiment reported in Fig. 5 and discussed on pages 4830 and 4831, the concentration of Ca^{2+} used to inhibit Ca^{2+} release was, in fact, 100 μM not 3.3 μM as reported. Consequently, although Ca^{2+} release was measurably reduced by Ca^{2+} in the experiments of Fig. 5, the data neither support nor preclude the possibility that physiological Ca^{2+} levels ($\leq$10 μM) can inhibit Ca^{2+} release.