phase. Fluoride reacts with neopentyl chloride to yield neopentyl fluoride.[9] However, neopentyl phenyl carbonate shows scarcely any extrusion of CO_2 in the mass spectrum (<0.05% of the phenol radical cation intensity). The labeled neopentyl phenyl carbonate ion **4** yields only $PhOH^{\bullet+}$ and $PhOD^{\bullet+}$ in the metastable ion mass spectrum (MIKES[5]), with no detectable M - CO_2 (*m/z* 166). In both MIKES and CAD, the $PhOH^{\bullet+}$:$PhOD^{\bullet+}$ ratios are the same as observed for $(CH_3)_3CCD_2OPh$.[15] We consider this result to be of special significance, for it implies that CO_2 expulsion for R = neopentyl yields the same [*tert*-amyl$^+$ PhO$^\bullet$] complex as does neopentyl phenyl ether,[16] as eq 5 depicts. The absence of any

$$(CH_3)_3CCD_2OC(=O)OPh^{\bullet+} \longrightarrow [(CH_3)_3CCD_2O\overset{+}{C}{\equiv}O \quad PhO^\bullet] \xrightarrow{-CO_2} PhOD^{\bullet+} \text{ or } PhOH^{\bullet+} \longleftarrow [(CH_3)_2\overset{+}{C}CD_2CH_3 \quad PhO^\bullet] \quad (5)$$

ion from recombination of these two fragments[17] suggests that proton transfer takes place at least 1000 times faster than reformation of a covalent bond for the low-energy ions in the MIKES.

In summary, the unimolecular decompositions of alkyl phenyl carbonate radical cations can be described using a familiar mechanistic vocabulary. For R = *sec*-butyl, expulsion of CO_2 occurs by means of bond fission and then backside displacement. We attribute the small amount of scrambling to a competing S_N1 pathway. For R = neopentyl, there is rearrangement followed by E1 elimination without any nucleophilic substitution. Viewed in this way, the pattern of gas-phase reactivity mirrors the trend seen in solution.

Acknowledgment. We are grateful to Prof. J. L. Holmes for providing a preprint of ref 18 prior to publication. This work was supported by the CNRS, the NSF (Grant CHE 88-02086), and a Fulbright travel grant to T.H.M.

(15) Sozzi, G.; Audier, H. E.; Mourgues, P.; Milliet, A. *Org. Mass Spectrom.* **1987**, *22*, 746-747.
(16) Morton, T. H. *J. Am. Chem. Soc.* **1980**, *102*, 1596-1602.
(17) We can rule out the *tert*-amyl phenyl ether ion $CH_3CD_2C(CH_3)_2OPh^{\bullet+}$ as a fleeting intermediate, since that ion is known to give only $PhOD^{\bullet+}$ in the MIKES.[15] Also, since $(CH_3)_3CCH_2OPh^{\bullet+}$ and *sec*-$BuOPh^{\bullet+}$ both have high barriers for metastable decomposition (48 and 55 kJ mol^{-1}, respectively, above the thermodynamic thresholds[18]), it seems unlikely that loss of CO_2 forms neopentyl ether ions that decompose faster than they can be detected.
(18) Harnish, D.; Holmes, J. L. *J. Am. Chem. Soc.* In press.

Elimination of Electrooxidizable Interferants in Glucose Electrodes

Ruben Maidan* and Adam Heller*

Department of Chemical Engineering
The University of Texas at Austin
Austin, Texas 78712-1062

Received June 13, 1991
Revised Manuscript Received September 25, 1991

Amperometric glucose electrodes, now in common use, are not as selective as they are intended to be, responding also to electrooxidizable interferants present in the analyzed medium.[1] A much studied assay is that of glucose in physiological fluids.

(1) Scheller, F. W.; Pfeiffer, D.; Schubert, F.; Renneberg, R.; Kirstein, D. In *Biosensors Fundamentals and Applications*; Turner, A. P. F., Karube, I., Wilson, G. S., Eds.; Oxford University Press: Oxford, 1987; pp 315-346.

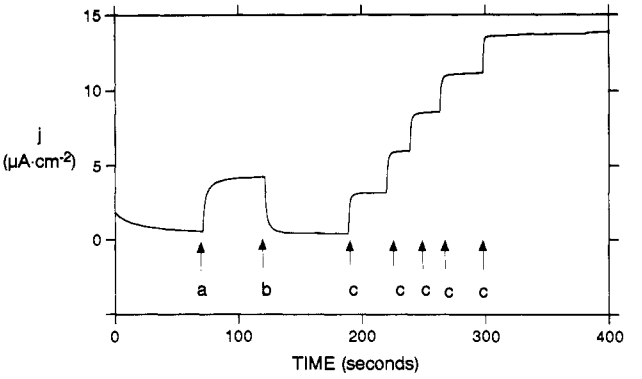

Figure 1. Oxidation currents measured with a bilayer electrode composed by an HRP film immobilized on top of an electrically "wired" GOD film. Ascorbate currents are eliminated by the HRP layer in the presence of hydrogen peroxide. The glucose concentration is unaffected and is measured by the "wired" GOD gel sensing layer: (a) ascorbate injection, 0.1 mM final concentration; (b) H_2O_2 injection, 0.1 mM final concentration; (c) glucose injections, 1.0 mM concentration steps. Three electrode stirred cell, glassy carbon working electrode poised at 0.5 V vs SCE, 0.1 M phosphate buffer pH 7.2, NaCl 0.1 M.

Glucose oxidase based electrodes respond also to ascorbate and urate ions and to *p*-acetamidophenol (Tylenol). These interferants may be oxidized both at the electrode surface or by a diffusing mediator or enzyme bound electron relay. Methods proposed to overcome their interference, based on size exclusion,[2] electrostatic repulsion,[3] electrochemical preoxidation,[4] or specific enzymatic reactions[5] partially solve but do not eliminate the problem of electrooxidizable interferants. Here we report on an enzymatic method that simplifies their combined elimination.

The enzyme horseradish peroxidase (HRP) catalyzes the oxidation of a range of compounds by hydrogen peroxide.[6] We find that urate, ascorbate, and *p*-acetamidophenol but not glucose are enzymatically oxidized. Interferants can thus be eliminated by their HRP-catalyzed oxidation by hydrogen peroxide (eq 1). Using electrodes covered by a thin layer of glutaraldehyde immobilized HRP (300 U·cm^{-2}) the electrooxidation currents for the different interferants in the presence or absence of H_2O_2 were measured and are shown for ascorbate in Figure 1. When ascorbate is injected in the cell, a substantial oxidation current is observed. This current is no longer measurable after addition of H_2O_2, implying that ascorbate is oxidized to an electrochemically inert species. Subsequent additions of ascorbate do not result in an oxidation current provided that the H_2O_2 is not exhausted. The oxidation current for ascorbate is decreased by a factor of more than 2500 in the presence of H_2O_2. Control experiments show that both H_2O_2 and HRP must be present for the elimination of ascorbate. The results for ascorbate, urate, or *p*-acetamidophenol or their various combinations are similar.

$$\text{interferant} + \text{glucose} + H_2O_2 \xrightarrow{\text{HRP}} \text{oxidized interferant} + \text{glucose} + 2H_2O \quad (1)$$

The observed elimination of interferants was implemented in "wired" glucose oxidase electrodes where an HRP layer covered

(2) Palleschi, G.; Nabi Rahni, M. A.; Lubrano, G. J.; Ngwainbi, J. N.; Guilbault, G. G. *Anal. Biochem.* **1986**, *159*, 114-121.
(3) (a) Harrison, D. J.; Turner, R. F. B.; Baltes, H. P. *Anal. Chem.* **1988**, *60*, 2002-2007. (b) Sasso, S. V.; Pierce, R. J.; Walla, R.; Yacynych, A. M. *Anal. Chem.* **1990**, *62*, 1111-1117. (c) Gorton, L.; Karan, H. I.; Hale, P. D.; Inagaki, T.; Okamoto, Y.; Skotheim, T. A. *Anal. Chim. Acta* **1990**, *228*, 23-30.
(4) (a) Yao, T.; Kobayashi, Y.; Sato, M. *Anal. Chim. Acta* **1983**, *153*, 337-340. (b) Ikeda, T.; Katasho, I.; Senda, M. *Anal. Sci.* **1985**, *1*, 455-457.
(5) (a) Nagy, G.; Rice, M. E.; Adams, R. N. *Life Sci.* **1982**, *31*, 2611-2616. (b) Scheller, F.; Renneberg, R. *Anal. Chim. Acta* **1983**, *152*, 265-269.
(6) (a) Maehly, A. C. *Methods Enzymol.* **1955**, *2*, 801-813. (b) Dunford, H. B.; Stillman, J. S. *Coord. Chem. Rev.* **1976**, *19*, 187-251.
(7) Gregg, B. A.; Heller, A. *J. Phys. Chem.* **1991**, *95*, 5970-5975; **1991**, *95*, 5976-5980.

0002-7863/91/1513-9003$02.50/0

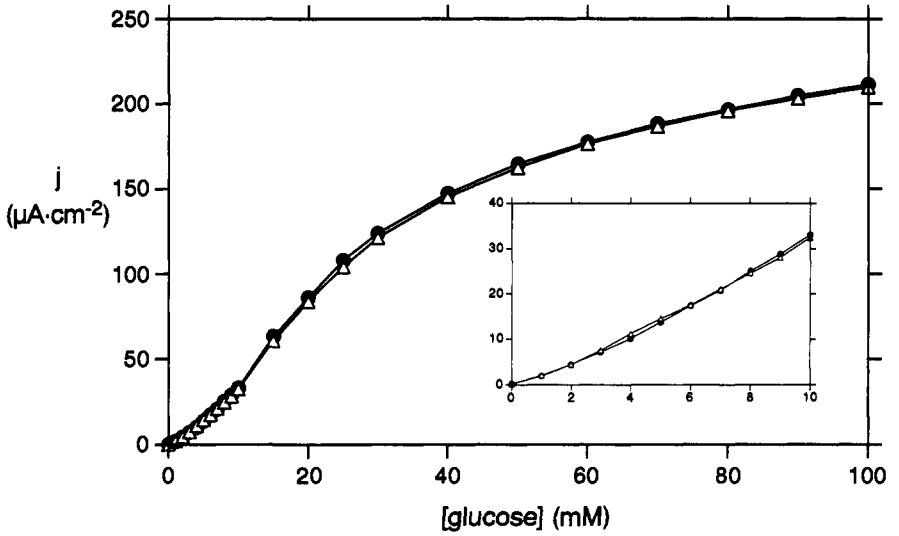

Figure 2. Calibration curve for glucose using the bilayer electrode of Figure 1 in the presence of 1.0 mM hydrogen peroxide: (a) (Δ) without interferants present and (b) (●) in the simultaneous presence of 0.1 mM ascorbate, 0.1 mM *p*-acetamidophenol, and 0.5 mM urate. The insert shows the calibration in the physiological concentration range of glucose.

a sensing layer. The glucose oxidase (GOD) was electrically "wired" to the electrode with a redox epoxy gel matrix.[7] This electrode is based on a 130 kilodalton poly(vinylpyridine) complex of osmium bis(bipyridyl) chloride with GOD cross-linked with a 400 dalton polyethyleneglycol diglycidyl ether. This glucose electrode of high current density and fast response is sensitive to electrooxidizable interferants. This sensitivity is eliminated by coating the glucose sensing layer with a layer of immobilized HRP. While the interferants are eliminated, the response to glucose is unaffected (Figure 1). The current, increasing monotonically with glucose concentration, does not change when the concentration of the interferants is increased. An electrode potential of +0.5 V (SCE) was maintained throughout these measurements to prevent potentially interfering HRP-catalyzed electroreduction of H_2O_2. Figure 2 shows calibration curves under elimination conditions for glucose and for glucose in the presence of the interferants at their physiological levels. It is seen that the glucose response is not affected by the addition of interferants even though in the absence of hydrogen peroxide the current density for the combined interferants is 10^2 $\mu A \cdot cm^{-2}$.

Hydrogen peroxide, essential for eliminating the interferants, cannot be externally added in in vivo assays but can be produced in situ by oxidases; thus, lactate oxidase (LOD) generates hydrogen peroxide in the presence of lactate and oxygen (eq 2). Using an electrode containing LOD (250 $U \cdot cm^{-2}$) coimmobilized with HRP and physiological levels of lactate (0.5 mM) we oxidatively stripped the interferants and accurately measured glucose levels without adding hydrogen peroxide. The results we obtained were similar to those shown in Figure 2. A control experiment performed to examine the effect of addition of lactate (1.5 mM) on the electrocatalytic oxidation current for 5 mM glucose showed that the change in the current was less than 2%. Since electrooxidation of glucose at the wired GOD does not involve O_2, the removal of oxygen by lactate (eq 2) has only a marginal effect on the glucose assay. The current does not change with lactate concentration, because LOD is not "wired" to the electrode being physically separated from the redox epoxy. The one purpose of LOD in this structure is to produce in situ hydrogen peroxide for the HRP-catalyzed elimination of interferants. The LOD-catalyzed generation of H_2O_2 is O_2 dependent, and no peroxide is formed in the total absence of oxygen. In the physiological range oxygen concentrations provide, however, the excess hydrogen peroxide needed, and thus fluctuations in oxygen concentration do not affect the elimination of interferants.

$$2CH_3CH(OH)COO^- + O_2 \xrightarrow{LOD} 2CH_3COCOO^- + H_2O_2 \quad (2)$$

In summary, peroxidase-based layers effectively eliminate interferants in the presence of hydrogen peroxide without affecting the glucose quantitation, allowing selective measurement of its concentration.

Acknowledgment. This work was supported in part by the National Science Foundation. R.M. gratefully acknowledges his Chaim Weizmann postdoctoral fellowship.

The Ring Opening and Unusual Coupling of 3,3-Dimethylthietane Ligands in a Dirhenium Carbonyl Complex

Richard D. Adams,* Judy A. Belinski, and John Schierlmann

Department of Chemistry, University of South Carolina Columbia, South Carolina 29208

Received July 15, 1991

Recent reports have described the first examples of ring-opening reactions of thietane ligands by metal cluster complexes.[1-6] A range of processes have been observed: thermal,[1,2,6] photochemical,[3] and nucleophile promoted,[4,5] which suggests that a variety of mechanisms may be operative. We have recently reported the first example of a facile, metal-promoted, head-to-tail ring-opening oligomerization of 3,3-dimethylthietane, DMT, through a process that is initiated by a bridging coordination of the first molecule to a triosmium cluster.[4] We have now discovered a novel photoassisted ring opening and coupling of two DMT ligands in a dirhenium complex that culminates with the formation of a disulfide link between two metallacycles.

The reaction of $Re_2(CO)_8[\mu\text{-}C(H)CBu^n](\mu\text{-}H)$[7] with DMT in refluxing CH_2Cl_2 yielded the complex 1,2-$Re_2(CO)_8(SCH_2CMe_2CH_2)_2$ (**1**) in 71% yield.[8] Compound **1** was char-

(1) Adams, R. D.; Pompeo, M. P. *Organometallics* **1990**, *9*, 2651.
(2) Yamamoto, J. H.; Yap, G. P. A.; Jensen, C. M. *J. Am. Chem. Soc.* **1991**, *113*, 5060.
(3) Adams, R. D.; Pompeo, M. P. *Organometallics* **1990**, *9*, 1718.
(4) Adams, R. D.; Pompeo, M. P. *J. Am. Chem. Soc.* **1991**, *113*, 1619.
(5) Adams, R. D.; Belinski, J. A.; Pompeo, M. P. *Organometallics* **1991**, *10*, 2539.
(6) Adams, R. D.; Pompeo, M. P. *Organometallics*, in press.
(7) Nubel, P. O.; Brown, T. L. *J. Am. Chem. Soc.* **1982**, *104*, 4955.

0002-7863/91/1513-9004$02.50/0