Scheme I

Figure 2. Photochemical isophorone deconjugation at various concentrations of acetic acid: [isophorone] = 0.145 M.
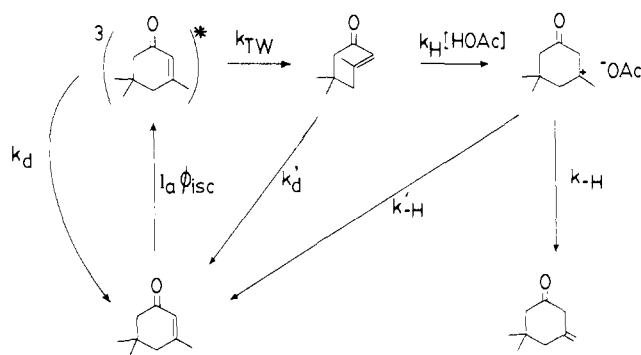
The quantum yield of deconjugation of isophorone solutions containing 4×10^{-3} M acetic acid showed a systematic decline from 0.044 to 0.035 when the isophorone concentration was gradually increased from 0.1 to 1 M. This is inconsistent with a mechanism of deconjugation which is bimolecular in isophorone but is attributable to self quenching of isophorone triplets by ground-state isophorone.[8]

The photochemical deconjugation reaction is reported to fail for 3-alkylcyclohexenones if a 3-methyl group is replaced by larger alkyl groups.[4b] However, we find that if 4×10^{-3} M acetic acid is present, then the reaction does proceed with 5,5-dimethyl-3-ethylcyclohexenone to give a mixture of (*E*)- and (*Z*)-**3**; **4** is also formed as a secondary (Norrish Type I) photoproduct at high conversions.
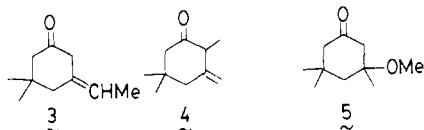
The simplest explanation of our observations is that the carbon–carbon double bond of the triplet excited state of isophorone is protonated by the acid with Markovnikov regiochemistry to give a carbocation which eliminates to isophorone and the deconjugated product. However, this would require that interception of the triplet with 4×10^{-3} M acetic acid be much more efficient than triplet decay which would require a rate constant for protonation of the order of 10^{12} M^{-1} s^{-1} by acetic acid in benzene. Clearly a longer lived intermediate formed from the triplet is required for reaction with the acid. By analogy with the acid-catalyzed photochemical reactions of cyclohexenes[9] and the results of flash photolysis studies of the reaction of 4,4-dimethylcyclohexenone with alkenes[10] we suggest that the intermediate is a ground-state twisted cyclohexenone (a *trans*-cyclohexenone). This gives rise to the mechanism shown in Scheme I which in turn leads to eq 3 for the dependency of the deconjugation quantum yield, Φ_p, upon

$$\frac{1}{\Phi_p} = K\left\{1 + \frac{k_d'}{k_H[\text{HOAc}]}\right\} \quad (3)$$

where $\dfrac{1}{K} = \Phi_{isc}\left\{\dfrac{k_{-H}}{k_{-H} + k_{-H'}}\right\}\left\{\dfrac{k_{TW}}{k_d + k_{TW}}\right\}$

acid concentration.[11] The fit of the data from Figure 1 to eq 3 is shown in Figure 2. From the slope and the intercept of the plot the value of k_d'/k_H can be determined and is 1.9×10^{-4} M. The fastest value k_H can adopt is the rate of diffusion (1.5×10^{10} M^{-1} s^{-1} in benzene); and hence k_d' must be $\lesssim \sim 10^6$ s^{-1}. This corresponds to an intermediate with a lifetime greater than a
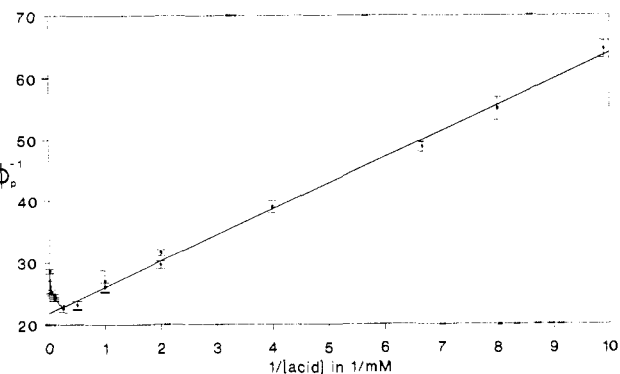
microsecond which compares with 9 µs for *trans*-1-phenylcyclohexene.[9a]

If a highly strained twisted enone or a carbocation are intermediates in the reaction, then they should be trappable by nucleophiles.[12] Indeed, irradiation of isophorone in benzene containing 10% methanol gave a mixture of deconjugated product **2** and the anticipated methyl ether **5** in the ratio 3:1.

Our observation of acid catalysis suggests that the previously reported[4,5] results for the photochemical deconjugation reaction of cyclohexenones need to be reexamined. In addition, our evidence for a twisted intermediate may have consequence for the other reactions of cyclohexenones such as rearrangement and cycloaddition where a similar intermediate has also been proposed;[10] the observation of acid catalysis may also be of relevance to the understanding of the mechanism of photochemical addition of nucleophiles to cyclohexenones and larger ring cycloenones.[12]

(12) (a) Noyori, R.; Wanatabe, A.; Kato, M. *Tetrahedron Lett.* **1968**, 5443. (b) Majeti, S.; Gibson, T. W. *Tetrahedron Lett.* **1973**, 4889. (c) Dunkelblum, E.; Hart, H.; Jeffares, M. *J. Org. Chem.* **1978**, *43*, 3409. (d) Hart, H.; Dunkelblum, E. *J. Am. Chem. Soc.* **1978**, *100*, 5141. (e) Hart, H.; Chen, B.; Jeffares, M. *J. Org. Chem.* **1979**, *44*, 2722. (f) Cornell, D. G.; Avram, E.; Filipescu, N. *Steroids* **1979**, *33*, 485 (CA 42226). (g) d'Angelo, J.; Ortuno, R.-M.; Brault, J.-F.; Ficini, J.; Riche, C.; Bouchaudy, J.-F. *Tetrahedron Lett.* **1983**, *24*, 1489. (h) Sedee, A. G. J.; Beijersbergen van Henegouwen, G. M. J.; Guijt, W.; Haasnoot, C. A. G. *Pharm. Weekbl. Sci. Ed.* **1985**, *7*, 202.

Long-Range Electron Transfer in Structurally Engineered Pentaammineruthenium (Histidine-62)cytochrome *c*

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In many biological processes, long-range electron transfer (ET) plays a key role.[1] When the three-dimensional structures of proteins are accurately known, use of modified proteins[1a,2] and protein–protein complexes[3,4] provides an experimental approach

(9) (a) Bonneau, R.; Joussot-Dubien, J.; Salem, L.; Yarwood, A. J. *J. Am. Chem. Soc.* **1976**, *98*, 4329. (b) Herz, J. E.; Gonzalez, E. *Steroids* **1968**, *12*, 551. (c) Dauben, W. G.; van Riel, H. C. H. A.; Robbins, J. D.; Wagner, G. J. *J. Am. Chem. Soc.* **1979**, *101*, 6383.
(10) Schuster, D. I.; Brown, P. B.; Capponi, L. J.; Rhodes, C. A.; Scaiano, J. C.; Tucker, P. C.; Weir, D. *J. Am. Chem. Soc.* **1987**, *109*, 2533.
(11) A discussion of the kinetics of the acid-catalyzed photochemical reactions can be found in ref 9 and also in the following: Wubbels, G. G. *Acc. Chem. Res.* **1983**, *16*, 285.

(1) (a) Gray, H. B.; Malmström, B. G. *Biochemistry* **1989**, *28*, 7499-7505. (b) Marcus, R. A.; Sutin, N. *Biochim. Biophys. Acta* **1985**, *811*, 265-322. (c) Hatefi, Y. *Annu. Rev. Biochem.* **1985**, *54*, 1015-1069. (d) Dixit, B. P. S. N.; Vanderkooi, J. M. *Curr. Top. Bioenerg.* **1984**, *13*, 159-202.
(2) (a) Gray, H. B. *Chem. Soc. Rev.* **1986**, *15*, 17-30. (b) Mayo, S. L.; Ellis, W. R., Jr.; Crutchley, R. J.; Gray, H. B. *Science (Washington, D.C.)* **1986**, *233*, 948-952. (c) Scott, R. A.; Mauk, A. G.; Gray, H. B. *J. Chem. Educ.* **1985**, *62*, 932-938.

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Communications to the Editor

to study ET rates between two metal centers. For Ru(His)-modified proteins, the introduction of histidine residues at any desired surface location by site-directed mutagenesis opens the way for systematic investigations of ET pathways.[2b]

Some studies suggest that long-range donor–acceptor electronic coupling can be enhanced by aromatic groups or sulfur atoms in the intervening protein medium.[5,6] To examine this possibility, we created a yeast cytochrome *c* variant with a surface histidine at residue 62 by preparing the N62H mutant.[7] Computer modeling of the ruthenated mutant, whose structure should be virtually identical[8] with that of the native protein determined by X-ray diffraction,[9] shows that the putative pathway for electron transfer to the heme iron contains both an aromatic residue (Trp59) and a sulfur of methionine (Met62).[10]

The N62H mutant was prepared by oligonucleotide-directed mutagenesis[11] on the yeast iso-1-cytochrome *c* gene (CYC1) cloned into M13mp8 viral DNA.[12,13] The 17-mer d(ACATGTT-*ATG*TTCGTCC) was used to direct the change of the AAT codon for asparagine to the CAT codon for histidine at position 62 in the iso-1-cytochrome *c* gene. Individual viral colonies were screened for the gene mutation by the dot blot method,[14] and the base substitution was confirmed by dideoxy sequencing.[15] The
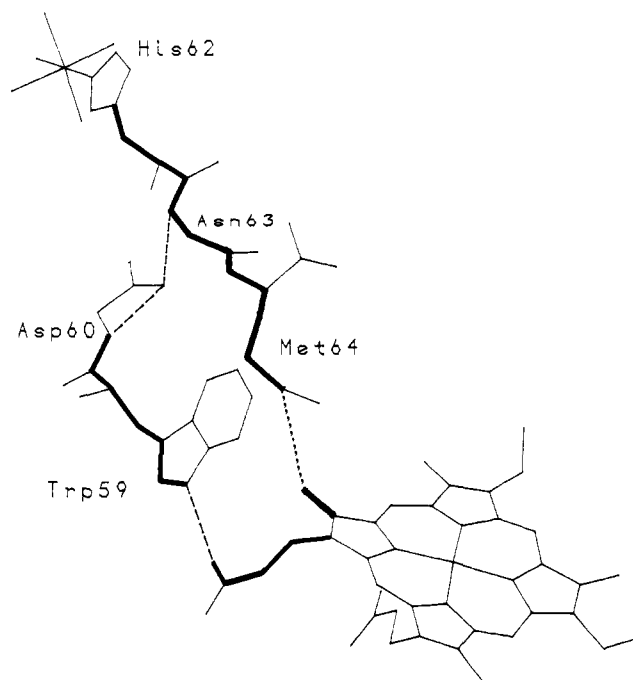

Figure 1. The ET medium between ruthenated His62 and the heme, derived from side-chain replacement and molecular mechanics minimization of the native yeast iso-1-cytochrome *c* structure.[10] For clarity, the iron axial ligands, His18 and Met80, have been omitted, as have the Asn63 and Asp60 side chains. The two best Beratan–Onuchic pathways[30] are shown in bold. Long dashed lines (---) denote hydrogen bonds, and short dashed lines (-·-) are through-space interactions. Calculations according to ref 29 give $[H_{ab(His62-Met64)}/H_{ab(His33-Pro30)}]^2 = 0.013$ and $[H_{ab(His62-Trp59)}/H_{ab(His33-Pro30)}]^2 = 0.0092$.

mutant CYC1 gene was prepared for expression in yeast by the method of Smith and co-workers.[12b] The N62H mutant was excised from the M13RF DNA and inserted into the yeast expression vector, YEp213 (contains ampicillin and tetracycline resistance genes and the Leu2+ gene from *Saccharomyces cerevisiae* as markers),[16] to produce the YEp213/CYC1(N62H) hybrid plasmid.[17] This plasmid was then transformed into the *S. cerevisiae* strain GM-3C-2[18] (a yeast strain deficient in both production of cytochrome *c* and biosynthesis of leucine) by the LiCl method.[19,20] A 170-L fermentation of transformed GM-3C-2 cells producing the N62H mutant yielded 330 mg of pure protein after isolation by standard methods.[13] SDS–polyacrylamide gel electrophoresis after FPLC purification gave a single protein band with mobility corresponding to 13.4 kD, as expected for cytochrome *c*.

The N62H cytochrome *c* mutant[21] was modified with [a₅Ru-

(3) (a) McLendon, G. *Acc. Chem. Res.* **1988**, *21*, 160–167. (b) McLendon, G.; Miller, J. R.; Simolo, K.; Taylor, K.; Mauk, A. G.; English, A. M. In *Excited States and Reactive Intermediates*; Lever, A. B. P., Ed.; ACS Symposium Series 307; American Chemical Society: Washington, DC, 1986; pp 150–165.

(4) Peterson-Kennedy, S. E.; McGourty, J. L.; Ho, P. S.; Sutoris, C. J.; Liang, N.; Zemel, H.; Blough, N. V.; Margoliash, E.; Hoffman, B. M. *Coord. Chem. Rev.* **1985**, *64*, 125–133.

(5) (a) Tollin, G.; Hanson, L. K.; Caffrey, M.; Meyer, T. E.; Cusanovich, M. A. *Proc. Natl. Acad. Sci. U.S.A.* **1986**, *83*, 3693–3697. (b) Morgan, R. S.; Tatsch, C. E.; Gushard, R. H.; McAdon, J. M.; Warme, P. K. *Int. J. Pept. Protein Res.* **1978**, *11*, 209–217.

(6) (a) Axup, A. W.; Albin, M.; Mayo, S. L.; Crutchley, R. J.; Gray, H. B. *J. Am. Chem. Soc.* **1988**, *110*, 435–439. (b) Cowan, J. A.; Gray, H. B. *Chem. Scr.* **1988**, *28A*, 21–26. (c) Liang, N.; Pielak, G. J.; Mauk, A. G.; Smith, M.; Hoffman, B. M. *Proc. Natl. Acad. Sci. U.S.A.* **1987**, *84*, 1249–1252. (d) Liang, N.; Mauk, A. G.; Pielak, G. J.; Johnson, J. A.; Smith, M.; Hoffman, B. M. *Science (Washington, D.C.)* **1988**, *240*, 311–313.

(7) The N62H (tuna numbering) derivative is a triple mutant built on a base protein with Cys102Ser (prevents protein dimerization by disulfide formation) and His39Gln (removes the native surface histidine) mutations (relative to the wild-type protein): Mayo, S. L. Ph.D. Thesis, California Institute of Technology, 1988.

(8) The higher variability of surface compared to internal amino acids in a protein as a function of evolution indicates that surface-exposed sites are less likely to affect protein structure or function: Go, M.; Miyazawa, S. *Int. J. Pept. Protein Res.* **1980**, *15*, 211–224.

(9) Louie, G. V.; Hutcheon, W. L. B.; Brayer, G. D. *J. Mol. Biol.* **1988**, *199*, 295–314.

(10) The computer model of the ruthenated N62H mutant was generated by side-chain substitution using the Biograf/III software package (version 1.34, Biograf was designed and written by S. L. Mayo, B. D. Olafson, and W. A. Goddard III). The three-dimensional structure of yeast iso-1-cytochrome *c* was derived from the tuna and rice structures by side-chain substitution and molecular mechanics minimization (see: Mayo, ref 7). An 18 × 18 conformational search about the α–β, β–γ dihedral angles of the ruthenated histidine 62 side chain was performed. The 10 lowest energy conformations of that side chain were then further minimized by molecular mechanics methods (Biograf/III), holding the remainder of the protein rigid. The lowest energy conformation was taken as the most likely position of the His62-a₅Ru side chain.

(11) The phosphorothioate method was used: (a) Taylor, J. W.; Ott, J.; Eckstein, F. *Nucl. Acids Res.* **1985**, *13*, 8764–8785. (b) Nakamaye, K.; Eckstein, F. *Nucl. Acids Res.* **1986**, *14*, 9679–9698. (c) (Anonymous) *Oligonucleotide-directed In Vitro Mutagenesis System*; Amersham Corp: 1987.

(12) (a) The yeast iso-1-cytochrome *c* gene was a kind gift of Professor M. Smith of the University of British Columbia. (b) Pielak, G. J.; Mauk, A. G.; Smith, M. *Nature* **1985**, *313*, 152–154.

(13) Cutler, R. L.; Pielak, G. J.; Mauk, A. G.; Smith, M. *Protein Eng.* **1987**, *1*, 95–99.

(14) (a) Zoller, M. J.; Smith, M. *Nucl. Acids Res.* **1982**, *10*, 6487–6500. (b) Zoller, M. J.; Smith, M. *DNA* **1984**, *3*, 479–488.

(15) (a) Sanger, F.; Nicklen, S.; Coulson, A. R. *Proc. Natl. Acad. Sci. U.S.A.* **1977**, *74*, 5463–5467. (b) Biggin, M. D.; Gibson, T. J.; Hong, G. F. *Proc. Natl. Acad. Sci. U.S.A.* **1983**, *80*, 3963–3965.

(16) Hicks, J. B.; Strathern, J. N.; Klar, A. J. S.; Dellaporta, S. L. In *Genetic Engineering*, Setlow, J. K., Hollaender, A., Eds.; Plenum Press: New York, 1982; Vol. 4, pp 219–248. YEp213 also contains the origin of replication from the *S. cerevisiae* 2μ chromosome. The cloning size is in the tetracycline gene. Upon successful insertion of the CYC1 gene, the YEp213 plasmid can no longer confer tetracycline resistance.

(17) The YEp213/CYC1(N62H) plasmid DNA was amplified in *Escherichia coli* HB101 cells and sequenced by the dideoxy method to confirm the presence of the mutant gene.

(18) (a) *S. cerevisiae* strain GM-3C-2 has the phenotype [α, leu2-3, leu2-112, trp1-1, his4-519, cyc1-1, cyp3-1]. (b) Faye, G.; Leung, D. W.; Tatchell, K.; Hall, B. D.; Smith, M. *Proc. Natl. Acad. Sci. U.S.A.* **1981**, *78*, 2258–2262.

(19) Ito, H.; Fukuda, Y.; Murata, K.; Kimura, A. *J. Bacteriol.* **1983**, *153*, 163–168.

(20) Yeast colonies capable of growth on a nonfermentable carbon source, glycerol, and on leucine-deficient media, were presumed to contain YEp213/CYC1 (N62H). The mutant plasmid was removed from such yeast colonies (which should cause them to lose the ability to carry out oxidative metabolism), to ensure that the GM-3C-2 cells had not spontaneously reverted to a wild-type cell line capable of producing cytochrome *c*.

(21) The UV–vis spectrum of N62H cytochrome *c* is identical with that of the wild-type protein; the heme(Fe³⁺/²⁺) reduction potential is 268 mV vs NHE (differential pulse polarography; 7.5 mM 4,4′-bipyridine mediator; 50 mM sodium phosphate, pH 7.0, 0.4 M NaCl).

$(H_2O)](PF_6)_2$ (a = NH_3);[22] the products were separated by FPLC.[23] The singly modified product was characterized by differential pulse polarography; the heme($Fe^{3+/2+}$) reduction potential is 275 mV (NHE); the reduction potential of the $a_5Ru(His62)^{3+/2+}$ moiety is 75 mV (NHE), as expected.[24] The $Ru^{2+} \rightarrow Fe^{3+}$ ($-\Delta G° = 0.2$ eV) ET rate was measured by using the $[Ru(bpy)_3]^{2+}$/EDTA flash photolysis technique.[24] Electron transfer was monitored by the increase in absorbance at 550 nm, attributable to reduction of the heme iron. The kinetics were first order over three half-lives ($\sigma = 0.98$),[25] with $k_{obsd} = 1.7$ (1) s^{-1}.

A simple exponential edge–edge distance dependence $[\exp[-\beta(d - d_0)]$, with $\beta = 0.9$ Å^{-1} and no correction for difference in reorganization energy or driving force][6a] predicts a $Ru^{2+} \rightarrow Fe^{3+}$ ET rate for the N62H mutant of 0.4–2.0 s^{-1} relative to the 30 s^{-1} observed[24] for $a_5Ru(His33)$cytochrome c.[26] In the a_5Ru-(His33) derivative of horse heart cytochrome c, the ET pathway consists only of aliphatic residues.[2b,27] The finding that the rate for the ruthenated N62H mutant agrees strikingly with that calculated relative to horse heart cytochrome c suggests that the mere presence of aromatic residues and/or polarizable sulfur atoms along the pathway for electron transfer does not necessarily create conditions for significantly stronger donor–acceptor electronic coupling through the protein medium.[28]

Beratan and Onuchic have proposed a theoretical framework for long-range donor–acceptor coupling involving pathways that are combinations of covalent-bond, hydrogen-bond, and through-space interactions.[29] Using this approach, we have estimated the pathways of strongest coupling between the ruthenated histidine and the heme for N62H (mutant, see Figure 1) and for His33 (horse heart cytochrome c).[30] A comparison of the best pathways gives $k_{ET(His62)} = k_{ET(His33)}[H_{ab(His62-Met64)}/H_{ab(His33-Pro30)}]^2 = 0.4$ s^{-1} relative to 30 s^{-1} for His33 (horse heart cytochrome c).[31] This value also agrees well with the observed rate for the ruthenated N62H mutant.

(22) Callahan, R. W.; Brown, G. M.; Meyer, T. J. *Inorg. Chem.* 1975, *14*, 1443–1453.

(23) N62H cytochrome c (50 mg) was reacted with a 40-fold excess of $[a_5Ru(H_2O)](PF_6)_2$ (80 mg, 0.16 mmol) in 16 mL of deoxygenated HEPES, pH 7.0, for 45 min in an argon atmosphere. The reaction was terminated by separating the protein from the unreacted ruthenium complex on a Sephadex G-25 column. The protein was oxidized with [CoEDTA]$^-$ prior to FPLC purification.

(24) Nocera, D. G.; Winkler, J. R.; Yocom, K. M.; Bordignon, E.; Gray, H. B. *J. Am. Chem. Soc.* 1984, *106*, 5145–5150.

(25) Flash conditions were as follows: 100 mM sodium phosphate, pH 7.0, 8.3 mM EDTA, 65 μM [Ru(bpy)$_3$]$^{2+}$, and 2 μM ruthenated N62H cytochrome c. No concentration dependence of k_{obsd} was seen over a 4-fold concentration range.

(26) The rate for k_{His62} was obtained by using $d_{His62} = 14.7–15.6$ Å (ref 10); $d_{His33} = 10.8–11.7$ Å (ref 6a).

(27) Meade, T. J.; Gray, H. B.; Winkler, J. R. *J. Am. Chem. Soc.* 1989, *111*, 4353–4356.

(28) Kuki and Wolynes came to a similar conclusion after making an extensive theoretical analysis of the long-range ET pathway in a ruthenated myoglobin: Kuki, A.; Wolynes, P. G. *Science (Washington, D.C.)* 1987, *236*, 1647–1652.

(29) (a) Beratan, D. N.; Onuchic, J. N. *Photosynth. Res.*, in press. (b) Beratan, D. N.; Onuchic, J. N.; Hopfield, J. J. *J. Chem. Phys.* 1987, *86*, 4488–4498. (c) Cowan, J. A.; Upmacis, R. K.; Beratan, D. N.; Onuchic, J. N.; Gray, H. B. *Ann. N.Y. Acad. Sci.* 1989, *550*, 68–84.

(30) Optimal pathways were determined by using a computer program written by J. N. Betts. The calculated pathway for horse heart cytochrome c (His33–Leu32–Asn31–Pro30–His18–Fe) is abbreviated His33–Pro30.

(31) No range is reported for the rate calculation based on the pathway model, since the best pathway (not a range of pathways) from His62 of the N62H mutant is being compared with the best pathway from His33 (horse heart cytochrome c).

Radical Cation Cope Rearrangement of 1,5-Hexadiyne to 1,2,4,5-Hexatetraene (Bis(allene)) at 77 K

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Although the formation of chair cyclohexane-1,4-diyl radical cation intermediates in the oxidation of 1,5-hexadienes[1,2] can be regarded as the first step in a Cope-like reaction, the subsequent retrocyclization required to complete the rearrangement is quite unlikely to occur in degenerate or nearly degenerate systems in view of the manifestly greater stability of the cyclic intermediate.[1] Indeed, this return step was previously calculated to be endothermic by 34 kcal mol^{-1} for the parent 1,5-hexadiene.[3] The identification of cycloolefinic and aromatic products in these oxidations[1,4] also clearly points to this irreversibility. It therefore seems likely that some,[2] if not all,[5] of the previously reported radical cation induced Cope rearrangements of aryl-substituted 1,5-hexadienes[2,5] actually proceed through back electron transfer[2] to form neutral cyclohexane-1,4-diyl precursors which can easily undergo the necessary cleavage to the rearranged 1,5-hexadienes.[4] At any rate, a Cope-type rearrangement has not hitherto been demonstrated exclusively at the radical cation stage, and here we report the first direct observation of such a reaction.

Acetylenic Cope processes leading to allenes[6] provide examples of extremely nondegenerate systems with an appreciable net driving force that should be augmented in the radical cation because of the higher ionization potentials associated with acetylenes.[7] In studying the radiolytic oxidation of 1,5-hexadiyne (1) in Freon matrices, we observed an intense and well-defined ESR spectrum (Figure 1a) in several haloethanes (CF_3CCl_3, CF_2ClCCl_3, $CF_2ClCFCl_2$, and $CFCl_2CFCl_2$) which is readily analyzed as a quintet ($a(4H) = 28.6$ G) of triplets ($a(2H) = 3.8$ G) with a g factor of 2.0024. This is clearly the spectrum of a symmetrically delocalized species, and since the corresponding spectrum (b) from 1,6-dideuterio-1,5-hexadiyne is a simple quintet[8]

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(1) (a) Guo, Q.-X.; Qin, X.-Z.; Wang, J. T.; Williams, F. *J. Am. Chem. Soc.* 1988, *110*, 1974. (b) Williams, F.; Guo, Q.-X.; Bebout, D. C.; Carpenter, B. K. *J. Am. Chem. Soc.* 1989, *111*, 4133.

(2) Miyashi, T.; Konno, A.; Takahashi, Y. *J. Am. Chem. Soc.* 1988, *110*, 3676.

(3) Bauld, N. L.; Bellville, D. J.; Pabon, R.; Chelsky, R.; Green, G. *J. Am. Chem. Soc.* 1983, *105*, 2378.

(4) Adam, W.; Grabowski, S.; Miranda, A. A.; Rübenacker, M. *J. Chem. Soc., Chem. Commun.* 1988, 142.

(5) Lorenz, K.; Bauld, N. L. *J. Catal.* 1983, *95*, 613. In this reference, the photoassisted, zeolite-catalyzed rearrangement of 1,3,4-triphenyl-1,5-hexadiene to 1,3,6-triphenyl-1,5-hexadiene is attributed to a radical cation Cope process. A referee has pointed out that this reaction should have enough thermodynamic driving force to outweigh the barrier imposed by the intrinsic stability of the cyclic 1,4-diyl radical cation structure, this driving force coming from the development of the conjugation energy from two styrene-like systems in the product radical cation compared to only one such system in the reactant radical cation. The above-referenced study also reports, however, that the attempted hole acquisition of this rearrangement using the tris(p-bromophenyl)aminium hexachloroantimonate reagent failed to take place although this reagent is an effective catalyst for radical cation Diels–Alder processes (Reynolds, D. W.; Bauld, N. L. *Tetrahedron* 1986, *42*, 6189). We conclude that the precise stage of back electron transfer, and therefore the reaction pathway, in the photoassisted, zeolite-catalyzed Cope rearrangement of 1,3,4-triphenyl-1,5-hexadiene is conjectural.

(6) Viola, A.; Collins, J. J.; Filipp, N. *Tetrahedron* 1981, *37*, 3765.

(7) Kimura, K.; Katsumata, S.; Achiba, Y.; Yamazaki, T.; Iwata, S. *Handbook of HeI Photoelectron Spectra of Fundamental Organic Molecules*; Japan Scientific Societies Press: Tokyo, 1981; pp 57–71.

(8) The minor spectral components present between the lines of the quintet in Figure 1b belong to a quartet ($a(3H) = 28.6$ G) of triplets ($a(1D) = 4.4$ G) pattern that can be assigned to the isotopic radical cation in which one of the four strongly coupled hydrogens has been replaced by deuterium. This species is presumably produced either from a small amount of the correspondingly labeled 1 or from 1-d_2 as the result of H–D exchange during the radical cation rearrangement. In the latter case, the expected broadening from the deuterium hfs may obscure the small hfs to one hydrogen.