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Secondary traumatic shock

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Introduction

From earliest times medical men noted a peculiar clinical
picture following many surgical procedures or following
some sudden and severe injuries. Many names were given
to this condition. James Latta, in 1795, used the word
"shock". Since then that term has been used and still
is being used to designate the above noted condition.

In this paper the various views of shock will be considered
from both angles. In this manner the reader should get
a general idea as to what is being done, both experi-
mentally and clinically, with secondary traumatic shock.

Definition

Considerable dispute still reigns over the definition
of shock. There seems to be a clinical syndrome which
most workers agree to call shock, yet some differ in
defining this condition. We must differentiate clearly
between primary and secondary shock. Primary shock
is defined by Holt (38) as a circulatory collapse due
to inhibition of constrictor tone by influences operat-
ing throughout the nervous system. It is characterized
by a rapid onset associated with a fall in blood pressure,
but without a decrease in blood volume. For one to two
hours the blood pressure may be too low to read.

"Psychogenic" shock (Phemister and Livingston 54) is
the result of a psychic distress--a physical strain
without associated tissue injury to any extent. This
is characterized by a low blood pressure, slow pulse,
shallow respirations, and fainting. As a result of
the fainting the patient falls; the horizontal position
restores the cerebral circulation and recovery is
usually prompt. In cases where the patient is origin-
ally horizontal, no fainting results. The above
stated workers (54) report a case of an operation of
a chondroma of the phalanx. On the first operation

primary shock was experienced by the patient. During
the second operation the blood pressure did not fall
from the normal level. It is believed that the psychic
factor is the cause but that the pain helps in the
precipitation of the shock. Psychic shock is rarely,
if ever, the cause of death in healthy individuals.
Primary shock will not be discussed any more. It is
mentioned merely to distinguish it from secondary
shock.

In this paper we are concerned primarily with secondary
shock which presents itself clinically by a low blood
pressure, low basal metabolic rate, increased cardiac
rate, and diminished renal excretion. Later, the
coaguability and the oxygen of the blood are decreased,
the blood chlorides fall below normal and the blood
non-protein nitrogen increases. Clement (18).

A more complete picture of the clinical syndrome of
secondary shock given by Makel (44) is increased
pulse rate, low blood pressure, exposed parts pale,
and a tinge of cyanosis, sweating, thirst, restlessness,
and increased respiratory rate. In severe shock there
may be air hunger owing to the clouding of the sensorium.

The above are really descriptions of the clinical state

and not definitions of the condition. McKesson, according to Perkins (53), defines shock as "a decompensation of the muscular elements of the cardio-vascular system leading to malnutrition and exhaustion".

Moon (51) defines shock as "a circulatory deficiency, neither cardiac nor vaso-motor in origin, characterized by decreased blood volume, decreased cardiac output (reduced volume flow) and by increased concentration of the blood". This definition seems very satisfactory; it rules out all shock states which are cardiac or vaso-motor in origin. Immediately primary shock is ruled out and we are dealing with secondary shock.

History

In Crile's book on Surgical Shock (20) he lists the
following early workers connected with surgical shock:
James Latta in 1795 was the first to use the word shock
in the sense that it is now employed. In 1827 Travers
described a certain form of shock as "prostration with
excitement". In 1834 J. A. Delcasse thought that the
effects of violence were transmitted chiefly through
the osseous system whereby lining molecules were sepa-
rated from each other and shock was produced. Erichsen,
in 1864, explained shock in accidents due to the "sharp
vibration that is transmitted through everything".
Later came Vernevil who declared that shock is a series
of phenomenon following trauma of tissue, characterized
by a temporary depression of the functions of the parts
injured and provoking changes anatomically comparable
to those one observes normally in the stage of activity
and of repose.

Parson and Phemister (52) report that Sir Ashley Cooper,
Copland, Morris, and Hordon maintained that shock was due
to general depression of the nervous system through
gross mechanical insult. After Claude Bernard discovered
the sympathetic control of the blood vessels in 1852,

Weir, Mitchell, Moorehead, and Keen, in 1865, decided that
shock was a reflex motor paralysis. In 1870 Fisher noted
that injury caused a reflex paralysis of vasomotor nerves
with a fall in blood pressure and dilatation of the large
veins of the visceral regions. Against this theory was
Loven who, in 1866, discovered that stimulation of the cent-
ral end of a cerebrospinal nerve caused elevation instead
of depression of blood pressure.

Hefmeister, in 1885 (20), called attention to the great
number of deaths from shock in abdominal operations.
Malnutrition of the heart, fatty degeneration, general
weakness, and loss of blood were mentioned as important
causes. Agnew maintained that the determining cause of
shock must reach that portion of the nervous system from
which the heart and lungs receive their motor endowments.

With the above review of the older ideas of shock product-
ion we shall now consider the various theories of shock
which have been worked on quite extensively in the past
two or three decades.

Theories of Shock Production
Theory of Capillary Congestion

The idea behind this theory is that the blood goes out
into the capillary bed thus reducing the total circulat-

ing blood volume and resulting in a lowered blood press-
ure. Erlanger, Gesell, Gasser, and Elliott (25) pro-
duced shock by traumatization of the intestines, by
injury of the adrenals, and by mechanical obstruction
of large vessels. They noted a marked capillary distent-
ion; the venules of the intestinal villi were also dis-
tended with solid masses of red cells. This was back
in 1917. Since that time much work has been done on
other theories which will be mentioned later--theories
which involve factors that should have been considered
in the above experiment. They failed to take into con-
sideration the possibilities of toxemia, exudation, and
nervous impulses as causative factors. Even though
there was capillary congestion we still do not know
what causes it. No idea was advanced for the actual
cause of capillary congestion.

Theory of Acapnia

Henderson (36) found that shock is associated with a
decreased venous return of blood to the heart. This
theory is an attempt to explain this decrease. Pain,
sorrow, etc. cause excessive respiration which results
in a reduction of the carbon dioxide content of the
blood and a state of acapnia. The conditions leading
to the shock state are hyperpnoea, acapnia, failure of

venopressor mechanism, venous anoxemia, tissue as-
phyxia, and acidosis and acute oligemia.

Very little supports this theory. There is the well-
known fact (Larson 39) that the severely wounded often
do not experience much pain, and the observation that
patients in shock do not always have excessive breathing.

Treatment with carbon-dioxide has not been as successful
as might be expected were the explanation correct.

Cannon and Bayliss (13) in 1919 were able to produce
shock experimentally by trauma and the respiration was
controlled artifically.

Henderson (36) observed the fall in blood pressure
following over ventilation of the lungs in man, so-
called acapnia shock. This state is approached in
experimental animals after reflex hyperpnea from stim-
ulation of sensory nerves under light anesthesia. It
is antagonized by deepening the anesthesia. The fall
in arterial pressure is due to arteriolar dilatation.

Theory of Inhibition

Meltzer (48) in 1908 noted that dissection of the skin

of the abdomen caused inhibition of peristalsis. He
assumed that injury caused an inhibition of all functions
of the body. But Cannon (15) in 1923 emphasized that
the cessation of intestinal movements was not due to
inhibition of activity in the central nervous system
but rather to activity there. Meltzer thought that the
action of depressor reflexes were responsible for the
lowering of arterial pressure. Such depressors (Larson 39)
play no part in secondary shock.

Theory of Fat Emboli

This idea (Larson 39) is based on the fact that shock
from traumatization of an extremity is often more severe
if bones are crushed, which presumably would favor the
production of fat emboli. But this favors any other
mechanism equally well, since broken bone fragments would
obviously produce considerable laceration of tissue.
The picture of fat embolism is usually that of pulmonary
embolism, with raised venous pressure, and is obviously
different from the picture of shock. Porter (55) in-
jected fat in small quantities in the vertebral artery
in dogs and obtained death from medullary embolism.
But the fall in blood pressure is here due to arter-
iolar dilatation which is not present in secondary shock.
Parsons and Phemister (52) conducted ten experiments

in regard to this theory. After trauma they found very
little fat in the various organs and came to the con-
clusion that fat emboli is not a factor in shock pro-
duction.

Theory of Adrenal Hypo and Hyper Function

Gley and Quinquand, two Frenchmen mentioned by Blalock
in 1933 (7) found that the blood pressure does not go
down until several hours after the removal of the adrenal
glands. This evidence would disprove the idea of hypo-
activity. Mann (46) found that the phenomenon of shock
was not due to removal of the adrenal glands. Blalock
and Johnson (10) found that hemorrhage or gross trauma
causes at first a decrease in cardiac output which is
later followed by a fall in blood pressure and that
after adrenalectomy these changes appear in the reverse
order.

The theory of overactivity (Blalock 11) is based upon
the observation that there is an increased adrenal
secretion as a result of severe pain or over exertion.
Shock can be produced by the injection of large amounts
of adrenalin. However, the amounts necessary is far
greater that that evoked by reflex stimulation.

With the above facts at hand it appears that neither
hypo nor hyper function of the adrenals can play a very
large role in shock production.

Theory of Decreased Alkali Reserve

This idea has been practically dropped by present day
workers. There is no direct evidence that supports this
theory. Moon (51) concludes that all evidence supports
the conclusion that the decrease in the reserve alkali
of the blood in shock is the result of decreased oxi-
dation due to circulatory deficiency. It is the effect
and not the cause of the circulatory disturbance, and
develops similarly whenever anoxemia is marked.

Exhaustion Theory

In 1914 Crile (21) was under the impression that the
"motor mechanism, which through its phylogenitic assoc-
iation with injury to the individual, is responsible for
the discharge of energy which is occasioned by the pres-
ence or thought of danger". When these discharges of
energy are intense enough shock is produced. Shock is
the result of the excessive conversion of potential into
kinetic energy in response to adequate stimuli. The
essential lesions of shock are in the cells of the brain,
suprarenals, and liver, and are caused by the conversion

of potential enery into kinetic energy at the expense
of certain chemical compounds stored in the cells of these
organs. There is strong evidence that animals capable
of being shocked are those whose self-preservation
originally depended upon some form of motor activity.
In each individual, Crile maintained, there is a limited
amount of potential energy stored in the brain, supra-
renals, liver and that the motor activity diminishes it.
If the expenditure is due to traumatic or to psychic
stimuli which lead to no obvious work performed, shock
is produced. From experiments on dogs Crile concluded
that ether anesthesia offers no protection to the brain
cells against the effect of trauma and that the lipoid-
solvent anesthesia probably breaks the arc which maintains
consciousness beyond the brain cells somewhere in the
efferent pathway. "The afferent pathwey from the seat
of injury unbroken the afferent stimuli reach and modify
the brain cells readily as if no anesthesia had been
given and it would seem that the brain cell changes must
be due to the discharge of energy in a futile effort
to escape from injury."

Ewing, in "The Nature of Shock", (27) showed that pro-
longed stimulation of the somatic nerves in mammals does

not bring on circulatory failure. Seelig and Lyon's (60)
experimental results indicate that the symptom complex
known as shock is not due primarily to vasomotor exhaust-
ion.

Erlanger, Gesell and Gasser (26) have shown that in the
early stages of shock, vasomotor tone is normal or in-
creased. As the shock progresses and the blood pressure
falls, the vessels lose some tone, but up to death will
show some reactions. Cannon concludes that there is no
true exhaustion of the vasomotor center.

The balance of power seems to be definitely against
the idea of exhaustion. Present day workers have dropped
the exhaustion idea.

Toxemia Theory

There is the popular belief that shock is due to a
toxic substance liberated into the blood stream follow-
ing trauma or some other exciting factor. The first
evidence of a chemical substance producing shock was
demonstrated in 1891 when Heidenhain (35) introduced
peptone intravenously into animals and produced exhaustion,
rapid pulse, and respiration, and low blood pressure.

Dale and Laidlaw (22) in 1910 introduced histamine intravenously and produced a similar but more shock-like effect through its vasodilatation action on the capillaries and arterioles.

During the World War, according to Cannon (17), both French and English reported that shock was associated with tissue injury and even here shock did not come on promptly but came only after delay.

Quenu (56) in 1918 showed that any means employed to delay or prevent absorption from the injured part delayed or prevented the development of shock.

McNee (42) showed that the application of a tourniquet to a badly smashed leg or amputation of the leg led to rapid and maintained improvement. Bayliss and Cannon (14), experimenting on dogs, showed that tissue damage led to a gradual drop in blood pressure, resulted in recovery of the patient when the injured region was isolated from the rest of the body, and resulted in failure of the blood pressure to fall as the vessels to the injured region were blocked so congestion developed.

Dale, Laidlaw, and Richards (23) have shown that hist-

amine occurs in tissue and when injected in minute amounts
it reduces the amount of circulating blood and lowers
the blood pressure. The effects in the above work of
Bayliss and Cannon are believed to be analagous to the
hypotensive effect in this experiment.

McIvar and Haggert (41) were able to induce shock in a
second cat by crossed circulation from a cat in which
tissues were damaged.

Sir Thomas Lewis (40), working on the skin, found that
mechanical, electrical, photo, and chemical stimulation
liberated a histamine-like substance which caused red-
ness, edema, and sometimes bled formation. He believed
that this vasodilator or substance was liberated by the
irritated vascularized portion of the epithelium and
that in extensive irritation of the skin, this vaso-
dilator substance was poured into the blood stream and
general circulatory changes resulted. When persons
with urticaria factitio are treated by extensive strok-
ing and combing the face becomes flushed, there is a
general rise in skin temperature, and a small temporary
fall in blood pressure. It is believed that deeper
tissues would respond the same. This experiment indicat-
ed, in the opinion of the author, that circulatory fail-

ure in extensive injury results from liberation of a histamine-like substance to the general circulation. On the other hand considerable work has been done which does not support the traumatic toxemia theory.

Thorpe (65) found that skeletal muscle contains an exceedingly small amount of histamine. Smith (64) and Parsons and Phemister (52) were unable to determine any toxic substance. They attributed the low blood pressure to the extravasation into smashed tissue. Blalock, Beard, and Johnson (9) found the same to be true.

In 1930 Simonart (61) confirmed the onset of shock following muscle damage but inferred that the fall in pressure was caused by afferent impulses from the injured areas. He cut the afferent nerves and the fall in blood pressure did not occur. This finding supports the nerve impulse idea which will be discussed later.

Parsons and Phemister (52) hammered the lower extremity of thirty-five animals and found a lowered blood pressure, increased pulse rate, accelerated and shallow respiration. If these findings were due to a toxic substance massage of the traumatized limb might have forced out more toxic material and produced a still further lower-

ing of the blood pressure. This was not found to be true.
The concentration of the blood was determined in these
animals in search of evidence of traumatic toxemia.
Blood counts were made from the ears. The red cells and
hemoglobin were found to be reduced. At no time was there
any evidence found of concentration of capillary blood.
The blood from the traumatized limb was directed to the
good limb and no vasodilatation and fall in general
blood pressure was noted. After the limb was hammered
and the blood pressure dropped, the limb was bled.
Death was produced when only a small amount of blood was
taken. When the low pressure was due to histamine in-
jection much more blood could be taken before death
resulted. These findings fail to demonstrate that either
hemolyzed or extravasated blood returning to the circul-
ation or a toxin is the explanation of the low blood
pressure of the traumatized animal and favor hemorrhage
as the cause.

To determine if a toxic substance could be found in
devitalized muscle which gave rise to shock the right
rectus muscle was implanted in other dogs and observed
for three weeks. No signs of intoxication were noted
at any time. Parsons and Phemister (52).

Parsons and Phemister (52) did some work on the chemical
control of circulation and found that vasodilatation
is not due to a histamine-like substance liberated from
broken down cells. They came to this conclusion in the
following manner. When histamine was injected there
was a marked and prolonged fall in blood pressure where-
as very large amounts of extensively hemolyzed or severe-
ly traumatized blood, when circulated through a peri-
pheral artery, did not lower the blood pressure and when
circulated through a vein an initial fall in blood press-
ure for only one or two minutes was noted.

When a constrictor is applied to a limb the vessels of
the limb constrict, on release of the constrictor there
is dilatation. Anrep (2) believed that the dilatation
may be due to the metabolites formed in the tissues
during the peroid of constriction. Parsons and Phemister
(52) perfused blood taken during the stage of dilatation
back into the recovered limb and were unable to demon-
strate any vasodilatation. These findings would tend
to rule out Anrep's idea of metabolites being the cause
of the dilatation. It seems logical to believe that the
vasodilatation following the constriction might cause
a drop in the blood pressure. After release of constrict-
ors which were applied to the thigh and arm for fifteen

minutes there was no change noted in the blood pressure.
Experiments were also done on animals in which either
the femoral artery or the aorta was tied off or obstructed
for varying lengths of time. No change in blood pressure
was noted on their release. Neither was there any vaso-
dilatation noted on release.

Dragstedt and Mead (24) approached the toxemia theory
from the pharmalogical side. They anesthetized dogs
with ether and sodium barbital, and recorded blood press-
ure, cannulated the thoracic duct for collection of
lymph. Then they traumatized the intestines, limbs, or
both and produced shock. Samples of blood and lymph
were collected and tested on isolated strips of guinea
pig or by intravenous injection into an etherized,
atropinized cat and noting the effect on blood pressure.
The blood was centrifuged before testing, the lymph
was tested directly. Their methods will detect histamine
in concentration around 1:250,000. Nine experiments
were run and in no instance was there any indication
of a physiological substance being present. Other
workers who took the blood from a traumatized extremity
during shock and injected it into a normal animal and
failed to produce a depressor effect are Smith in 1930,
Schneider in 1930, Holt and MacDonald in 1934, and

O'Shaughnessy and Slome in 1934-5.

Slome and O'Shaughnessy (62) in 1935 did a number of
experiments and came to the conclusion that the amount
of depressor substance which can be extracted from
tissue subjected to trauma is insufficient to produce
the phenomenon of shock. Complete occlusion of the
venous return from the limb failed to produce shock,
perfusion of the traumatized limb failed to produce
any evidence of the presence in the traumatized area
of a vasodilator substance. No depressor toxin could
be demonstrated in the systemic circulation of shocked
animals, and post mortem appearance in animals dead from
shock differs from those seen after death from histamine
poisoning. Therefore they concluded that the toxemic
theory of shock can not be substantiated.

Rome and Wilson (57) demonstrated that the depressor
effect of injection of extracts of traumatized tissue
recorded by some investigators as evidence of the toxic
theory are due to blood coagulation and to particles of
fat and tissue in these extracts.

Larson (39) commenting on the work of Cannon and Bayliss,
says that it is obvious that ligation of the entire

traumatized extremity will prevent local fluid loss,
and this favors the local fluid loss theory as strongly
as the toxemia theory.

The general survey seems to show that the toxic factor
certainly is not the proven cause of shock. All attempts
to isolate such a factor have been fruitless. With
our present experimental knowledge of the subject we
cannot say that shock is due to a toxic substance.
More conclusive work will have to be done before we can
get such a fixed idea.

Nervous Theory

Malcolm (45) noted that in shock the arteries were over
contracted and assumed that the lessened capacity forced
plasma out of the vessels into the tissues, but he did
not explain the primary constriction. Seelig and Lyon
(60) also found this vasoconstriction. Several years
later Erlanger (26) found a vaso-constriction in all
the types of experimental shock he studied. He suggest-
ed that the causative factor was a reduced circulation
through the action of painful stimuli, and of a certain
amount of hemorrhage into the vasoconstrictor mechanism.

Freeman, according to Cannon (17), believes that not

only pain but also cold, emotional excitement, asphyxia,
and hemorrhage have one physical action in common.
They evoke higher activity of the sympatho-adrenal system
and cause a pronounced vasoconstriction except in the
heart and skeletal muscle. Cannon (13) showed that
continued pain, prolonged exposure to cold, persistent
fear and other great emotional excitement, and restless-
ness act favorable to the induction of shock. These all
involve special action of the nervous system.

Cannon and Britton (16) used animals manifesting sham
rage. Under brief ether anesthesia the cerebral cortex
was swiftly destroyed. When the cat recovered from the
anesthesia it displayed a supreme degree of sham rage.
The sympatho-adrenal system is stimulated in this way.
The blood from fifteen of these experimental animals was
tested and showed a drop in blood volume of 22%.

Freeman, in some unpublished work quoted from Cannon
(27), lowered the blood volume as much as 27% by injecting
adrenalin for two hours at a physiological rate of out-
put from the glands in response to painful stimuli.
Ergotoxine blocks the vasoconstrictor effect of both
adrenalin and sympathetic nerve impulses. After ergo-
toxine neither adrenalin or sham rage caused a decrease

in blood volume. Clawing, lashing of the tail were
present to show that the cat was in the pseudoaffect-
ive state. The conclusion was therefore, justified that
sympatho-adrenal stimulation was responsible for the
phenomenon.

Another test of hyperactivity of the sympatho-adrenal
system was done by Cannon (17). He completely sympa-
thectomized the animals. The blood volume failed to
fall. At the end of five hours the blood pressure was
still at the original level. When the sympathetico-
adrenal was present the blood pressure fell to a shock
level in two to three hours, as the blood volume decreased.
Absence of a reduction in blood volume in completely
sympathectomized animals manifesting sham rage indicates
that toxic factors are not operating. It could not be
explained by local loss of fluids into traumatized
tissue because in the experiments with sham rage the
trauma was in the cranial cavity where swelling of
tissue is limited. Also the damage to the brain in
sympathectomized animals was the same as that in normal
animals, yet the blood volume was not reduced. Neither
a toxic factor, therefore, nor hemorrhagic factor was
present.

Hartman (34) and others say that the sympathetico-adrenal
system also dilates the blood vessels of skeletal muscles.
The capillary bed is thus increased by the opening of
primarily closed channels. The redistribution of blood
thus affected is associated with an outpouring of fluids
through the capillary walls to spaces between the muscle
fibers. Cannon (17). But it is also ordinarily assoc-
iated with muscle activity which would force the blood
into the lymphatics and back to the general circulation.
In the early stage of shock the arterioles are relaxed
which means greater filtration pressure in the recip-
ient capillary bed. Thus fluid and blood goes to the
peri-vascular intermuscular spaces. Injured men move
little so the fluid remains in the injured area. Thus
the blood volume might be reduced by greater filtration
into the muscle where there is vasodilatation than in
the skin and viscera where there is vasoconstriction.
In the stage of shock when the blood volume is reduced
the increased capacity of the dilated vascular area in
muscles which remain idle might play a significant part
in the diminished return of blood to the heart.

Parsons and Phemister (52) stimulated the sciatic and
the anterior crural nerve by forceps and faradic current.
At the beginning of stimulation there was a rise in blood

pressure due to the Loven reflex. The rise was sustained
as long as the stimulus was actively applied. In no case
did they find a fall in blood pressure.

In an attempt to determine the role of the nervous system
in animals where circulatory failure was produced by
traumatizing the limb experiments were performed in which
the limb was denervated before trauma and in others the
nerves were left intact. Where the nerves of the limb
were intact, hammering resulted in a fall in blood
pressure, the amount being directly dependent upon the
amount of injury. Where the nerves were cut there was no
drop in blood pressure.

Brain injury rarely brings on shock, while in cervical
and throcsic cord injury there is an immediate fall in
blood pressure due to paralysis of the sympathetic system
with a resultant vasomotor dilatation. In operations
of the abdomen the blood pressure fell as soon as the
operation was started and stayed down until the end of
the operation at which time it came up. This was noted
while working on the stomach, gallbladder, and lower
abdomen. This is believed to support the neurogenic
theory. Phemister and Livingston (54).

Intraabdominal manipulations, especially of the intest-
ines, are said to cause dilatation and accumulation of
blood in the mesentery. Phemister and Livingston .(54)
did not find intestinal congestion at autopsy, and more-
over, the reaction was also noted more often when work-
ing on the stomach and gall bladder when the bowels
were not disturbed than when the intestines were worked
on. They believed that the vagi, sympathetic, and the
intercostal-abdominal nerves to the peritoneum and ab-
dominal wall are the pathways of the impulses. They
believe, but have not proven, that vagi stimulation
carries impulses to the medullary center.

Fluid loss was measured by Slome and O'Shaughnessy (63)
in the same manner as Blalock did and found it not enough
to say that it is the sole factor determining the de-
cline in blood pressure. There, arterial supply of the
limb was occluded prior to trauma. Fluid could not be
lost in this case, since the limb was occluded of its
blood supply, it was both ischemic and anesthetic, so
nervous factors could not operate. No shock was pro-
duced during the period of occlusion. When blood was
supplied to the limb by cross circulation, shock was
produced.

Simonart (61) demonstrated that if the nerve was sect-
ioned before trauma there was no shock produced. Slome
and O'Shaughnessy (63) confirmed this finding and also
found that similar protection could be obtained by cont-
inuous spinal anesthesia. They concluded that local
toxin production plays no role in these experiments.
They also believed that traumatic shock is caused by
the operation of two factors: fluid loss in the traum-
atized area and nerve impulses from that area.

Slome and O'Shaughnessy (63) amputated the traumatized
limbs of shocked animals and in most cases got a marked
improvement in the condition of the animal. This im-
provement may be due to either the cessation of fluid
loss or to removal of the source of origin of the nerve
impulses. Records were taken of action currents on the
nerves of traumatized limbs and compared with records
from the opposite untraumatized limbs. No abnormal
impulses were recognized for three-fourths to one hour
after traumatization. After this interval of time almost
a continuous barrage of impulses rapidly developed.
These were shown to be centripetal afferent impulses.
The absence of impulses for a period accords with the
gradual decline in blood pressure which sets in at this
time. This seems to be quite confirmatory of the nervous

theory.

In 1930 Parsons and Phemister (52) decided that reflex
vasomotor paralysis or exhaustion did not account for
the circulatory failure since intensive stimulation of
somatic nerves produced elevation instead of a fall in
blood pressure and equal amounts of trauma to denervated
and innervated extremities produced the same amount of
fall in pressure in each.

This survey seems to indicate that nervous factors have
something to do in shock production. Certainly with the
strong experimental evidence in favor of this theory it
cannot be passed over lightly. The survey seems to show
that there is more experimental results in favor of this
theory than there is against it.

Plasma Exudation

Considerable experimental work has been done to support
the view that local fluid loss or plasma exudation may
be the cause of secondary shock. In 1930 Blalock (7)
concluded that the increased weight of a limb can account
for the loss of the circulating blood volume. At this
time he also transfused normal animals with blood from
a shocked animal and was unable to establish a toxic
factor. Blalock and Parsons and Phemister (52) traum-

atized one of the hind limbs of a number of dogs. They
noted that there was considerable fluid loss in the
groin so the legs were amputated higher than usual,
bisected and each lind leg weighed. They found that
there was sufficient loss of blood into the injured
part to account for the decline in blood pressure. The
degree of injury determined whether plasma or blood was
lost into the damaged tissue and it was also demonstrated
that even when it was mainly a plasma loss from which
the circulation suffered it could not be sufficient
to cause death. Later Blalock (8) manipulated the
intestines of twelve dogs and determined the fluid
loss as follows: the dogs were weighed before the ex-
periment, and after the experiment the fluid was sponged
out of the peritoneal cavity and the dog was again weighed;
the difference in weight between the intestines of the
traumatized dogs and those of a normal dog of the same
size were determined. This gave the amount of fluid
lost from the circulating blood. The average loss was
3.98% of the body weight. The blood pressure was also
decreased and there was hemoconcentration. Beard and
Blalock (4), by intestinal manipulation in dogs, pro-
duced extensive visceral weeping of the visceral peri-
toneal surfaces that is an important causative factor
in the resultant shock-like syndrome. They found a re-
duction in blood pressure to a shock level with

concentration of the blood and an outpouring of fluid
similar in chemical composition to blood plasma.

Wilson and Roome (67) constricted the hind limbs of
various dogs for two to twenty hours. Two-thirds of them
died at intervals of from five to fifty-two hours after
release of the constrictor. After release of the con-
strictor the legs swelled. The fluid was analyzed and
found similar to blood plasma; the extremity gained in
weight. The animal could be saved by amputation and
transfusion but not by transfusion alone or by amputation
alone. They concluded that the fall in blood pressure
and death was due chiefly to the withdrawal of water and
plasma from the general circulation to produce local
swelling of the limb.

Plasma-like fluid is lost in a variety of conditions
including burns, freezing, bile peritonitis, pneumonia,
acute peritonitis, intestinal manipulation, externally
strangulated colostomy loops, tissue autolyses in vivo,
mesenteric obstruction, and release of a constrictor.
The type of trauma can be thermal, chemical, bacterial,
mechanical manipulation, and capillary injury due to
inadequate circulation. (Harkins and Harmon 29,30,32.)
They report three cases of externally strangulated

colostomy loops, all of which showed an enormous amount
of serosanguinous material taken up by the dressing which
resembles blood plasma. These patients had a reduced
blood pressure. The loops were decompressed, fluid was
given and the blood pressure came up.

Harkins and Roome (31) believe that if a simple crushed
hand can display an increase in volume of several hundred
cubic centimeters it is easy to conceive that a more
extensive body injury could easily produce a change of
several times as much. They maintain that extensive
hemorrhage has long been recognized as shock producing;
concealed hemorrhage into body cavities and hollow
organs has been recognized but not diagnosed, whole
concealed hemorrhage into the tissue spaces has not been
universally considered as an important clinical cause of
secondary shock.

The extensive concealed hemorrhage and plasma exudation
in clinical cases substantiates the experimental observa-
tions of others that such local loss of fluid from the
circulating blood stream is a factor of importance in
production of secondary surgical shock or traumatic
shock.

In crossed circulation experiments conducted by Bell,

Clark, and Cuthbertson (5) a leg of a cat was trauma-
tized and then the blood from this leg was transfused
to a recipient animal. There was a marked fall in
blood pressure in the donor resulting in death. The
blood pressure of the recipient remained practically
unaffected. They also determined local fluid loss by
comparing both hind quarters in regard to weight differ-
ence following trauma to one limb, and concluded that
local fluid loss is a contributory if not the main cause
of fall in blood pressure. The induction of this state,
they stated, appears to be dependent on the integrity of
the nervous paths to the injured area. Moreover, the
crossed circulation experiment does not support the
traumatic toxemic theory.

Parsons and Phemister (52) traumatized one hind limb
of experimental animals and weighed both quarters and
found an average weight increase somewhat more than that
of the blood which, if withdrawn of the same weight,
will result in death. The limb was examined and found to
contain much more hemorrhage than edema. They say that
it is preferable to speak of hemorrhage rather than
shock or shock due to hemorrhage where acute loss of
blood in wounds, whether closed or open, is the cause
of marked circulatory embarrassment or failure.

In 1931 Harris and Blalock (33) showed that shock is not

associated with a general increase in capillary permeability and a loss of fluid throughout the body.

A summary of the above experimental work points to the fact that local fluid loss certainly does play a major role in shock production. Some work does not, however, support this idea. With our present knowledge of shock it seems that local fluid loss and nervous impulses are two factors which do play major roles in shock production. At least these two ideas have considerable experimental work to back them up. However, we must bear in mind that a toxic factor may alter the permeability of the capillaries so that the blood plasma may get out of the circulatory system and produce the local loss.

Possible Mechanism

With the previous review of what has been done both experimentally and clinically to reach a possible answer as to what the actual cause of shock is, one can see that the answer is rather vague. Without an actual knowledge of the cause of shock we certainly cannot explain the mechanism behind its production. In review of the various theories we found that each theory had its own mechanism of shock production. In this paragraph we want to

discuss the mechanism behind shock production after the blood pressure has fallen.

Meek (47) states that Gesell has showed that shock is often produced when the blood volume is reduced from 7-17%. The flow of blood through an organ may be reduced as much as 60% by reduction of the blood volume by 10%. The decreased blood volume results in a lowered alkali reserve. This is not the cause of shock but a secondary feature. This is followed by acidosis. The decreased carbon dioxide of the blood interferes with the unloading of oxygen from the hemoglobin and reduces the tonus of both the respiratory and cardio-vascular centers. This means a vicious circle for the initial loss of effective blood volume is now augmented by stasis in the smaller blood vessels due to loss of central tonus, local peripheral dilatation from the accumulation of acids, and capillary transudation because of damage from the lack of oxygen. The acid salts resulting from the decreased oxygen aid in reducing the volume of the blood stream by the withdrawal of fluids to the acid-laden cells. The problem of lost blood thus involves a great deal more than that lost by mere hemorrhage or extra-vasation. The latter may even be almost entirely masked by the fixation of fluids in the tissue cells.

Moon (51) says that two major factors are operative in
the development of shock:

1. Atony and dilatation of the capillaries
and venules in extensive visceral areas is the first
factor. This leads to reduced blood volume, and retards
the circulation. The oxygen to the tissues is reduced
in proportion to the decreased volume flow of blood.

2. The second factor is anoxia. The accum-
alation of wastes due to deficient oxygenation will,
of itself, result in atony and dilatation of minute
vessels.
Either of the two above factors operating will bring the
other into action. Thus a vicious circle is set up as
is shown by the following diagram of Moon's. The
essential mechanism in which the circulation becomes
deficient is a difference in the volume of blood and the
volume-capacity of the vascular system. The mechanism
of origin must then be sought in:

1. Those factors which reduce the volume of
blood-hemorrhage, transudation, loss by perspiration,
vomiting, and diarrhea.

2. Those factors which increase the volume-
capacity of the circulatory system-dilatation of cap-
illaries. The same factor that does this may increase

the capillary permeability.

These two factors usually work together.

Neurogenic factors
　　Neuro. lesions
　　Emot. reactions
　　Heat stroke?

Permeability
Hemoconcentration
Petechiae
Stasis
Edema
Effusion

Agents or conditions
Injurious to capill.

Cap. Atony → Pathology

Clinical
Trauma, burns,
extensive surgery
Intoxications
　　Drugs
　　Metabolic
　　Bacterial
Serum disease
Abdominal
　　Obstruction
　　Perforation
　　Peritonitis
　　Pancreatitis

Prod. of def. ox.

Reduced Blood vol.

Hemorrhage
　　Loss of fluid

Reduced Vol. flow

Cardiac Ineffic.
　　Myocardial
　　　　coronary occ.
　　Endocardial
　　　　valvular
　　Functional
　　　　heart block

Experimental
Muscle pulp
Tissue extracts
Burns, trauma
Obst. to bowel
Peptone, venoms
Bact. products
Bile & its salts
Drugs
Histamine
Anaphylaxis
Narcotics

Tissue anoxemia

Reduced delivery of O₂

Sympatho-adrenal
　　Hyperactivity
　　Adrenalin

Asphyxia
　　All causes

Cir. Obstruction
　　Art., venous

The vicious circle and associated factors

37.

Austin (3) classifies acute circulatory failure on what
he calls a physiological basis. The clasification is
as follows:

1. Hematogenous type in which there is de-
creased blood volume, vasoconstriction, decreased card-
iac output, decreased in blood pressure.

2. Neurogenic type in which there is an immed-
iate vasodilatation depending on a diminished constrictor
tone due to action through the nervous system.

3. Vasogenic type in which vasodilatation is
induced by agents acting directly on the vessels.

4. Cardiogenic type in which the primary dist-
urbance is in the heart; here a venous engorgment results.
It is also possible to have a combination of these.
This classification is of very little help in getting
at the mechanism. True enough it tells us what produces
the drop in the blood pressure. The fault is that it
does not tell us what factor or factors start the various
systems to work or what stops them from working.

Moon and Morgan (49) have done much work on shock cases,
not all of which were cases of traumatic shock. Never-
theless, the findings are similar. Regarding the mech-
anism of death in intestinal obstruction they came to
the following conclusions:

1. The physiological disturbances following intestinal obstruction are those usually seen in the shock syndrome.

2. Gross and microscopic visceral changes are identical with those which accompany shock produced by various means. They are distention and engorgment of capillaries and venules, edema and ecchymosis of the lungs and gastrointestinal mucosa and effusion into the serous cavities. Evidence of injury to the capillaries is seen in the capillary hemorrhages, the formation of edema fluid of high protein content and the rapidity with which colloidal dyes escape from the blood into the tissues.

3. The probable cause of the shock syndrome following intestinal obstruction is intoxication by histamine absorbed from the obstructed bowel. Any of the agents alone will produce the shock-like syndrome in animals.

4. Intestinal obstruction is one of the many conditions which will produce characteristically the shock syndrome.

Pathology

As regards the pathology of shock little has been done.
Moon and his associates are practically the only workers
who have done anything in this line. Necropsy of shock
cases showed the following findings. The superficial
veins were collapsed and bloodless, the blood in the
heart, large vessels and parenchyma of the organ was
dark and cyanotic, there was ecchymosis and the blood
and blood-tinged fluid in the cavities in severe cases.
The bowels were atonic, relaxed and distended, mesen-
teric vessels were engorged. The mucosa was congested,
edematous and frequently contained ecchymosis. The lungs
were congested. Microscopically the capillaries were
distended, the venules were engorged, hemorrhage from
capillaries was numerous. The liver and kidneys were
congested, the cells showed parenchymatous degeneration.
The spleen usually had less blood than normal. The heart
was a purplish cyanotic color, the venules and capill-
aries of the myocardium was engorged. Moon (49,51).

Referring to the above Moon says "these circulatory
phenomenona are the same in character as seen in early
acute inflammation, and their mechanism or origin is
similar. The results from systemic effects of various
agents which cause dilatation and increase permeability

of the walls of the capillaries and venules. Such
vascular changes result in the sequestration of blood
in the minute vessels and the transudation of plasma,
thereby producing edema, reducing the blood volume and
increasing its concentration. This combination of
results reduces both the effective and the total blood
volume".

The Medical Research Council showed that circulatory
failure is not due to cardiac or vasomotor inefficiency.
The vasomotor mechanism is active and the peripheral
arteries are not atonic but are maximally contracted.
They thought that wound shock resulted from the absorp-
tion of injurious substances from the traumatized tissue.
Moon's observation of necropsies supports this.

<p align="center">Clinical Picture</p>

<p align="center">Symptomatology</p>

Without a thorough knowledge of what the shock syndrome
presents clinically it would be practically impossible
to say whether a patient is in shock or not.

Makel (44) describes the clinical pictures as follows:
increased pulse rate, low blood pressure, exposed parts
pale, and a tinge of cyanosis is often noted, sweating,

complaints of thirst, restlessness, and increased respira-
tory rate. Following injury a patient with a systolic
blood pressure of one hundred should be considered as
being in potential shock. When the systolic blood press-
ure goes below ninety mm. of mercury the patient is in
actual shock and treatment should be instituted. A
patient in shock may have a slow pulse (70-80), there-
fore the pulse rate should not mislead the examiner.
The temperature as a rule is sub-normal. In severe
shock there is often an early leucocytosis. It should
be understood that the above symptoms are not present
until several hours after injury or whatever the caus-
ative factor may be in the individual case. Besides
the symptoms mentioned above Mahaffey (43) includes a
rapid, thready pulse as a rule, respirations, besides
being increased in rate are shallow, superficial, feeble,
and irregular or gasping, and the exposed parts, besides
being cold, are moist. The patient may be unconscious.
If conscious he is dull, listless, and expressionless,
and often displays muscular relaxation. The pupils are
dilated and react slowly. There may be involuntary
micturation.

Laboratory Findings

Hemoconcentration has already been mentioned as a constant finding in shock. It is an early finding. Other laboratory findings include (Moon 50) a diminished renal excretion and a lowered basal metabolic rate. Later, the coagulability and oxygen of the blood are decreased, blood chlorides are below normal and the blood non-protein nitrogen and blood sugar are increased. It has been suggested that the diminished renal excretion is due to the lowered renal function incident to anoxemia or to hemoconcentration. Likewise, some workers feel that the high blood non-protein nitrogen may be due to decomposition of protein in the tissues. Early in shock the white cell count is low, later a moderate leucocytosis is present. As regards sodium-potassium level in shock, Bisgard (6) produced traumatic shock in dogs and came to the conclusion that there was no consistent alteration of serum sodium, or potassium. The same was found to be true of blood chlorides.

When Is Patient In Shock?

Naturally there is no definite line between being in shock and not being in shock. Just as in any other abnormal condition of the body the symptoms vary with the individual. For that reason we cannot say that

below one particular blood pressure reading the patient is in a state of shock. However, Clement (18) divides surgical shock due to anesthesia into three degrees. He bases this classification on the degree of fall in blood pressure and increase in pulse rate. In the first degree are those cases with a fifteen per cent decrease in pulse rate without an increase in blood pressure or a ten per cent decrease in blood pressure without a decrease in pulse rate. When in second degree shock they have a twenty five per cent increase in pulse rate plus a ten to twenty five per cent increase in blood pressure. Those in third degree shock have a pulse rate of one hundred and ascending plus progressively falling blood pressures reaching eighty mm. mercury systolic or less and a pulse pressure of twenty mm. mercury or less and progressively diminishing. This is McKesson's shock index. The first and second degrees are not fatal. The third may be fatal.

Makel (44), as stated beforehand, declares a patient in potential shock when the systolic blood pressure gets down to one hundred mm. mercury. When the systolic blood pressure gets below ninety mm. mercury, the patient is in actual shock.

Differential Diagnosis

Shock should not be confused with hemorrhage. In both the pulse and respirations are slow, the temperature subnormal, and the blood pressure diminished, but following hemorrhage the blood concentration is unchanged at first but soon is lowered as fluids are absorbed from the tissue into the blood. In hemorrhage there is no retention of nitrogen products, no hematuria, the coaguability of the blood is not affected characteristically, and there is no tendency to diarrhea and vomiting. When fluids are supplied therapeutically they are retained. In shock the hemoconcentration is present before the circulatory inefficiency manifests itself. Fluid is not absorbed from the tissues and if injected passes out rapidly. The vomitus and excreta contain hemoglobin and albumin. At necropsy the viscera is congested and edematous and contains hemorrhage from the capillaries in shock, whereas in hemorrhage the tissues are pale, dry and anemic. In addition Mahaffey (43) mentions the fact that in hemorrhage there is impaired vision, restlessness, anxiety, thirst, nausea, and increased pulse rate, all of which appear slowly. In shock the symptoms appear rapidly.

Treatment

Prevention And Treatment

With the above knowledge we can direct treatment toward
the possible causes. The actual cause is still in doubt,
nevertheless we have numerous theories regarding cause.
We can attempt to eliminate what we think are causative
factors. It is far easier to prevent shock in most
cases than it is to overcome it once it is present.

Shock frequently develops following accidents which do
some bodily damage such as producing fractures or con-
siderable soft tissue injury. Since fractures produce
trauma all fractures should be splinted at the site of
fracture. Exposure should be guarded against. The pat-
ient should be kept warm. Where there is excess pain
morphine should be given hypodermically. Head injury
should be put to bed, made comfortable, kept warm, and
not disturbed by immediate X-ray. The same general
plan should be followed in all accident cases--rest,
eliminate pain, keep warm.(Perkins 53.)

In the operating room there are four contributing factors
to the onset of shock: prolonged deep anesthesia, hemorr-
hage, manipulation of abdominal contents, and prolonged
operating time. Preventative measures should therefore
be directed toward eliminating the contributing factors

as much as possible. The lightest plane of anesthesia possible to work in should be maintained. Operating time should be cut down but efficiency should not be sacrificed for speed. The patient should be examined before operation and evaluated as to what his chances of surgical success are. During the operation the pulse pressure and pulse rate should be taken routinely. (Clement 18.) If the patient has been dehydrated preoperatively because of vomiting, high fever, sweating, frequent enemata, the fluids should be replaced before the operation is started. Fluids should be given continuously during the operation. Before the operation is begun a needle should be put in the anticubital fossa or a canula inserted through a small longitudinal incision in the skin at the beginning of the great saphenous vein just above the medial malleolus of the ankle. Forty to sixty drops of five per cent glucose in normal saline per minute is recommended. When there has been much vomiting and chloride loss one and five tenths per cent saline is substituted for the nine tenths per cent. The dextrose is nutritive, anti-ketogenic, and stimulating. (Trimble 66.)

Coller (19) says that about one thousand cc of fluid is lost during an operation and the first four hours

after. Furthermore, the basic fluid requirement daily
is about three thousand cc. Thus, to keep a balance,
at least three thousand cc. are needed daily. The fear
of embarrassing the heart by giving saline or glucose
is unfounded. Infusions given at the rate of one
thousand cc. per thirty minutes means only six minims
passing through the ventricle at each systole. Therefore,
it is difficult to conceive that the heart is being
over burdened.

When the systolic blood pressure falls below ninety mm.
mercury blood loss should be strictly guarded against
since a very small loss may be serious here. In low or
declining blood pressure gas-oxygen anesthesia should
be given surgical cases rather than ether or chloroform.
Deep ether anesthesia decreases the ability to withstand
hemorrhage. In case of much blood loss spinal anesthesia
should not be used. The vasodilatation will produce
a further decline in pressure. (Blalock 11.)

Sir Stanton Hicks (37) lists the treatment and the prevent-
ion of shock under three headings:

      1. Maintenance of the circulatory blood volume.

      2. Avoidance of extra demands up on the cir-
culation.

3. Mitigation or avoidance of any influences
likely to stimulate the sympathetico-adrenal system or
to undermine the metabolic resistance of the tissue cells.

Under the first heading we are concerned with dehydration
and osmotic pressure of the plasm proteins. Most workers
agree that five per cent dextrose in normal saline works
well here. The only drawback to it is that it will not
stay in the circulatory bed very long. Many agree that
six per cent gum acacia in nine-tenths per cent saline
is retained well, however, some feel that the acacia is
kept in the system too long. Sarma (59) found that acacia
leaves the blood stream in about six days, is detected
in the urine immediately after injection, and may be found
in the blood and urine as long as ten to fourteen days
after administration. Basing his reasoning on these
findings and on the fact that it has the same viscosity
as whole blood and the same osmotic pressure as the plasma,
Sarma feels that acacia solution is a desirable substance
to use in the treatment of shock. All workers do not
agree. Anderich and Gibson (1) showed large proportions
of acacia deposited in the hepatic cells of the liver.
Hall (28), experimenting on dogs, showed an impairment
of liver function in carbohydrate and plasma protein
metabolism.

Under the second heading are placed those measures taken
to insure warmth and to prevent any movements either due
to pain or apprehension of the subject yet unaware of the
danger of his condition. Quietness is essential. It is
necessary to bear in mind the effect of overheating because
the increased flow of blood to the skin, brought about by
the heat regulatory mechanism,may, in severe cases of de-
pleted circulatory volume, further diminish the cardial
filling and lower the blood pressure. It is only necess-
ary to prevent heat loss. Heat need not be applied.
Under the third heading are included such influences as
are known to predispose to shock. These are exposure.
to cold, starvation, and prolonged anesthesia.

If the early stage of shock is recognized and the diag-
nosis is certain, (Coller 19), morphine can be given as
a preventative. It stops the movements due to pain which
are likely to increase the loss of blood or plasma into
the damaged area and to increase the demands upon the
heart due to muscular response and emotional interference
with the cardiac rate while it lessons the activity of
the sympathetic system and consequent vasoconstriction.
It depresses the respiratory center and therefore is dang-
erous. Later shock is developed and the respiratory
center is depressed by anoxemia. Mahaffey (43) believes

in giving morphine in small doses to relieve pain, rest-
lessness, nervousness, and to stimulate the heart.
Besides giving glucose intravenously he gives normal sal-
ine or five per cent glucose in saline per rectum. Hot
tea and coffee are given if the patient can drink.
Small transfusions of three hundred to four hundred cc of
blood is the method of choice of replacing fluids to raise
blood pressure. Aside from morphine Mahaffey finds other
drugs useful. Atropine is given for the moist skin.
Neo-synephrine hydrochloride given subcutaneously or
intramuscularly has a more lasting effect in raising the
blood pressure than has pituitrin, ephedrine or epine-
phrine.

A patient in shock should be handled very little. Shift-
ing the circulating blood suddenly can so interfere with
the venous return as to cause circulatory collapse. Hicks (37).

Bond and Wright (12) recommend using intravenous lyophile
serum in the treatment of hemorrhage and traumatic shock.
Blood is rapidly frozen and is rapidly dehydrated from the
frozen state under a high vacuum. It is readily dissolved
in water to make an isotonic or hypertonic solution.
Because of its rapid solubility it is called "lyophile".

The immediate availability, its theoretical suitability, and its immediate action upon shocked animals suggests its use as a valuable treatment for clinical shock.

The anesthetic may be the causative factor. When the blood pressure and pulse indicate the approach of shock the anesthesia should be lightened. The Depression test should be applied. That is, the oxygen content of the mixture should be increased. A reversal of the systolic pressure upward indicates a positive test and means that the depression has been due, in part at least, to too deep anesthesia. No change in blood pressure would indicate that the depression is due to factors other than the anesthesia and would call for other measures in treatment. Clement (18).

In conclusion, we can attempt to prevent shock by:

1. Proper treatment of emergencies--avoidance of excessive trauma, pain, exposure.

2. Proper preoperative and postoperative care and avoidance of prolonged deep anesthesia, hemorrhage, excessive manipulation of abdominal contents, and prolonged operating time.

3. Recognizing circulatory deficiency before it is fully developed by hemoconcentration.

Once shock has developed the treatment is very similar to that mentioned above:

1. Morphine in small doses to relieve pain, restlessness, nervousness.

2. Keep the patient warm.

3. Supply intravenous to raise blood pressure.

4. Quiet is essential.

5. Drugs, other than morphine, are of little importance. Vasoconstricting drugs produce only a temporary rise in blood pressure.

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