Control of the Splanchnic Circulation in Man

ROLE OF BETA-ADRENERGIC RECEPTORS

By Henry L. Price, M.D., Lee H. Cooperman, M.D., and John C. Warden, M.B.

ABSTRACT
Some effects of the β-adrenergic receptor blocker, propranolol, were studied in 20 normal, fasting, conscious men. The measurements made included cardiac output, splanchnic blood flow and oxygen consumption, arterial and hepatic venous blood pressure, and heart rate. The intravenous administration of propranolol (0.13 mg/kg) was followed by significant reductions in splanchnic blood flow and oxygen consumption, in cardiac output and in heart rate. Splanchnic perfusion pressure was unchanged; the splanchnic vascular resistance was significantly elevated. Previous treatment with glucose did not alter these findings. Phenoxybenzamine pretreatment lessened the increase in splanchnic vascular resistance which propranolol ordinarily caused. Ganglionic blockade with hexamethonium prevented all of the changes which propranolol produced in untreated individuals. These results may best be explained by assuming that the splanchnic circulation in man is influenced both by α receptors, which cause vasoconstriction when activated, and by β receptors, which when activated cause vasodilatation and increase oxygen consumption.

ADDITIONAL KEY WORDS
alpha-adrenergic receptor blockade
splanchnic vascular resistance
beta-adrenergic receptor blockade

One of us has reported (1) that the administration of propranolol (a β-adrenergic receptor blocking drug of high specificity) to human subjects anesthetized with cyclopropane caused a marked reduction in splanchnic blood flow. This reduction resulted from increased vascular resistance and was usually accompanied by a diminution in local oxygen consumption. Since cyclopropane is believed to increase the impulse frequency in sympathetic nerves supplying the abdominal viscera (2), these observations raised the question whether some of the activity of these fibers caused vasodilation and stimulation of metabolism. If this were true, the increase in vascular resistance attending the administration of propranolol could be explained as the result of reducing or abolishing tonic β-receptor stimulant activity which has, to date, remained undiscovered. In the experiments to be described, this possibility was examined and found to be likely.

Methods
The subjects studied were 20 healthy, adult, male volunteers. At preliminary meetings an informed consent was obtained, a medical history taken, and a physical examination, electrocardiogram, urinalysis and blood count were performed. On the day of study, each subject reported to the laboratory in the early morning, having fasted since the previous evening.

Under local anesthesia, a 100-cm no. 7 Lehman catheter was inserted into an antecubital vein and advanced as far as possible into a right hepatic vein and then withdrawn sufficiently (1-2 cm) to permit free aspiration of blood. In the opposite arm a 60-cm radiopaque no. 15 catheter, to be used for dye injection, was inserted percutaneously into a vein and positioned within the thorax.
### General Hemodynamic Changes Following Treatment

<table>
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<tr>
<th>Group</th>
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<th>Treatment</th>
<th>Heart rate (beat/min)</th>
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</table>

**Abbreviations:** TPR = total peripheral resistance; E₁ and E₂ = first and second experimental periods; C = control. Significance: referred to column at left.

Cardiac output was measured in duplicate by the indicator-dilution technique using a Waters densitometer and 5 or 10 mg of indocyanine green dye. Splanchnic blood flow was estimated by infusion of indocyanine green dye as described by Caesar et al. (3) with corrections introduced by Nielsen (4). No subject extracted less than 50% of arterial indocyanine green dye in a single hepatic passage. Splanchnic oxygen consumption was estimated by multiplying blood flow and the arteriovenous oxygen content difference as measured.
CONTROL OF THE SPLANCHNIC CIRCULATION

<table>
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<tr>
<th>Arterial pressure (mm Hg)</th>
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<th>TPR (mm Hg/liter per min)</th>
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The pH of arterial and venous blood were measured using an Instrumentation Laboratories electrode assembly model 113-S1. Blood glucose was determined by an enzymatic method.

During the 30-min study period, three determinations of splanchnic blood flow and single determinations of arterial and hepatic venous 

Pco2, Po2, pH, O2 content and CO2 content were made. Glucose concentration was estimated in 15 individuals. Concentrations of epinephrine and norepinephrine in arterial plasma were determined in 11 cases by the method of Price and Price (7).
Local Hemodynamics and Metabolic Changes Following Treatment

<table>
<thead>
<tr>
<th>Group</th>
<th>Subject</th>
<th>Perfusion pressure (mm Hg)</th>
<th>SBF (liter/min)</th>
<th>SVR (mm Hg/liter per min)</th>
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<td>P &lt; 0.05</td>
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Perfusion pressure = mean arterial minus mean hepatic venous pressure; SBF = splanchnic blood flow; SVR = splanchnic vascular resistance (perfusion pressure/splanchnic blood flow); \( Q_{02} \) = splanchnic oxygen consumption, \( P_{O2} \) = hepatic venous oxygen tension; RQ = respiratory quotient; and others as in Table 1.

The indocyanine green dye clearance technique estimates hepatic blood flow directly since the liver is the only organ capable of removing the dye from the circulation. The metabolic measurements, however, apply to the entire splanchic area, i.e., those viscera (liver, stomach, pancreas, gallbladder, small intestine, large intestine above the sigmoid colon and spleen) from which venous drainage eventually enters the hepatic vein.

Following the control period, the first 5 subjects were given propranolol (0.13 mg/kg iv) over a 10-min period after which the measurements detailed above were repeated. Five other individuals received 300 ml of 10% glucose solution (one half as a rapid intravenous infusion and the remainder slowly during the remainder of the study). Five subjects received hexamethonium (0.5 mg/kg in divided doses). A final 5 subjects were given phenoxybenzamine (0.7 mg/kg) by an infusion during a 20-min period followed by a 40-min wait (to permit maximal action of the drug to occur). In addition, all subjects received approximately 200 ml of 0.9% NaCl solution to replace blood taken in sampling. The dose of propranolol selected is the same as that used in our previous study (1); that of phenoxybenzamine was the largest amount which could be tolerated consistently without the occurrence of fainting. The last 15 individuals were subjected to three study periods, namely, (1) control, (2) following treatment with hemodynamic and metabolic measurements repeated as above, and (3) a period of 20 to 40 min after administration of the drug.
CONTROL OF THE SPLANCHNIC CIRCULATION

![Image](image-url)

EJ 67 47 51 67 53.6
P < 0.05

P < 0.05

Results

The principal results are shown in Tables 1 and 2. In Table 2 each value for flow, resistance, and perfusion pressure is an average of three individual determinations. In brief, the administration of propranolol to the fasting subjects (group I) was followed by a reduction in splanchnic blood flow, oxygen consumption, and indocyanine green dye clearance. Heart rate also was reduced. Calculated splanchnic vascular resistance increased, as did the splanchnic respiratory quotient, but perfusion pressure (mean arterial minus mean venous) was unaltered. Cardiac output was reduced in 4 of 5 subjects.

An infusion of glucose (group II) had no apparent effect, except to increase respiratory quotient, and the subsequent administration of propranolol caused the same directional changes as in the absence of previous treatment with glucose, except that respira-

Circulation Research, Vol. XXI, September 1967

![Image](image-url)
### TABLE 3

**Effect of Previous Treatment on Changes following Propranolol Administration**

<table>
<thead>
<tr>
<th>Previous treatment</th>
<th>SBF (liter/min)</th>
<th>SVR (mm Hg/liter per min)</th>
<th>P=0s (mm Hg)</th>
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<td>PBZ</td>
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<td>1.51</td>
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<td>P &lt; 0.05</td>
<td>P &lt; 0.01</td>
</tr>
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</table>

**Abbreviations:** Cyclo = subjects anesthetized with cyclopropane; PBZ = subjects who had previous treatment with phenoxybenzamine; others as in Table 2.

**Significance:** referred to control.

The respiratory quotient did not increase further in response to the blocking agent. Comparison of the magnitude of the various responses to propranolol showed no other significant difference depending upon previous treatment except that the increase in splanchnic vascular resistance was less (P < 0.01).

Phenoxybenzamine (group III) had no apparent initial effect except to increase heart rate. It apparently altered the response to propranolol as follows: (1) the respiratory quotient was still increased, but no longer consistently so, (2) the increase in splanchnic vascular resistance was smaller (P < 0.001), and (3) the reduction in blood flow was less (P < 0.05).

In contrast, hexamethonium pretreatment (group IV) apparently prevented all of the actions of propranolol that were observed in the untreated subjects and in those who received either the glucose or the phenoxybenzamine pretreatment. The direct actions of hexamethonium were to reduce splanchnic indocyanine green dye clearance, perfusion pressure, and blood flow.

In no subject was an elevated concentration of a catecholamine detected in arterial plasma; the levels in the fasting subjects ranged from zero to 0.12 μg/liter epinephrine and from zero to 0.48 μg/liter norepinephrine. Pao2 ranged from 73 to 107 mm Hg and Paco2 from 34 to 45 mm Hg. Arterial glucose concentrations ranged from 76 to 110 mg per 100 ml of blood before treatment and were unaffected by hexamethonium, phenoxybenzamine, or propranolol. During glucose administration the concentration in arterial blood averaged 147 mg per 100 ml of blood. The hepatic extraction of indocyanine green dye was unaltered by any of the drugs which were administered or by glucose.

**Discussion**

These results suggest that the splanchnic vasculature in man is influenced by sympathetic nerves which contain vasodilator as well as vasoconstrictor fibers, or by identical fibers which have both effects, depending upon whether α- or β-receptors are activated. Our evidence for this is as follows:

First, propranolol is an exceptionally specific β-receptor blocker and is apparently devoid of direct sympathomimetic or vascular actions (8, 9). Among its effects in this study were an increase in splanchnic vascular resistance and a reduction in blood flow and oxygen consumption. The present results (group IV) support the conclusion that these actions depend upon the presence of tonic sympathetic nervous activity, (i.e. are not caused by nonspecific drug actions). A possible exception to this statement would occur if propranolol, like certain α-receptor blocking agents, interfered with the uptake and restorage of the norepinephrine liberated upon the arrival of nervous impulses at sympathetic nerve terminals. This possibility has not yet been examined, but it is believed to be unlikely.

Second, propranolol can exert its effect in...
the presence of \(\alpha\)-receptor blockade. Although we have no proof that \(\alpha\)-receptor blockade was total and complete this finding apparently rules out the possibilities that propranolol acts only directly (via \(\alpha\)-receptor stimulation) or that it acts only reflexly (via increased \(\alpha\)-adrenergic nervous activity).

Third, there was no detectable level of epinephrine in the plasma of our subjects. The method used can detect a concentration of 0.1 \(\mu\)g/liter in plasma which corresponds to a secretion rate of about 0.1 \(\mu\)g/min, an amount believed to be physiologically insignificant (10). The rate at which norepinephrine is secreted from the adrenal medulla in resting man is also believed to be physiologically insignificant (10) and, moreover, the effect of infusing norepinephrine intravenously is to cause splanchic vasoconstriction, not dilatation (11). The effect of giving propranolol consequently could not have depended upon blocking an action mediated via circulating catecholamines.

Although \(\alpha\)-receptor blockade did not suppress the response of the splanchic vasculature to propranolol, it did reduce it, and this would favor either the existence of a tonically active sympathetic vasoconstrictor pathway in our subjects at rest or the reflex activation of \(\alpha\)-receptors by propranolol. It is interesting in that the previous study (1) performed in 5 anesthetized subjects, who had an abnormally elevated level of sympathetic tone, the administration of propranolol caused a significantly greater increase in splanchic vascular resistance than it did in the present investigation. Table 3 compares the effects of propranolol on splanchic vascular resistance, splanchic blood flow and venous oxygen tension in three groups of individuals studied by us who (presumably) had different initial levels of sympathetic tone. The data recorded during administration of cyclopropane were obtained in an earlier study (1), but have not previously been fully reported. The increase in resistance on giving propranolol is significantly augmented by cyclopropane administration and reduced by previous treatment with phenoxybenzamine.

These differences are reflected in quantitatively similar reductions in splanchic blood flow. It is of interest that the hepatic venous oxygen tension, while insignificantly affected by propranolol in fasting, conscious subjects, was conspicuously diminished in those receiving cyclopropane to a mean level approximating 30 mm Hg. From the standpoint of splanchic oxygen availability, it may be unwise to administer a \(\beta\)-receptor blocking agent when sympathetic nervous outflow is augmented.

The existence of both \(\alpha\)- and \(\beta\)-receptors in the splanchic viscera could also explain the curious result (12) that hemorrhage, although undoubtedly increasing sympathetic nervous activity, may not increase splanchic vascular resistance. Although it is stated that sympathetic vasodilator fibers do not supply the splanchic viscera (13, 14) the evidence for this statement is not convincing.

Since the vasodilator effects of \(\beta\)-receptor activation are accompanied by metabolic alterations, we were not surprised to find that the increase in splanchic vascular resistance caused by propranolol was accompanied by metabolic changes. In particular, \(\beta\)-receptor blockade resulted in a reduced splanchic oxygen consumption and an increased respiratory quotient. Splanchic oxygen extraction and the Po\(_2\) of hepatic venous blood in our normal, unanesthetized subjects were unaltered by propranolol, suggesting that the reduction in splanchic blood flow resulted from a quantitatively similar diminution in oxygen demand.

It has been estimated that roughly 80\% of the oxygen consumed by the liver is used to oxidize free fatty acids (15). Since the rate at which free fatty acids are oxidized depends directly upon their concentration in plasma (16), it is not unreasonable to expect that an agent, such as propranolol, which blocks the mobilization of free fatty acids (17, 18) will reduce hepatic oxygen consumption. In addition, the presence of circulating free fatty acids causes inhibition of pyruvate kinase, thus leading to gluconeogenesis (19). For this
reason the hepatic respiratory quotient should be reduced by the presence of free fatty acids and increased by any inhibition of free fatty acid mobilization. Thus, not only the circulatory, but also the metabolic, effects of propranolol may best be explained as consequences of the interruption of a tonic β activity.

With respect to the low splanchnic respiratory quotient values that we observed, our results resemble those of Rowell and co-workers (20). It is possible that ketosis accounts in part for this finding, since the formation of ketones from free fatty acids requires oxygen but does not produce carbon dioxide. The effects of glucose and propranolol on this ratio were those to be expected. We cannot explain the occasional apparent uptake of carbon dioxide by the splanchnic viscera, a phenomenon also noted by Rowell et al.

References

Control of the Splanchnic Circulation in Man: ROLE OF BETA-ADRENERGIC RECEPTORS
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doi: 10.1161/01.RES.21.3.333

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