Table 1. Mean time of day for entry into and arousal from torpor. The standard error of the mean is given in minutes in parentheses. The sample size varied from 15 to 35. Weight maintenance in the absence of torpor requires about 3.5 g of seeds each day (17.5 percent mean body weight).

| Food ration (g) | Mean time (hr) | |
|---|---|---|
| | Entry | Arousal |
| 0.4 | 2018 (39) | 1142 (22) |
| 1.0 | 2324 (30) | 1212 (21) |
| 1.5 | 0148 (48) | 1324 (26) |
| 2.0 | 0424 (28) | 1224 (12) |
| 2.5 | 0436 (30) | 1230 (22) |

weight did not change and some of its food was uneaten. All mice remained in good condition during the experiments and survived collectively more than 150 periods of torpor.

The mice showed the same responses during oxygen consumption experiments that they did in the paired jars. Figure 1 is a record of the oxygen consumption of one mouse for 24 hours. All the mice followed this pattern, showing a moderate increase in oxygen consumption prior to the beginning of torpor, and a large increase during arousal. With a few exceptions, oxygen consumption declined smoothly as a mouse entered a period of torpor. This decline continued at a decreasing rate for more than 5 hours before a minimum oxygen consumption of about 0.15 $cm^3$ per gram of body weight per hour was attained, corresponding to a body temperature of 16°C.

Experiments with other *P. californicus* have demonstrated that mice which maintain high body temperatures (38°C) behave as typical homeotherms. Their metabolic rate reaches a minimum of 0.97 $cm^3$ of oxygen per gram per hour at an ambient temperature of 32.5°C. Above and below this temperature, metabolism rises. A comparison of the minimum metabolic rate at a body temperature of 16°C and the minimum metabolic rate at a body temperature of 38°C yields a $Q_{10}$ of 2.4, indicating that the depression of metabolism from the basal level to the level in torpor is temperature dependent in the manner typical of biological systems. During arousal at an ambient temperature of 23°C, the body temperature of two mice increased at a maximum rate of 0.67°C per minute.

The major advantage of diurnal torpor appears to be energy conservation. Energy conservation could be of critical importance to a small animal such as *P. californicus* which lives in a region of severe seasonal drought. The effectiveness of diurnal torpor when food is scarce was shown by one mouse which was able to maintain its original weight on a food ration that was only 43 percent of the food required when it maintained a continuously high body temperature.

The genus *Perognathus* contains many desert dwellers. Diurnal torpor should be as advantageous for the desert species as it is for *P. californicus*. I have obtained quantitative measurements of diurnal torpidity in *P. baileyi, P. fallax, P. flavus, P. formosus*, and *P. longimembris*, and other species are known to enter torpidity. Thus, diurnal torpor may be characteristic of the genus (*6*).

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## Platinized Silver Chloride Electrode

*Abstract.* A hybrid electrode made by platinizing silver–silver chloride has been found to combine the stable potential and low direct-current resistance properties of a silver–silver chloride electrode with the low high-frequency impedance characteristic of a platinized platinum electrode.

The silver–silver chloride electrode made by slow plating of silver chloride from KCl solution onto silver can be a convenient and satisfactory potential electrode. Its resistance at low frequencies and direct current may reach about 5 ohm $cm^2$ for a deposit of about 2 coul/$cm^2$ but increases rapidly with either more or less plating. Platinum black plated onto platinum or other metals is a most satisfactory and widely used electrode for alternating current and transient purposes and may have an impedance less than 1 ohm $cm^2$ at 1 kcy/sec after a deposition of about 10 coul/$cm^2$. However, it does not have either a definite electrode potential or direct-current resistance as ordinarily used.

The hybrid electrode was made by first depositing a heavy layer of silver chloride on silver in 0.5*M* KCl with 10 or more coulombs per square centimeter at 2 ma/$cm^2$ to give a resistance of more than 300 ohm $cm^2$. It was then platinized in Kohlrausch solution (*1*) with 1 to 5 coul/$cm^2$ at about the same current density. The electrode was then returned to 0.5*M* KCl, and a small charge was passed in the direction to deposit silver chloride.

The completed electrode then had an equilibrium potential within a few millivolts of that of the original silver–silver chloride with an impedance of a few ohm square centimeters at 20 cy/sec or less, approaching that of a good platinized electrode at higher frequencies.

As additional chloride was deposited from a KCl solution the potential remained undisturbed but the impedance increased rapidly, as if an additional layer of silver chloride were being laid down. With the current flow reversed (that is, in the direction to remove silver chloride), the potential remained reasonably constant until a charge was passed approximately equal to that used to deposit the original silver chloride layer. Beyond this point the impedance remained unchanged at high frequencies but rose at low frequencies and the electrode potential was poorly defined and unstable, as if the silver chloride had been completely removed, leaving the platinum black intact. When the electrode was again plated with chloride, the stable resistance and potential were promptly restored. Cycles of chloride deposition and removal have been repeated, within and about the ends of the useful range, without obvious deterioration of the electrical or mechanical characteristics of the electrode.

The electrode thus approaches the best properties of both silver–silver chloride and platinized electrodes for use from direct current to high frequencies so long as the silver chloride content is not exhausted or does not exceed that at which the platinum black was deposited.

The electrode development was stimulated by the work of Teorell and Wersäll (*2*) in which they used platinized silver electrodes. But their removal of any possible silver chloride by ammonia suggested, contrariwise, that the chloride might serve a useful purpose. Marmont (*3*) described a

"developed" silver chloride electrode of similar properties. But recent tests have shown it to have an impedance range several times higher and a "coulomb range" considerably smaller than the platinized product has. Also in one case the layer of reduced silver fell off after electrolytic removal of the underlying chloride.

No specific explanations of the structure or operation of this hybrid electrode are offered, and no more detailed description or further investigations of its properties are planned.

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## Immunological Competence of Placenta

*Abstract.* The presence of immunologically competent cells in placenta has been investigated by use of the experimental model of runt disease in inbred strains of mice. The injection of placental cells from C57/Bl mice into newborn Balb/C mice resulted in death in the 2nd to 4th week. Evidence is presented that death was caused by immunologically competent fetal cells.

Interest in the mechanisms of transplantation rejection has recently intensified. This has focused attention on what appears to be a remarkably successful homograft, the placenta.

In considering the immunological relations about the placental site, we first investigated whether placental tissue possesses antigens that stimulate tissue rejection. Using inbred strains of mice, we found that the injection of placenta cells from one strain into another induced accelerated rejection of a subsequent homologous skin graft and also the formation of cytotoxic antibodies (*1*). Uhr and Anderson, using a similar approach, reached the same conclusion (*2*).

The next question to be investigated was the presence of immunologically competent cells in placenta. This was undertaken by using the recently described "runt syndrome" as a model. The runt syndrome, characterized by slow growth and death, is produced by the injection of immunologically competent cells from one strain of mice into homologous newborn mice (*3*). The generally accepted explanation is that the newborn mouse is incapable of rejecting the transplanted cells, which survive and in effect "reject" the host, producing an immunological disease. The subject of this report is the results of experiments in which transplants of placenta cells were used.

Mice (C57/Bl and Balb/C) were obtained from the Jackson Memorial Laboratories. A breeding colony of each strain was maintained in our laboratories. Within 24 hours of the birth of a Balb/C litter, a C57 mouse estimated by size to be 17 to 20 days pregnant was selected, and an experiment was performed.

Cell suspensions were prepared in the customary manner, and 10 million cells of various tissues were injected intraperitoneally into newborn Balb/C mice. In preparing placenta cell suspensions, the maternal decidua was first carefully dissected away under a magnifying lens. The viability of the cell suspensions was checked with 0.1 percent eosin Y.

Each Balb/C litter was divided into two to four groups of two to three animals each, depending on the size of the litter; each group received a different type of cell suspension. This permitted a controlled comparison of results within each experiment. For example, in a litter of nine, three animals received C57 spleen cells, three received C57 liver cells and three received C57 placenta cells. The results of all experiments were so consistent that they have been pooled for presentation (Table 1).

A few deaths occurred in all experimental animals during the first few days. The deaths were undoubtedly related to the problems of birth and possibly to the trauma of the experimental procedure. Thereafter, no deaths occurred until after the first week—the latent period common in primary immunological reactions and in runt disease. After the first week, the deaths were limited almost entirely to those animals that had received injections of C57 placenta or spleen cells.

The results obtained with C57 placenta cells closely paralleled those obtained with C57 spleen cells, suggesting a mechanism common to both. However, the results with placenta were sufficiently unexpected to warrant a series of control experiments to exclude other possible explanations. Three major possibilities were considered other than immunological disease resulting from competent placenta cells. These were infection, the release of a lethal pharmacological agent by surviving cells, and the contamination of the cell suspension by competent maternal cells. The design of many of these experiments was suggested by previous work on runt disease (*3, 4*).

The possibility of infection is rendered unlikely by the survival of Balb/C newborn mice following the injection of C57 liver cells, placenta homogenate, or the combined injection of adult Balb/C spleen cells and C57 placenta cells. As a further check, a series of newborn Balb/C mice that had received intact placenta cells were sacrificed a week later, and suspensions of their livers and spleens were injected into another group of Balb/C animals without ill effect. It would be anticipated that an infectious agent would be detected by such serial transfer.

The second possibility, that of release of a lethal toxin by injected placenta cells, is excluded by the survival of Balb/C newborn mice receiving F1 pla-

Table 1. Effect of various cell suspensions in producing runt disease and death in newborn Balb/C mice. The number of animals that died are listed by age in days at the time of death. There were no deaths during the period 4 to 9 days. Fourteen newborn Balb/C mice received F1 placental suspensions (4 from C57 ♂ × Balb/C ♀ matings, 10 from Balb/C ♂ × C57 ♀), and all survived.

| Age at death (days) | No. of deaths with suspension indicated | | | | | |
|---|---|---|---|---|---|---|
| | C57 spleen | C57 placenta | Hank's sol. | C57 placenta homogenate | C57 liver | Balb/C spleen + C57 placenta |
| 0–3 | 3 | 4 | 1 | 1 | 2 | |
| 10–12 | 6 | 5 | | | | 1 |
| 13–15 | 2 | 6 | | | | |
| 16–18 | 1 | 5 | | | | |
| 19–21 | 4 | 6 | | | | |
| 22–24 | 1 | 2 | | | | |
| Living at 25 days | 4* | 0 | 6 | 11 | 7 | 9 |

* All were much smaller (runts) than controls.