



Fig. 9. Block diagram of the Bureau of Mines alkalized alumina process.

Fig. 10. Diagram showing the mechanical configuration of a Reinluft process pilot plant.

fects have been observed as a result of the protracted administration of N<sub>2</sub>O to control convulsions in tetanus victims. And, although any conceivable concentration of the gas as an air pollutant would be very low, the cumulative effects of continued exposure to this gas may be deleterious to human receptors. Catalysts and buffers. Dinman further observed that

In therapeutic medicine, bone marrow depressant ef-

with the addition of any suspended particulates, of less than 5 microns in size, the response of the human receptor to SO<sub>2</sub> may be accentuated. The reasons for this physiological reaction are twofold, and may be ascribed to

- 1. The interposition of particulate surfaces of relatively large areas for irritant gas adsorption, which would increase the gas concentration per unit volume.
- 2. The increased probability of impingement, which is a result of the different kinetic behavior of particulate vs.

Although many particulates tend to aggravate the biological damage potential of  $SO_3$ , it is apparent from actual case histories that the biopotency of these particulates is a function of their chemical properties. It is known that manganese catalyzes the conversion of SO<sub>2</sub> to H<sub>2</sub>SO<sub>4</sub>, but it is also possible for some particulates to buffer the physiological reaction of SO<sub>2</sub>.

In the phenomenon of buffering it is known that inhaled hydrogen sulfide (H<sub>5</sub>S) and other sulfhydryl com-pounds can protect mice from otherwise lethal exposures to ozone. Inhaled formaldehyde and SO2 produced much more resistance to air flow in guinea pigs when applied with a physiologically inert aerosol than did the same concentrations of the gases alone.

Knowing the concentration of a pollutant does not necessarily indicate its physiological effect upon a receptor. The presence of other contaminants may either inhibit or increase the expected effect. Yet, almost no research has been conducted to determine the effects on re-

However, in those persons with already thickened gas exchange membranes due to chronic lung disease, this increased thickness essentially causes a barrier to oxygen diffusion with resultant asphyxia, and further deterioration as a result of decreased oxygen availability to the vital

organs.
"... While there is much data from animal experimentation, there is relatively little human data even in normal persons. In such persons, 4-6 ppm of SO<sub>2</sub> produces consistently reproducible changes in airway resistance within 10 seconds to 4 minutes . . ."

Nitrous oxide is no laughing matter. Some medical experts regard nitrous oxide (NoO), the common "laughing gas" administered as an anesthetic by dentists for tooth extraction, in the same insidious category as SO<sub>2</sub>. Recent evidence indicates that N<sub>2</sub>O has teratogenic—and possibly carcinogenic—effects on animal and human receptors. In air pollution, this gas is emitted as a byproduct of hydrocarbon combustion.