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The effect of distention on blood flow through the intestine.

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THE EFFECT OF DISTENTION ON BLOOD FLOW

THROUGH THE INTESTINE

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INTRODUCTION

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There is universal agreement that pressure within the lumen of obstructed small intestine rises above normal (1, 2, 3, 4), and it has been suggested repeatedly that the increased pressure so impairs the circulation of blood through the intestine as to cause ischemic damage (5, 6, 7, 8, 9).

There is also universal agreement that distention of the gut under excessive pressures decreases the intestinal blood flow (8, 10, 11, 12). Attention has for the most part, however, been directed to the effect of pressures approaching the level of mean arterial pressure, which are considerably higher than the pressures usually observed in obstructed intestine. In the acutely obstructed small intestine of the dog, resting pressure is reported to range from 4 to 39.5 cm. of water, depending upon the level and type of the obstruction and the treatment of the animal (1, 2, 3, 4). During maximum contraction, pressure in the dog's obstructed small intestine has been reported to rise as high as 60 cm. of water (1). The latter value (approximately 44 mm. Hg.) probably represents the maximum lumen pressure developed at any time within obstructed small intestine in the dog.

The purpose of the present study was to re-investigate in greater detail the effect of pressures lying within the actually observed range.

METHODS

METHODS

Barbitalized dogs were used in all experiments.
Either carotid or femoral blood pressure records were
taken. The abdomen was opened widely in the mid-line
and the intestine exposed. Loops of lower ileum 6 to 12
cm. long, located 20 to 30 cm. from the cecum were isol-
ated between ligatures, brought outside the abdomen,
coated with liquid petrolatum, covered with cotton and
kept warm by means of a lamp.

The lower part of the superior mesenteric artery
was dissected enough to allow for placement of a con-
striction above the branch to the loop, and cannulation
of either another branch or the end portion of the art-
ery. All other branches from the artery below the con-
striction were tied off so that all blood flowing past
the constriction went to the part of the gut under study.
This arrangement is illustrated in figure 1. The cannula
downstream from the constriction was led off to either
a separate mercury manometer or to one limb of a diff-
erential manometer. The other side of the differential
manometer or another mercury manometer was connected
with the femoral or the carotid artery. When a differ-
ential manometer was employed, this connection was made
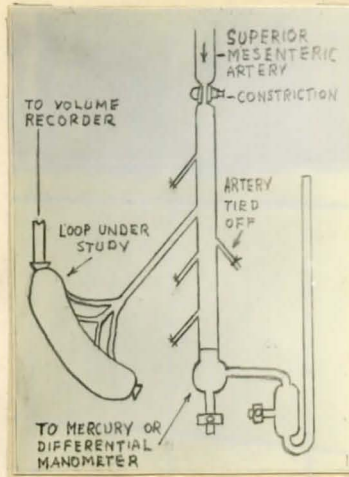through a Y tube, the other arm of which supplied the

Figure 1. Diagram of Placement of Constriction
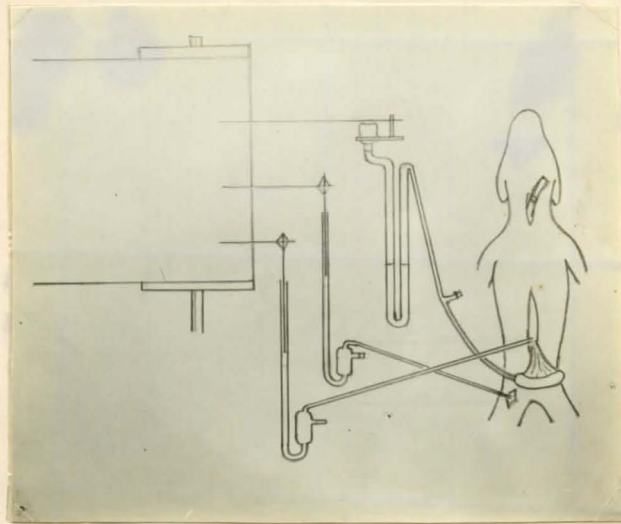and Mesenteric Manometer in Relation to Loop of
Gut under Study.

Figure 2. Diagram of Connections for Use of
Two Mercury Manometers.

record of systemic arterial pressure from a mercury man-
ometer. These connections are shown in figures 2, 3 and
4.

When a constriction is placed on the superior
mesenteric artery as shown in figure 1, a fall in press-
ure is recorded by the mesenteric manometer. This diff-
erential may be adjusted by tightening or loosening the
constriction as necessary to secure a drop of around 20
mm. of mercury. Such a drop creates a sufficiently sens-
itive flow meter and at the same time allows enough flow
to maintain the gut in good condition. A small screw
clamp was devised which permitted accurate adjustment
and appeared to cause little damage to the artery.

As an anticoagulant, the azo dye, chlorazol fast
pink * was used. This dye was purified according to the
method of Modell (13) and was injected into the cannulae.

The method of differential manometry, as employ-
ed by Lawson and Holt (14) was used in three modified
forms. The first set up consisted of two mercury manom-
eters of the single arm type. These manometers were
connected as in figure 2, in which one manometer records
carotid or femoral blood pressure, while the other re-
cords pressure in the superior mesenteric artery below

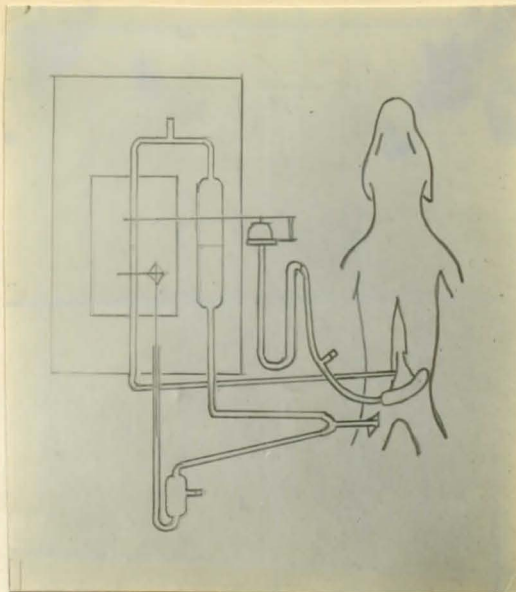* Marketed as Fastusol Pink by the General Dyestuff Corp-
oration.

4.

Figure 3. Diagram of Connections for Use of
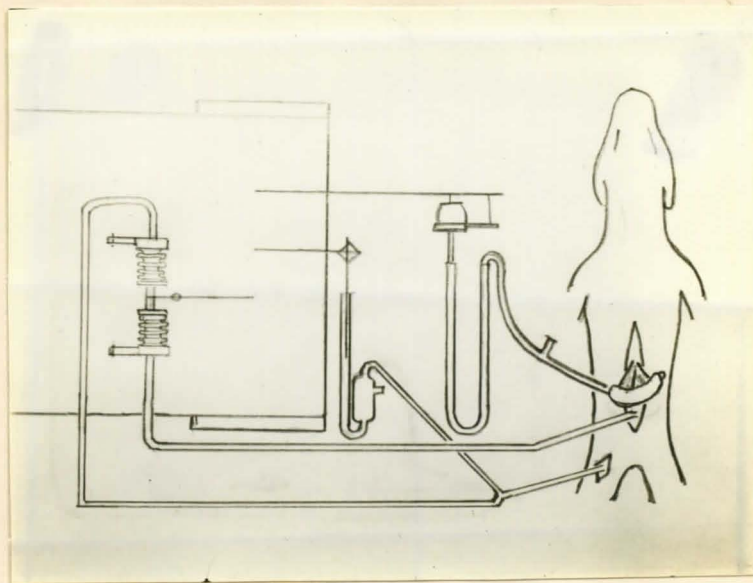Optically Recording Differential Manometer.

Figure 4. Diagram of Connections for Use of
Metal Bellows Differential Manometer.

the constriction. With the two manometer arrangement
the pressure difference at any time in the record is
measured by comparison on simultaneous ordinates. These
pressure differences may then be plotted out as a flow
curve above an arbitrary base line.

Because of the difficulty in maintaining a con-
stant constriction on the artery, in a few experiments
the femoral artery was cannulated and its flow led into
the superior mesenteric artery by means of rubber tub-
ing as shown in figure 5. By this means an easily ac-
cessible and adjustable screw clamp could be used to
create a constriction on the rubber tubing. In these
experiments, chlorazol fast pink in a dose of 100 mgm.
per kilogram was given intravenously.

The second method of differential manometry
made use of a modified Venturi meter which was so con-
structed that it functioned as a single arm manometer.
This was set up as shown in figure 3 and recorded optic-
ally by methods shown in figure 6. This manometer reg-
istered the pressure difference directly, increased
flow dropping the level of the fluid in the small arm.
Alcohol was used in the small arm of the manometer to
secure rapid drainage from the sides of the tubing. The
light used in this system passes through the small arm
of the manometer. The lower part of the tube, being
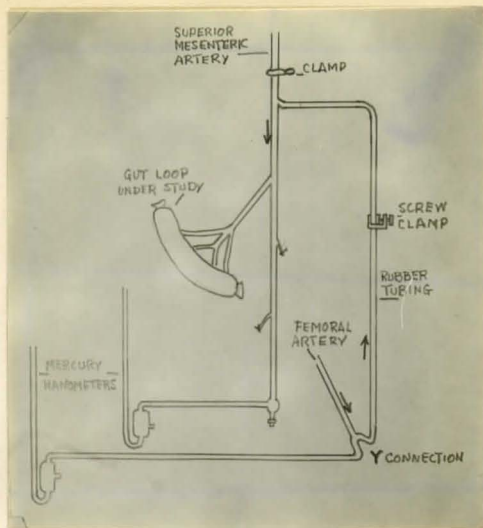filled with fluid, acts as a cylindrical lens. The rays

Figure 5.   Diagram of Connections for Use of
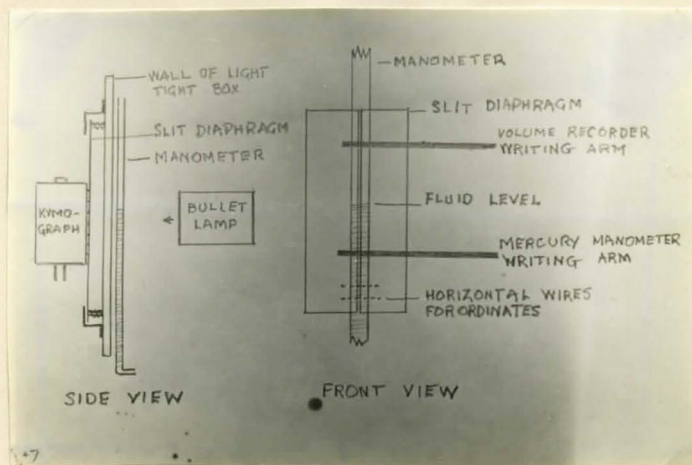Screw Clamp as Constriction on Rubber Tubing.

Figure 6.   Diagram of Essentials of Optically
Recording Differential Manometer.

are not brought to a focus on the sensitive paper but are merely allowed to fall, out of focus, on a sheet of tin which has in it a slit about 1 mm. wide. This slit diaphragm rests directly on the drum of the kymograph carrying the bromide paper. Since the light is slightly concentrated by the alcohol, the completed record shows flow as the boundary between a lower dark area and the upper, lighter area. Systemic blood pressure is recorded as the shadow of the writing arm of a mercury manometer in front of the flow meter. Intestinal distention was recorded as the shadow of the writing arm of a volume recorder connected with the open limb of a suitable manometer. This type of flow meter has as its chief advantage the fact that constriction of the artery sufficient for satisfactory sensitivity causes only slight decrease in the flow to the gut. The decrease in pressure below the constriction is approximately one thirteenth the decrease necessary to produce equal sensitivity when using mercury manometers. A serious disadvantage is that the records are not visible as they are made.

The third type of differential manometry employed two small bellows made of thin brass. These bellows were mounted in tandem and a writing lever fixed between their abutting ends. This system also records the pressure differences directly. It operates on the same principles

as the one described by Lawson and Holt (14) but is an improvement in that there is no rubber diaphragm to undergo elasticity changes. The bellows were filled with water and only the arterial cannulae received anticoagulant dye.

Large distentions were carried out by inflating with air a thin rubber balloon in the intestine against a mercury manometer. The pressure and the motility of the intestine were recorded from a volume recorder connected with the open arm of the manometer. With smaller distentions a water manometer was used. In order to maintain distention pressure constant in later experiments, the gut was inflated with water from a leveling bottle connected with the volume recorder.

In each of the methods of measuring arterial flow used, the flow is recorded in terms of difference of pressure. In order to convert this into volume flow it is necessary to calibrate the set up as suggested by Lawson and Holt (14). Since the value of the constriction may change in the course of an experiment, frequent calibration is required. As this involves damage to the intestine during the periods when its flow is shut off, calibration was usually not done.

Venous outflow was recorded directly in dogs which had received chlorazol fast pink as anticoagulant. Drops were recorded manually or with a pneumatic drop

counter or venous blood was allowed to drain into a bottle
to which was connected a volume recorder which recorded
air displacement.

RESULTS

RESULTS

Except to confirm the effects of high distention pressures described by other workers, attention was directed to the response to pressures below 60 mm. Hg. In a series of eighteen dogs, repeated brief inflations, varying in duration and pressure were carried out in order to establish typical responses. No inflations were maintained longer than 15 minutes, the usual duration being 2 to 5 minutes. For descriptive purposes the changes in flow during and following a brief period of inflation may be divided into five phases (fig.7).

Phase 1: Inflow usually began to fall off immediately with even small increases in lumen pressure (10-30 mm. Hg.), reaching the peak of reduction 8-10 seconds after the peak of lumen pressure. Coincident with the decreased inflow, there was an increase in venous return (fig.8).

Phase 2: Usually with inflations below 20 mm. Hg. and sometimes with considerably higher inflations, flow returned to control levels within about 10 seconds of the end of phase one, the whole response occupying 15-30 seconds (fig.9). With higher distention pressures, the phase of returning flow was prolonged. With sustained distentions between 20-30 mm. Hg., control flow levels were us-
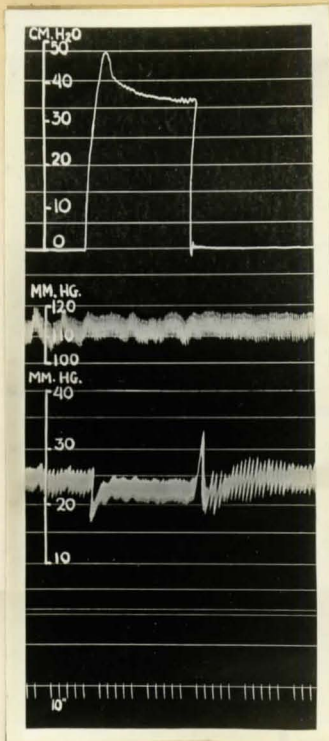
Figure 7. Typical Effect of Moderate Distention. From above down; Distention pressure in cm. H_2O; Systemic arterial pressure in mm. Hg.; Blood flow as pressure differences in mm. Hg. by metal bellows manometer; Time in 10 second intervals.
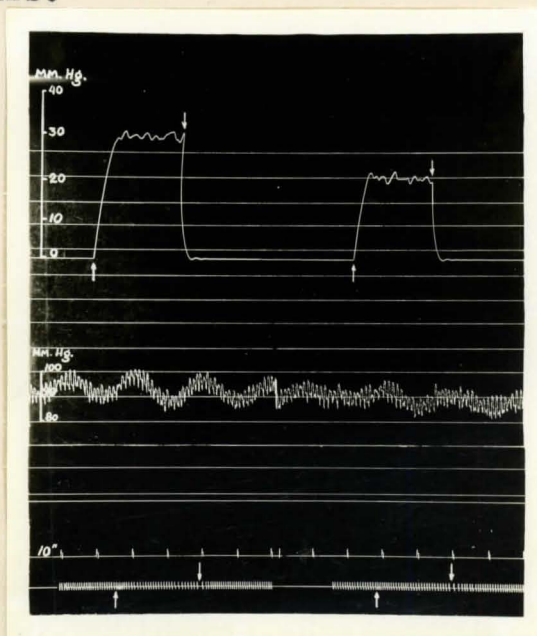
Figure 8. Effect of Distention on Venous Outflow. Construction as in fig. 7, but without inflow. Outflow at bottom, by drop recorder.
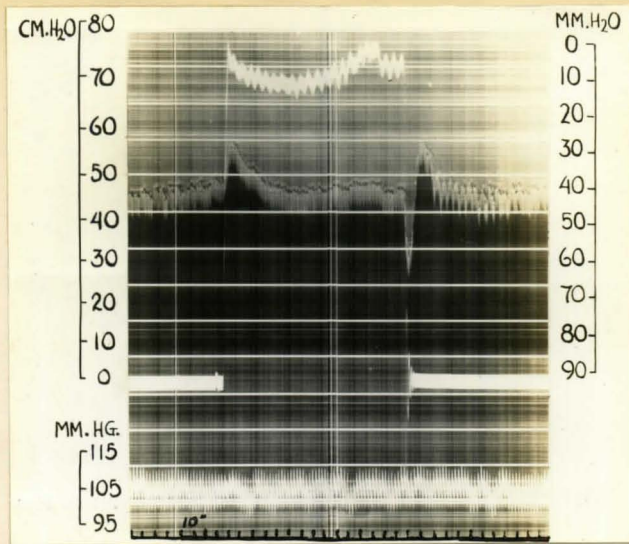
Figure 9.   Complete Compensation of Blood Flow During
Distention. Records from above down: Flow as pressure
differences in mm. water, by optical method: Distention
pressures in cm. water: Systemic arterial pressure in
mm. Hg.: Time in 10 second intervals.
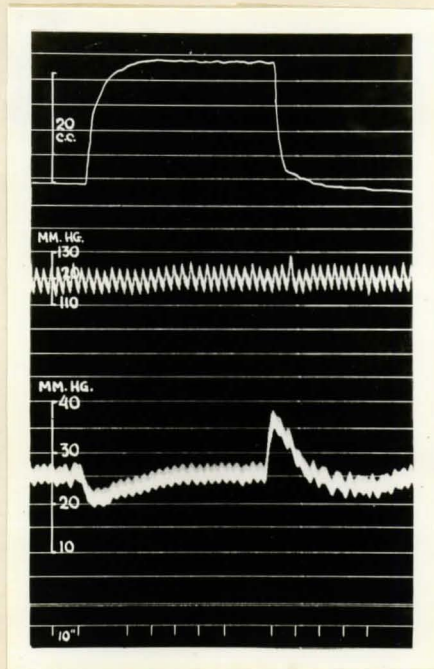
Figure 10.   Slow But Complete Compensation During
Distention.   Records as in fig. 7, except top record
is volume water accepted by gut from leveling bottle
at 40 cm. water distending pressure.

ually reached within 1-3 minutes (fig.10). With sustained distentions above 30 mm. Hg., flow usually reached a plateau, below the control, within 1-3 minutes. Throughout phase two, changes in venous return paralleled changes in inflow.

Phase 3: On deflation there was usually a sudden increase in inflow over the control level which reached its peak at the moment deflation was complete (fig.9). At the same time, venous outflow was decreased. Phase three appeared as brief post-deflation hyperemia even though flow had returned to the control level during the second phase. With brief (less than 20 seconds) or low pressure (less than 30 mm. Hg.) distentions, the third phase terminated the response, flow returning to control levels in 5-90 seconds (fig.11).

Phase 4: The hyperemia of phase three was often interrupted by a period of reduced inflow, during which strong contractions of the gut occurred (fig.12). This fourth phase was never observed in the absence of post-deflation hypermotility. Its duration was 10-50 seconds. The reduction in flow during this period was sometimes greater than at any time during the inflation. In the small group of dogs on which outflow measurements were made, phase four was not present. Hence observations on outflow during this phase are not available.
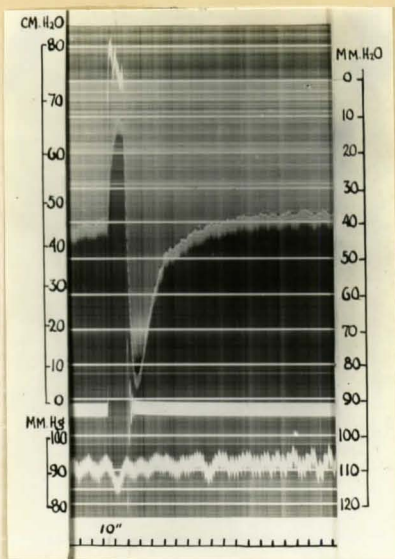
Phase 5: When the third phase was interrupted by

Figure 11. Termination of The Response with Phase Three.
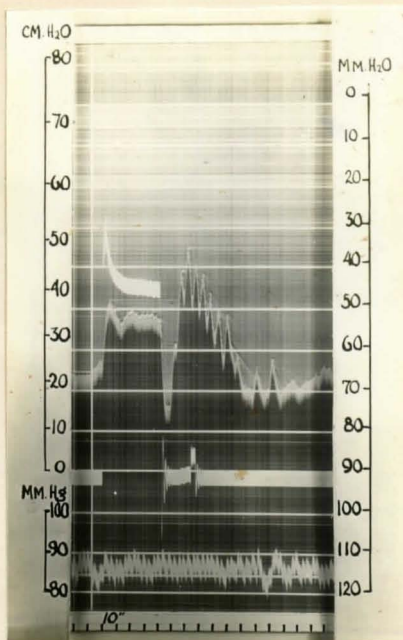Construction as in figure 9.

Figure 12. Showing An Exaggerated Phase Four. Construction as in figure 9.

the occurrence of phase four, the second portion of the hyperemia appeared on the records as a distinct fifth phase (fig.13). The greatest duration of the post-deflation period, including phases three, four and five, observed in our experiments was six minutes.

Atypical Responses: In four animals, distentions below 50 mm.Hg. consistently produced an increase in flow through the gut (fig.14). Two of these had undergone preliminary mesenteric denervation at least an hour before starting the experiment, and the loop of intestine was active and in high tonus. In three of these animals an increase in flow began immediately with the distention (fig.15). In the remaining animal, the increase over the control flow developed during phase two (fig.14). In all, the increase in flow persisted for 30-200 seconds after deflation, the flow level during phase five being nearly the same as the level reached during phase two. The post-deflation behavior (phases three, four and five) of the loops whose flow was increased during the distention was indistinguishable from that of loops whose flow was decreased (fig.14).

The Response to Inflation Under Constant Pressure: Although no parallelism was observed between the compensatory return of flow and the fall in lumen pressure during phase two, lumen pressure was held constant in the latter part of the study by inflating the loop with water from a
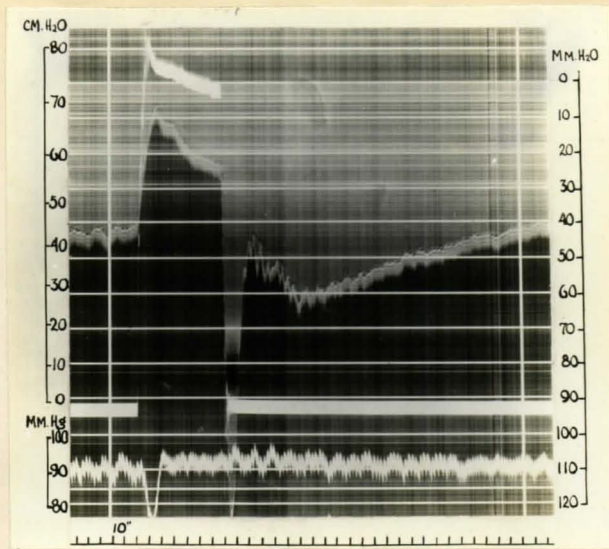
Figure 13. The Appearance of Phase Five. Record as in fig.9.
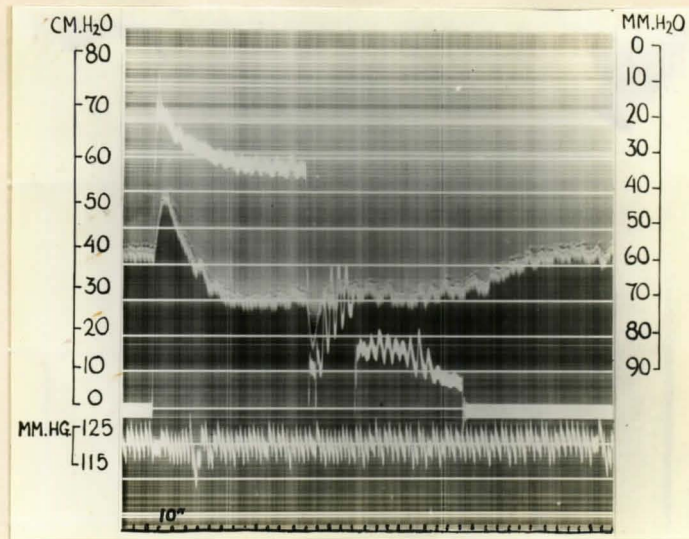
Figure 14. An Increase in Blood Flow During Distention. Record arranged as in fig. 9.

leveling bottle. In these experiments, with inflation pressure constant, the changes in flow during and following distention were not distinguishable from those in which lumen pressure fell as the gut enlarged (fig.10).

The Effect of Denervation: To investigate the possibility that distention acts through spinal reflexes to influence the response, an experiment was done with two loops of gut, one of which was inflated while inflow was measured in the other. Flow through the undistended loop was unaffected by inflation of the other. The possibility remained, however, that spinal reflexes modifying the response were strictly segmental in character. In ten animals, after typical responses had been elicited to various levels of inflation, the loops were denervated by stripping the artery and vein in the mesenteric pedicle, sometimes painting the vessels with 20% phenol in alcohol. In none of these was it possible to detect a significant change in the response.

The Effect of Stretch: After control observations had been made, in three dogs the loop was encased in plaster of Paris in order to study the effect of intra-lumen pressure uncomplicated by volume changes in the loop. In all cases, elevation of lumen pressure produced a simple, monophasic reduction in flow persisting throughout the period of inflation, with no tendency to return toward the control. The typical post-deflation behavior

18.

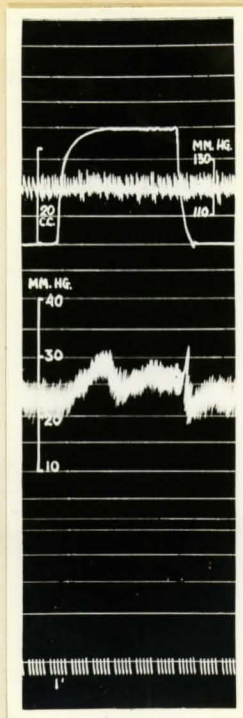Figure 15.   Immediate Increase in Flow on Distention.
From above down: Systemic blood pressure: Leveling
bottle record at 20 cm. H_2O distention pressure: Flow
by metal bellows manometer: Time record.
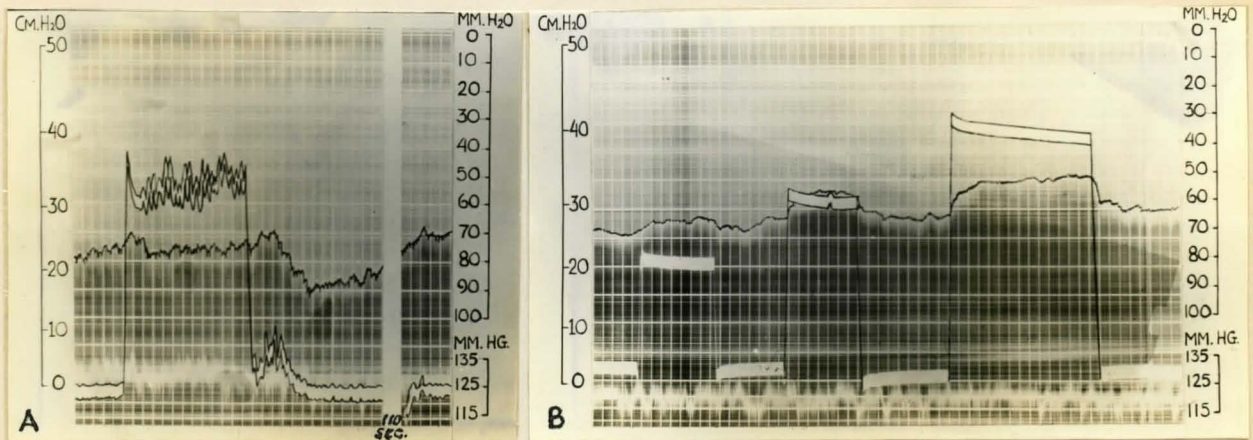
Figure 16.   Modification of Response by Encasing Gut
in Plaster.   A. Control, without cast.   B. Three in-
flations in cast.   Record as in fig. 9.

of the freely distended gut was never observed, flow
returning abruptly to control levels on deflation (fig.16).
That this is not due to absence of reactivity was shown
by the observation that clamping the artery for a period
of 90 seconds set up, on restoration of the flow, typical
reactive hyperemia.

In these experiments, any pressure applied to the
lumen is immediately transmitted to all layers of the gut
wall. With the gut free to enlarge, inflation pressure
is gradually applied to the outer layers as the muscular
layer is stretched. With lumen pressure constant there-
fore, it would be expected, from a purely mechanical stand-
point, that as the gut enlarges under inflation, flow
would progressively decline, reaching its greatest re-
duction at the time of greatest distention. Since the
behavior of the enlarging gut under inflation was the re-
verse of this, it was decided to study the effect of
stretch alone on flow.

For this purpose a short, decentralized loop was
laid open along its antimesenteric border and the cut
edges placed in bulldog paper clamps. In four dogs,
transverse stretch at the first trial caused a pure in-
crease in flow. In all of these, however, on successive
trials there was gradual loss of this response, and re-
placement by a response not unlike that to inflation as
tone in the intestine, increased by the trauma of the

preparation, deteriorated (fig.17). After application of pilocarpine to the intestine, restoring tone, transverse stretch again caused an increase in flow. Atropine reversed the effect of pilocarpine (fig.18).

These experiments demonstrate the existance of a mechanism set in operation by stretch, at least in the intestine in a state of high tone, which reduces the resistance in the walls of the intestine to the flow of blood. Since, furthermore, the intestine inflated in a plaster cast which prevents stretch, shows greater and more sustained reduction in flow than one which is permitted to enlarge, it seems likely that the stretch mechanism is largely responsible for the circulatory adaptation of the distended intestine.

The Effect of Local Anesthetics: It is conceivable that local reflexes set up by stretching the intestinal walls bring about vasodilatation which more or less compensates for the mechanical increase in resistance to flow in the distended loop. To test this possibility, the response of the gut was compared before and after application of 1% cocaine to the mucosa. In all cases, the behavior of the cocainized loop when distended, was strikingly similar to that of the loop encased in plaster (fig.19). That the modification of the response by cocaine was due neither to its sympathomimetic action nor to its paralytic effect on the muscular walls
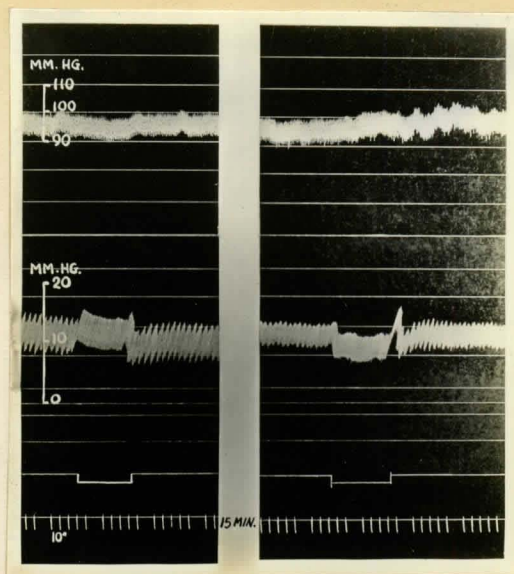
Figure 17. A. Increase in Flow Resulting from Stretch. B. Decreased Flow Resulting from Stretch Later in Experiment. From above down: Systemic blood pressure; Blood flow; Signal during stretch; Time.
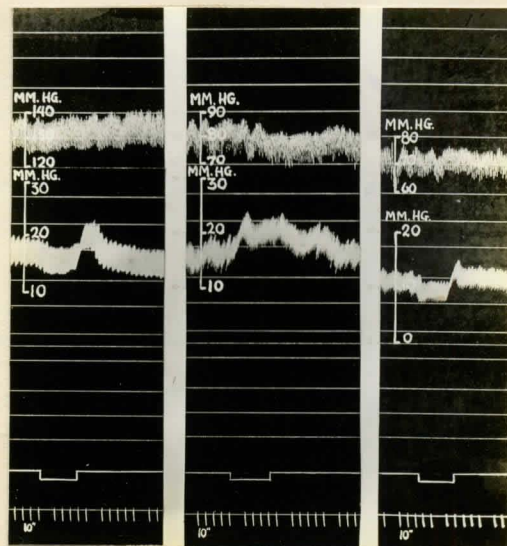
Figure 18. A. Decreased Flow Resulting from Stretch of Gut in Low Tone. B. Increased Flow Resulting from Stretch After Pilocarpine. C. Decreased Flow Resulting from Stretch After Atropine. Record as in fig. 17.
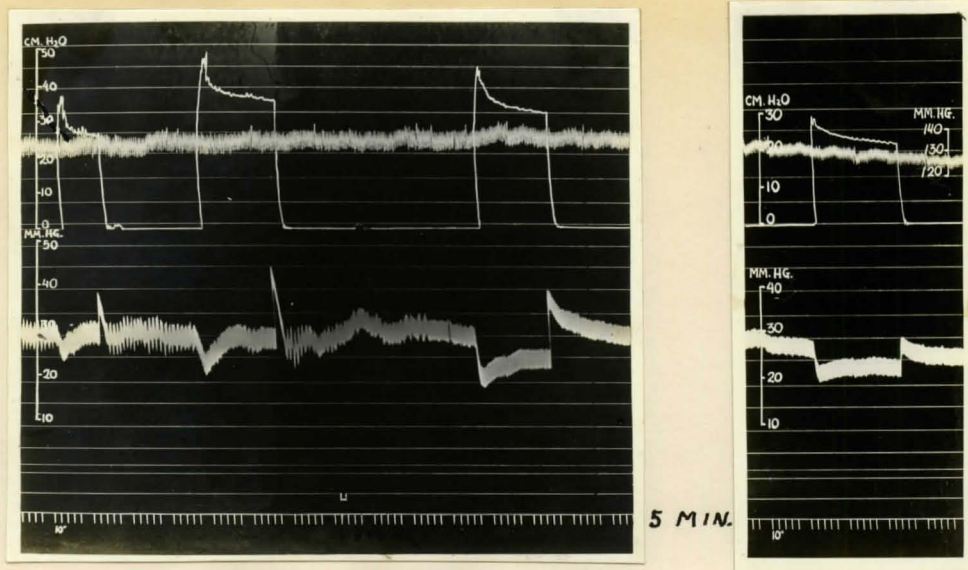
Figure 19. Modification of Response by Cocaine. From above down; Systemic blood pressure; Distention pressure; Blood flow; Signal for application of cocaine; Time.
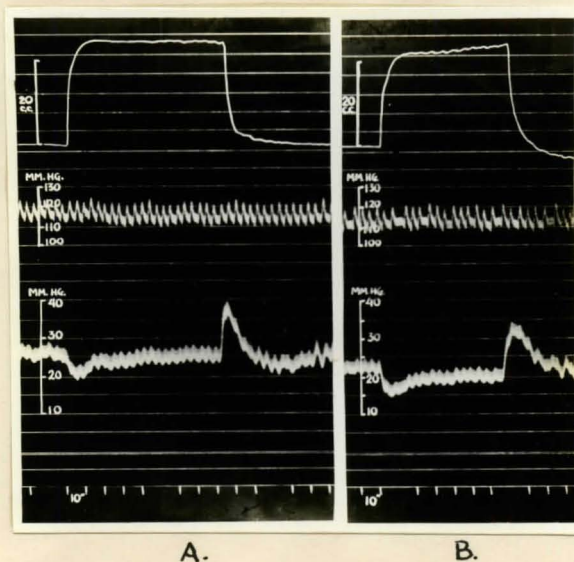
Figure 20. Modification of Response by Procaine. A. Before procaine. B. After procaine applied. Record arranged as in fig. 10. Distention pressure 40 cm. H_2O.

of the gut is suggested by the observation that procaine
applied to the mucosa produced similar modifications in
the response (fig.20).

DISCUSSION

DISCUSSION

The calibre of blood vessels depends upon their
coefficient of elasticity, the lateral pressure within
them, and the pressure exerted upon them from the out-
side (15). Although in the case of the larger vessels,
with a diameter of 5 mm. or more, a reduction in diam-
eter of nearly 50 percent is required to produce a sig-
nificant reduction in flow (16), in the smaller vessels,
such as the arterioles, capillaries, and venules, any
change in calibre produces changes in flow. It would be
expected, therefore, from the standpoint of theoretical
hemodynamics, that any increase in the extravascular
pressure exerted on a vascular bed would decrease blood
flow through the field by narrowing the smaller or small-
est vessels. This is actually true in some fields, such
as the lungs. That it does not appear to be true in
some others, such as the cranial circulation, has not
been adequately explained (17).

The intestine encased in a plaster jacket has its
blood flow uniformly reduced throughout a period of in-
creased intra-lumen pressure, as would be expected from
theoretical considerations. Although quantitative data
were not obtained, the decrease in flow, in terms of
pressure difference, appears to be proportional to every

rise in lumen pressure above atmospheric. It persists without further change until deflation.

This is in striking contrast with the behavior of the intestine which is permitted to distend under inflation. Although the initial decrease in flow upon inflation appears to be at least as great as in the encased gut, there is always some return of flow within a few seconds. With inflation pressures below about 30 mm. Hg. complete restoration of flow is usually achieved within 30-90 seconds. With higher inflation pressures up to the level of mean arterial pressure, return of flow is usually incomplete, but otherwise resembles, in its appearance on the records, the complete compensation for lower pressures. No study has been made on the flow changes with pressures above mean arterial pressure. It is not clear from their description whether this return of flow is the same as that reported by Dragstedt and his coworkers (12), and interpreted as a shunting of blood through the mesentery.

The return of flow under sustained lumen pressure as the gut distends is difficult of explanation upon a strictly extravascular hemodynamic basis. If it be assumed that the muscular layers of the gut, in a state of tonus, act along with the tough submucosa to protect the outer layers from lumen pressure, it would be expected that with increasing enlargement of the gut, pressure on

the outer layers would progressively increase, and flow
would progressively decline as the gut enlarges. The
penetration of the muscular layers by branches of the
vasa recta would favor angulation and occlusion, with
further reduction in flow, in the over-distended intest-
ine (5). In order to explain a partial return of flow
in the distended gut, on an extravascular basis, it would
be necessary to assume that distention so completely in-
hibits intestinal muscle tone that all the stress is
taken up by the submucosa and mucosa, pressure in and ex-
ternal to the muscle layers remaining constant. To pur-
sue this explanation further, a full return of flow under
inflation, or an increase in flow as was occasionally ob-
served, might be due to such inhibition of muscle tone as
actually to reduce pressure in these layers below control
levels, thus compensating, or over-compensating, for the
rise in pressure on the inner layers.

An alternative explanation assumes the existence
of local nervous mechanisms, set in operation by stretch
of the gut wall, which bring about vasodilatation to
compensate for the increased extravascular pressure.
That the compensating mechanisms are intrinsic to the
gut is shown by their persistence after mesenteric de-
centralization of the loop. The abolition or marked im-
pairment of compensation by the local anesthetics cocaine
and procaine supports this interpretation, but can be

used equally well in support of the non-vascular interpretation given above, if it be assumed that these local anesthetics impair local nervous mechanisms mediating the inhibition of intestinal muscle tone by inflation .

That the post-inflation hyperemia is not simply reactive hyperemia, in repayment of a flow deficit incurred during the inflation is shown by its absence after moderate inflations of the encased gut, its absence in the moderately inflated cocainized gut, and its occurrence in untreated intestines which had shown no flow deficit during inflation. It appears from the preliminary data available that the flow deficit in the intestine must reach a certain critical value before reactive hyperemia occurs, and that the deficit incurred with moderate distentions even in the encased and the cocainized gut is usually below this critical level. Excessive and prolonged distentions did, in the encased and the cocainized intestine, produce reactive hyperemia on deflation, showing that the gut is capable of reacting under these conditions.

If the hyperemia at the termination of a period of moderate distention is not reactive hyperemia, it probably represents persistence of the compensating mechanism beyond the period of distention. If the compensating mechanism were entirely extravascular, and de-

pendent upon inhibition of intestinal muscle tone, no post-distention hyperemia should occur, since the deflated intestine goes promptly into a brief period of hypertonus and hypermotility, then returns to control levels. Throughout this entire post-inflation period, for 30 seconds to 5 minutes, flow may be above normal. If the compensating mechanism were vascular however, it might be expected to persist after removal of the stimulus (distention). The behavior of the deflated gut is therefore in accordance with the theory that intrinsic vasodilator mechanisms are set in operation by distention. Upon the background of persistent vasodilatation, deflation hypermotility and hypertonus temporarily reduce the blood flow as would be expected from the observations of Anrep, Cerqua and Samaan (18), to introduce the fourth phase of the response.

The observations on simple transverse stretch, although clearly demonstrating that enlargement of the circumference of the intestine is capable of increasing blood flow, throw little light upon the mechanism. Since stretch in these experiments increased flow only when the intestine was in a state of high tonus, the extravascular interpretation of the mechanism may be regarded as favoured. Only in an intestine in high tonus would it be possible for muscular inhibition to occur so that extravascular pressure within the muscular lay-

ers falls below normal. It is difficult to conceive of a reflex mechanism in the gut wall, whose receptors are stimulated by stretch only when the gut is in high tonus. A complete study on the relationship between the compensating mechanism and intestinal muscular tone will have to be done in order to clear up this point.

The bearing of these data on the ischemic theory of intestinal damage in obstruction is questionable. It is clear from the data that under these experimental conditions, pressures such as have been observed in obstructed intestine in the dog do not reduce flow. The critical level of distention pressure above which there was incomplete compensation is about 30 mm. Hg., variations from this being for the most part above rather than below, many animals compensating completely for pressures as high as 40 mm. Hg. But the data also show that this critical level is subject to change by encasing the gut in plaster, or treating it with procaine or cocaine. Preliminary data on muscle tone suggest that if the gut is in high tonus after denervation or treatment with pilocarpine or eserine, flow may actually be increased by moderate distentions, the critical level for reduction being markedly elevated. It is therefore possible that in the intestine damaged by obstruction, the compensating mechanisms are impaired, so that even low lumen pressure can

cause ischemia. It would also be valuable to obtain
data on prolonged distentions below the critical level
under the present conditions, to determine susceptib-
ility of the compensating mechanisms to fatigue.

SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSIONS

The effects of moderate distention under pressures up to 60 mm. Hg. on blood flow through the intestine were studied by differential manometry and by measurement of venous return in acute experiments on barbitalized dogs.

Typically, more or less complete return of flow to control levels, following temporary reduction, occurred during the inflation. Usually the return of flow was complete with any distention below 30 mm. Hg., incomplete with greater distentions. A prolonged period of hyperemia usually followed deflation. It appeared to be unrelated to the flow deficit incurred during the distention, occurring without significant modification in a few experiments in which flow was increased during the distention.

Neither the compensatory recovery of flow during inflation, nor the post-inflation hyperemia appeared to be significantly modified by mesenteric denervation.

Enclosing the intestine in a plaster jacket to prevent distortion of the walls during inflation, prevented completely the return of flow during distention, and with moderate inflations, completely prevented the post-inflation hyperemia.

32.

Application of cocaine or procaine to the mucosa either prevented the return of flow during distention and the post-inflation hyperemia, or greatly reduced both.

Transverse stretching of the opened intestine, when the intestine was in a state of tonus, produced a simple increase in flow, which persisted for some time after the intestine was released.

It is concluded that a mechanism is set in operation in the intestine when its walls are stretched, which increases the flow of blood through the intestine. This mechanism appears to compensate for the increased resistance to flow offered by the inflated intestine, the compensation being complete below a critical level of lumen pressure of 30 mm. Hg. under the conditions of these experiments.

Since the compensating mechanism is abolished or impaired by local application of cocaine or procaine to the mucosa, it is suggested that it depends upon intrinsic nervous mechanisms.

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The Effect of Distention on Blood Flow
Through the Intestine

Approved by Reading Committee composed of
the following members:

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