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## ARTICLES

# Determinants of intrarenal oxygenation. I. Effects of diuretics

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To study renal cortical and medullary oxygen tensions, we used sensitive Clark-type O<sub>2</sub> microelectrodes, inserted by micromanipulators into the cortex and medulla of kidneys of anesthetized rats. As previously reported, under basal conditions, medullary PO<sub>2</sub> was significantly lower than cortical PO<sub>2</sub>.

Furosemide, which inhibits reabsorptive transport in the medullary thick ascending limb, increased medullary PO<sub>2</sub> from 16 +/- 4 to 35 +/- 4 mmHg ( $P < 0.0005$ ) without altering cortical PO<sub>2</sub>. This effect, reproduced by ethacrynic acid and bumetanide, was selective for loop diuretics and was directly due to decreased tubular O<sub>2</sub> consumption, since medullary blood flow was remarkably reduced by furosemide (-28 +/- 6% from baseline,  $P < 0.0001$ , as measured by a laser-Doppler probe). By contrast, acetazolamide, which decreases proximal tubule metabolism, selectively increased cortical PO<sub>2</sub>. These data are, in general, consistent with tubular metabolism as a major determinant of intrarenal oxygenation and suggest, in particular, that medullary reabsorptive work is at least in part responsible for renal medullary hypoxia.

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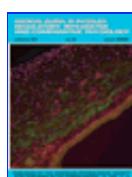
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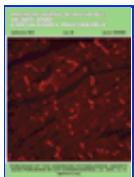


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