

pO₂, pH, and Redox Potential of Experimental Abscesses (38275)

ROBERT C. HAYS AND GERALD L. MANDELL¹

Department of Medicine, University of Virginia School of Medicine, Charlottesville, Virginia 22901

An abscess is a localized accumulation of leucocytes (predominantly polymorphonuclear neutrophils (PMN) with necrosis and partial liquefaction of cells and tissue to form pus. Abscesses may form in response to infection by pyogenic organisms or in response to chemical irritants such as turpentine. Undrained abscesses heal slowly, if at all, and antibiotic therapy is notoriously unsuccessful without concomitant surgical drainage.

PMN bactericidal functions in abscesses may be impaired by conditions in the abscess. Since recent work demonstrated that decreased pO₂ inhibits the antibacterial action of PMN's (1), we studied the pO₂, pH, and redox potential of experimental abscesses to determine if these factors may be important in bacteria-phagocyte interaction.

Methods. Induction of abscesses. White, male rats weighing 200–300 g were used for all experiments. The backs were shaved and abscesses were induced in 20 rats by the subcutaneous injection of 2 ml spirits of turpentine. After 2 days, groups of five rats received injections into the site of the turpentine abscesses of either: (a) 2×10^8 *Staph. aureus* (Woods 46 strain) in 2 ml trypticose soy broth; (b) 2×10^8 *E. coli* (type 0111-B4) in 2 ml trypticose soy broth; or (c) 2 ml of a fecal-saline suspension (seven pellets of rat feces were mixed in 10 ml sterile saline, centrifuged at 200g for 10 min, and the supernatant used for injection). A fourth group of five rats with turpentine-induced abscesses did not receive bacteria.

Subcutaneous cavities were produced by administering 25 ml ultrapure nitrogen gas into the subcutaneous tissues of four rats (2). The cavities were then injected with 2×10^8 *Staph. aureus* (Woods 46 strain), 2×10^8 *E. coli* (type

0111-B4), or 2 ml of a rat fecal-saline suspension.

Physiochemical determinations on pus. Seven days after the administration of turpentine (5 days after the administration of bacteria), the abscesses were aspirated and the contents examined for pO₂, pH, and redox potential. The pus was aspirated through 19-gauge needles into blood-gas syringes and maintained air-tight by placing a rubber stopper over the needle tip.

pO₂ measurements were done on an Instrumentation Laboratory Blood Gas Analyzer which was calibrated before each determination. The pH and redox potentials were determined under nitrogen gas flooding with a Corning semimicro Hydrogen Ion electrode.

Results. All of the rats receiving turpentine injections developed abscesses ranging in size from 2 to 4 cm in diameter. These were discrete, fluctuant masses from which it was generally easy to aspirate pus. Volumes of pus obtained ranged from 0.7 to 2.8 ml, being nonsanguineous but ranging from primarily serous to frankly purulent in appearance.

Microscopic examination of the pus revealed much amorphous, necrotic tissue. PMN's were recognizable in varying states of degeneration, and occasional bacteria were demonstrable.

Table I shows results of physiochemical determinations on pus aspirated from abscesses. Figure 1 shows pO₂ measurements from abscesses induced by various methods.

Discussion. The normal arterial pO₂ in most mammals is 80–100 mm Hg, and the normal venous pO₂ is 30–60 mm Hg. Microneedle electrode studies of human muscle showed a wide range of values from 0.5 to 100 mm Hg, with most measurements in the 20–40 mm Hg range (3). In 1930, Kempner and Peschel (4) studied pus from skin blisters induced by applying Cantharide patches. Among other abnormalities, they found that the pO₂ of the pus was

¹ Recipient of Research Career Development Award (GM-49504) from the National Institute of General Medical Sciences.

TABLE 1. Determinations on Pus Aspirated from 7-Day-Old Abscesses (Means and Range are Shown).

Abscess produced by injection of:	pO ₂ (mm Hg)	pH	Redox potential (mV)
Turpentine only (<i>N</i> = 5)	36.4 (0-58)	7.2 (7.1-7.3)	-10 (-20 to 0)
Turpentine + <i>Staph.</i> (<i>N</i> = 5)	32.6 (13-71)	6.7 (6.2-7.0)	+20 (+10 to +40)
Turpentine + <i>E. coli</i> (<i>N</i> = 5)	38.2 (1-75)	7.1 (6.95-7.2)	-4 (-10 to 0)
Turpentine + feces-saline (<i>N</i> = 5)	8.4 (0-23)	7.0 (6.7-7.1)	+3 (-5 to +20)
N ₂ staph. (<i>N</i> = 2)	8 (2-14)	7.18 (7.15-7.2)	-10
N ₂ + <i>E. coli</i> (<i>N</i> = 1)	12	7.1	0
N ₂ + feces-saline (<i>N</i> = 1)	0	7.0	0

as low as 21 mm Hg after 3 days. Although they used no bacteria, their work certainly suggested that a degree of anaerobiosis existed in abscess cavities.

It has been shown that PMN's have decreased bactericidal activity in hypoxic conditions. Studies indicate that although phagocytic activity is unaltered, the ability to kill ingested organisms is decreased. An important step in the killing of bacteria by PMN's is the production of H₂O₂ (5), and under anaerobic conditions (low pO₂), the ability of the PMN to produce H₂O₂ is decreased (6). The present studies show that pO₂ is reduced in abscess cavities to a variable degree. Since even partial hypoxia has been shown to interfere with PMN function (1), some of the

pO₂ levels found would certainly be low enough to retard PMN bactericidal function.

Pus from the abscesses was consistently more reduced than the normal subcutaneous tissue fluid (7). This may explain in part the propensity for obligate anaerobic organisms to be found in abscesses.

Thus, we found that pus from experimentally induced abscesses was reduced and hypoxic in relation to normal tissues and that these factors may be important in the pathogenesis and persistence of abscesses.

Summary. Abscesses were induced in rats by injection of turpentine or turpentine plus bacteria. Other abscesses were induced by subcutaneous nitrogen injections followed by bacteria. pO₂ of the abscesses was diminished (mean 25.3 mm Hg, range 0-75 mm Hg), the pH was acid (mean 7.0, range 6.2-7.2), and the oxidation-reduction potential was lowered (mean +1 mV, range -20 to +40 mV). Since low pO₂ interferes with PMN bactericidal activity, this may in part explain the frequent failure of undrained abscesses to resolve.

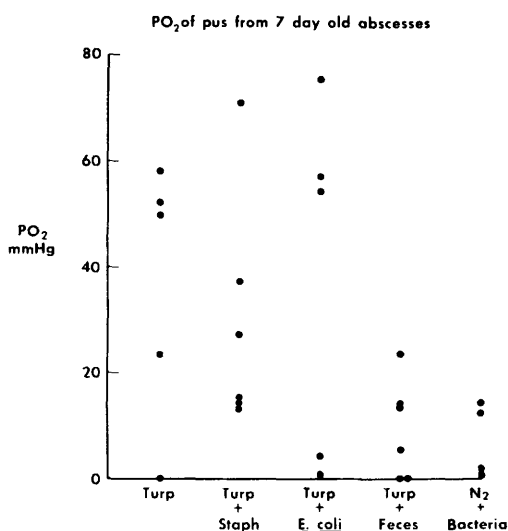


FIG. 1.

1. Mandell, G. L., *Inf. and Immunol.* **9**, 337 (1974).
2. Selye, H., *J. Amer. Med. Ass.* **152**, 1207 (1953).
3. Kunze, K., *Progr. Resp. Res.* **3**, 153 (1969).
4. Kempner, W., and Peschel, E., *Ztschr. F. Klin. Med.* **114**, 439 (1930).
5. Klebanoff, S. J., *J. Exp. Med.* **126**, 1063 (1967).
6. McRipley, R. J., and Sbarra, A. J., *J. Bacteriol.* **94**, 1417 (1967).
7. Fildes, P., *Br. J. Exp. Pathol.* **10**, 197 (1929).

Received March 19, 1974. P.S.E.B.M., 1974, Vol. 147.