STUDIES ON TRAUMATIC SHOCK: I—BLOOD VOLUME CHANGES IN TRAUMATIC SHOCK*

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DURING WORLD WAR I there was ample opportunity for a serious clinical study of traumatic shock. Many observations were made by competent groups of researchers in both the English and American medical corps. The experiences of these groups were reported by the joint English and American Research Councils, but the material was made more generally available by the publication of Cannon's[1] important monograph "Traumatic Shock" in 1923.

Since that time, although the remarkably thorough and painstaking investigations of Blalock directed attention to the importance of local blood loss as a causative factor in traumatic shock, no studies have been made on the blood volume of patients in shock as a result of trauma.

With the advent of the present global war, attention was again drawn to the urgent need for more data on clinical shock. By this time accurate clinical observations could be correlated with the blood volume of patients in shock because of the development of an adequate method by Gregersen, in 1935, for the estimation of plasma volume.

The present communication is a report of the estimation of plasma volume in a considerable number of patients in shock as a result of various types of trauma, and an attempt to correlate these blood volume studies with the manifestation of shock signs in these patients. We have tried to determine the relative importance of blood loss as an initiating and sustaining factor in traumatic shock.

We are fortunate in having at our disposal a considerable amount of clinical shock material very similar to the shock-patient group seen in modern warfare. One of our hospitals cares for a large urban Negro population, mostly of lower economic levels. The injuries sustained by these persons are caused, in general, by knife and gunshot wounds of the extremities, chest and abdomen. To this group of shock cases has been added a considerable number of traumatic injuries of the skeletal structures caused by automobile and industrial accidents.

THE ESTIMATION OF PLASMA VOLUME IN THE SHOCK STATE

There has been considerable hesitancy on the part of research workers

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CHART 1

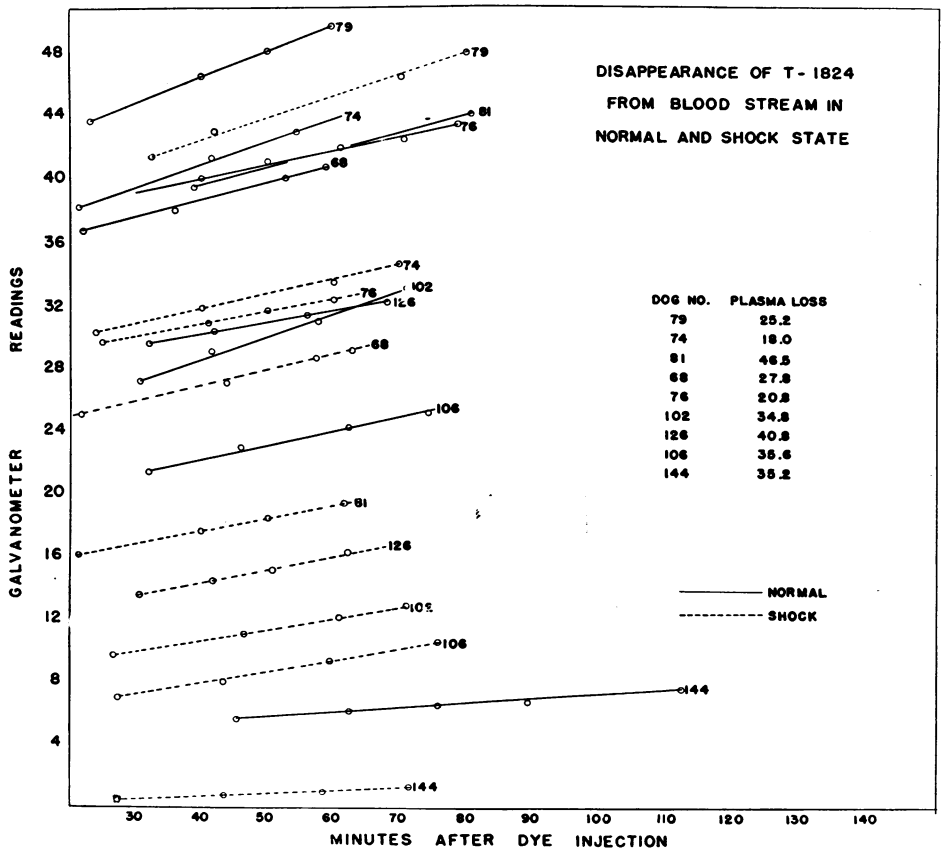

CHART 1.—Disappearance rate of dye T-1824 from the blood stream of dogs, during normal state and in shock from intestinal strangulation. The disappearance rates are practically identical, suggesting that there was no generalized increase in capillary permeability in the shock state.

to attempt the determination of plasma volume in the shock state, because of the current belief that there is in shock an increased capillary permeability. This generalized increase of capillary permeability, some maintain, will cause the injected dye to be lost from the vascular system into the tissue spaces rapidly, and in amounts great enough to seriously disturb the disappearance curve of the dye.

Insofar as we can find, the first determinations of plasma volume during the shock state were made by Keith,[2] and Robertson and Bock,[3] working for the Shock Committee during World War I. These investigators employed the vital red dye method of Keith, Rowntree and Geraghty,[4] and were able to demonstrate a rather serious depletion of plasma volume in wounded soldiers suffering from traumatic shock.

Following World War I, interest in the determination of plasma volume waned, probably due to the fact that considerable doubt was cast on the validity of the vital red method for the determination of plasma volume,

TABLE I

ACUTE BLOOD LOSS

Shock—None or Mild

Patient	Blood Pressure	Pulse Rate	Pulse Quality	Venous Filling	Extremities		Plasma Volume	Hemato-crit	Plasma Protein
					Temp.	Sweat.			
B. E.	110/80	120	Good	Good	Warm	Dry	39	42	6.8
B. N.	118/80	112	Poor	Poor	Cold	Dry	41	34	6.4
M. C.	100/60	92	Good	Good	Warm	Dry	38	31	7.7
J. R.	90/60	76	Poor	Good	Warm	Dry	45	43	6.8
A. C.	110/80	76	Good	Good	Warm	Dry	41	41	6.8
J. F.	80/60	68	Good	Poor	Warm	Moderate	38	39	5.7
R. A.	100/60	82	Fair	Good	Warm	Dry	42	39	6.6
J. H.	120/80	80	Good	Good	Warm	Dry	41	40	6.5
C. C.	92/66	80	Fair	?	Cold	Moderate	35	42	6.6
J. R.	68/48 to 130/90	64	Good	Good	Warm	Dry	42	32	5.4
A. S.	92/70	96	Good	Good	Warm	Dry	35	44	6.9
				Moderate or Severe					
L. H.	Unobt.	160	Poor	Poor	Cool	Moderate	20	32	5.4
B. P.	90/60	150	Poor	Poor	Cool	Dry	28	27	6.6
V. B.	45/0	130	Poor	Very poor	Cold	Marked	24	34	6.9
D. G.	75/50	126	Fair	Good	Cold	Moderate	28	24	7.1
M. K.	65/50	120	Poor	Very poor	Cool	Marked	26	31	6.6
L. D.	68/58	130	Poor	Poor	Warm	None	26	28	6.9
O. S.	80/60	72	Fair	Poor	Warm	Dry	25	27	4.9
A. R.	60/40	80	Poor	?	Warm	Marked	33	41	6.3
W. R.	80/50	108	Poor	Poor	Cool	Dry	31	44	6.8
C. J.	65/20	90	Poor	Poor	Cold	Marked	26	35	6.0
T. D.	110/80	130	Fair	Poor	Cold	Dry	28	45	5.5
A. G.	80/40	92	Fair	?	Warm	Dry	31	35	6.7
C. G.	62/20	90	Fair	Fair	Warm	Marked	38	35	6.0

even in the normal state. It was not until Gregersen[5] developed and standardized the dye method which employs the dye T-1824, called by some "Evans' blue dye," that much recent work has been done on the determination of plasma volume. Gregerson, Gibson and Stead,[6] employing the spectrophotocolorimeter, were able to show conclusively that estimations of plasma volume could be made by this method if it were used in the manner outlined by them. Later, Gibson and Evelyn[7] adapted this method to use the Evelyn photoelectric colorimeter; this, in the minds of some, has simplified considerably the estimation of plasma volume.

There have been few published studies on the use of the Gregersen-Gibson method for the determination of plasma volume during the shock state. The observations of Freeman, and his coworkers,[8] who used this method in experimental shock produced by continuous adrenalin infusion, have cast some doubt on the validity of the T-1824 method for the determination of plasma volume during shock. These workers found the dye in the pericardial lymph and other tissue fluids; one could infer from their observations that there might be rather disturbing losses of the dye from the vascular system during shock, losses possibly so great as to disturb seriously the disappearance curve of the dye.

The majority of our observations with the Gregersen-Gibson method for the estimation of plasma volume during the shock state have been made in patients in clinical traumatic shock and in the experimental shock prepara-

tion, described earlier by one of us,[9] namely, the production of shock by the strangulation of a short loop of ileum.

In Chart 1 is shown the dye disappearance curves in ten dogs in the normal and in the shock state. The solid line represents the disappearance curve of the animal in the normal state while the broken line represents the dye disappearance curve for the same animal after it had lost enough plasma to put it into more or less severe shock. At the right of each disappearance curve is given the number of the animal. It will be noted that the dye disappearance curves in both the shock and normal state practically parallel each other for the same animal.

In Chart 2 are given a small number of dye disappearance curves for human patients in severe shock compared with several disappearance curves found in patients with normal blood volumes. It will be seen that the dye disappearance rate is practically the same whether the patient is in severe shock or not. In several instances in patients in severe traumatic shock we have found increased dye disappearance rates. However, when the plasma volume determination was carried out on the following day we would often get a high dye disappearance rate even though the patient then showed no evidence of clinical shock.

CHART 2

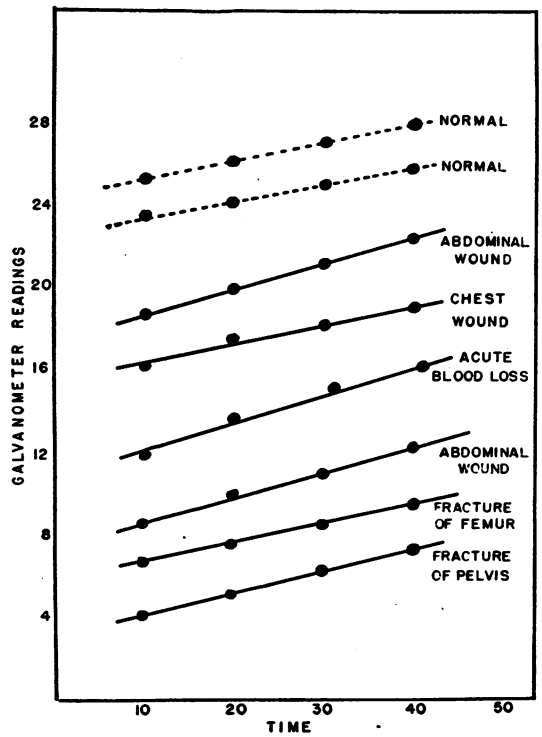

CHART 2.—Disappearance rate of dye T-1824 from the blood stream of human subjects during the state of traumatic shock.

Our experience with the use of the Gregersen-Gibson method for the estimation of plasma volume in well over 500 plasma volume determinations in the shock and normal states in patients has convinced us that the method gives valid data for the estimation of plasma volume during the shock state. The dye disappearance rates in clinical shock offer no evidence that would lead us to believe that there is generalized increased loss of the dye through the capillary wall in clinical traumatic shock.

Analysis of many dye disappearance rates in normal and shock patients has convinced us that for clinical purposes one can estimate plasma volume rapidly by using only one plasma sample, taken ten minutes after the injection of the dye. This, likewise, has been the experience of Gregersen, and his coworkers,[10] and Shaefer.[11] Throughout this research, however,

TABLE II

SKELETAL TRAUMA

Shock—None or Mild

Patient	Diagnosis	B.P.	Pulse Rate	Pulse Quality	Extremities: Venous Filling	Extremities: Temp.	Extremities: Sweat.	Plasma Volume	Hemato-crit	Plasma Protein
P. J.	Compd. fract. tibia, fibula.	140/80	72	Good	Good	Warm	Dry	39	42	7.0
D. T.	Fract. femur.....	120/80	80	Good	Good	Warm	Dry	45	42	7.0
A. O.	Fract. pelvis.....	110/70	90	Good	Good	Warm	Dry	38	40	6.7
M. N.	Compd. fract. tibia, fibula.	124/82	90	Good	Good	Warm	Dry	39	37	6.5
O. R.	Compd. fract. femur, tibia..	110/68	60	Good	Good	Warm	Dry	42	47	7.0
H. C.	Fract. femur.....	128/80	80	Good	Good	Warm	Dry	46	46	7.7
E. T.	Gunshot wound, compd. fract. of shoulder.....	120/80	76	Good	Good	Warm	Dry	40	43	7.2
F. B.	Fract. tibia, fibula.....	90/50	80	Fair	Fair	Warm	Dry	40	34	7.0
I. D.	Fract. pelvis.....	120/70	90	Good	Good	Warm	Dry	45	39	7.2
T. D.	Fract. femur.....	130/80	84	Fair	Good	Warm	Dry	43	43	7.5
F. F.	Dislocated hip.....	130/90	90	Good	Good	Warm	Dry	31	47	6.9
M. M.	Fract. femur, knee lacera-tion.....	110/65	104	Fair	Fair	Sl. cool	Dry	32		6.7
S. T.	Compd. fract. tibia, fibula..	142/110 to 90/50	112	Fair	Good	Warm	Dry	29	34	7.2
J. C.	Fract. femur tibia, fibula...	90/60 to 110/60	72	Good	Good	Warm	Dry	35	37	7.0
G. S.	Compd. fract. of femur.....	90/60	70	Good	Good	Warm	Dry	38	46	6.3
R. M.	Fract. of femur.....	96/64	100	Fair	Fair	Cool	Dry	25	33	6.1
S. H.	Compd. fract. of femur....	80/60	100	Fair	Fair	Cold	Moderate	32	49	7.1
A. J.	Fract. femur.....	184/90	80	Good	Good	Warm	Dry	48		6.9
C. W.	Fract. femur.....	110/80	88	Good	Good	Warm	Dry	40	38	7.0
L. H.	Traumatic amp. of arm...	130/90	82	Good	Good	Warm	Dry	41	44	6.2
E. M.	Fract. femur, basal skull fract.....	120/80	78	Good	Fair	Warm	Moderate	43	40	6.5
L. A.	Gunshot wd., fract. femur..	90/70	76	Fair	Good	Warm	Dry	42	40	7.0
J. C.	Fract. femur, radius & ulna	70/58	65	Good	Fair	Warm	Dry	45	33	5.3
W. F.	Compd. fract. tibia, fibula..	120/80	..	Good	Good	Warm	Dry	42	44	6.7
E. W.	Fract. pelvis.....	110/70	..	Good	Fair	Warm	Dry	38	40	6.9
P. H.	Fract. pelvis.....	130/80	..	Good	Good	Warm	Dry	42	39	7.1
J. A.	Fract. femur.....	130/90 to 80/60	80	Good	Good	Warm	Dry	32	43	7.7
J. B.	Fract. femur.....	80/60 to 110/60	96	Good	Good	Warm	Dry	36	46	7.5
	Moderate Shock									
B. L.	Fract. femur.....	90/60 to 70/50	56	Poor	Good	Warm	Dry	28	36	6.3
L. F.	Fract. femur, scapula	90/60	80	Good	Good	Cold	Marked	25	48	6.8
E. S.	Compd. fract. tibia, fibula...	136/68	118	Good	Good	Warm	Marked	31	39	5.9
R. H.	Fract. femur.....	70/45	100	Fair	Fair	Cool	Moderate	25	42	6.1
T. L.	Compd. fract. tibia, fibula...	92/50	76	Good	Good	Cool	Dry	25	48	6.6
J. H.	Compd. fract. of femur....	60/40	60	Poor	Fair	Cold	Dry	31	49	6.5
G. R.	Fract. tibia, fibula.....	80/40	80	Poor	Fair	Cold	Dry	31	39	6.9
N. F.	Fract. pelvis, & scapula...	104/60	120	Poor	Poor	Cold	Marked	31	31	6.0
T. W.	Fracture of pelvis.....	70/50	136	Poor	?	Cold	Marked	36	38	6.1
L.M.S.	Traumatic amputation ft., compd. fract. tibia & fibula.	75/40	68	Fair	?	Cool	Marked	32	43	6.6
	Severe Shock									
H. L.	Traumatic amp. of leg.....	130/50 to 70/50	100	Fair	Good	Cold	Marked	29	38	5.9
P. R.	Fract. pelvis.....	80/60	110	Fair	?	Cold	Marked	25	47	7.5
D. H.	Fract. femur & fibula.....	70/50	110	Poor	Poor	Cool	Marked	30	39	5.6
J. W.	Compd. fract. of tibia, fibu-la, fract. pelvis.....	125/60 to 75/50	110	Good	Good	Warm	Dry	25	31	7.3
H. G.	Compd. fract. of femur....	58/40	140	Poor	Very poor	Cold	Marked	26	38	6.0

TABLE II—(*Continued*)

Patient	Diagnosis	B.P.	Pulse Rate	Pulse Quality	Extremities: Venous Filling	Extremities: Temp.	Extremities: Sweat.	Plasma Volume	Hemato-crit	Plasma Protein
J. W.	Fract. both ankles, compd. fract. of humerus	80/50	108	Very poor	Poor	Cool	Moderate	32	42	6.9
C. H.	Compd. fract. both tibia, fibula	165/95 to 120/80	100 to 136	Fair	Fair	Cold	Moderate	26	44	5.4
F. L.	Compd. fract. tibia, fibula; fract. of humerus	60/?	130	Poor	?	Cold	Dry	25	46	6.9
E. K.	Compd. fract. tibia, fibula	82/70	116	Poor	?	Warm	Dry	18	44	7.1
H. M.	Crushed pelvis	60/40	160	Poor	Poor	Warm	Dry	26	33	6.6
S. C.	Fract. femur & pelvis	88/60	100	Fair	Poor	Cold	Dry	25	38	?
R. T.	Fract. pelvis & unobt. femur		120	Poor	Fair	Cold	Dry	24	?	?
E. G.	Multiple compd. fract. of legs, fract. pelvis	90/60	88	Poor	Poor	Cool	Dry	28	?	?
L. G.	Fract. femur	80/40	90	Fair	Poor	Cool	Moderate	26	32	5.4
H. T.	Comp. fract. femur	80/45	108	Poor	Fair	Cool	Dry	35	42	6.7
W. C.	Fract. tibia, fibula	70/30	110	Poor	Poor	Cold	Moderate	28	42	?
A. T.	Fract. femur & lumbar vertebra	56/0	90 to 150		Poor?	Cool	Dry	22	?	?

we have tried to get at least four to six samples after the injection of the dye, so that we could estimate the dye disappearance rate.

Of importance is the fact that in this study all blood samples were taken without the use of the tourniquet. In patients in severe shock it is necessary to draw the blood samples from either an artery or the femoral vein. It has been our custom recently to use the femoral vein for blood sampling almost routinely in patients in severe shock so that the blood is drawn without any stasis.

Hematocrit estimations were made using the Sanford-Magath six-cubic centimeter graduated centrifuge tube; total protein was determined by the Kagan[12] method, which employs the falling-drop principle.

THE RELATION OF PLASMA VOLUME TO CLINICAL SHOCK

A well-organized city ambulance service enables us to observe our shock patients fairly soon after the injury has been received, as a result of which we have been dealing largely with patients in relatively early shock. The patients were brought directly to the Emergency Rooms, where they were placed immediately on a stretcher in the head-down position. No treatment was administered until after the arrival of a member of the "shock team" who were on 24-hour call. As soon as a hasty diagnosis and estimate of the likely severity of shock was established, a plasma volume determination was begun.*

During the time required to secure serial blood samples, clinical observations on the early signs of shock were made and recorded. These observations will be discussed in a subsequent section of this paper.

For convenience of analysis and presentation, we have classified our shock cases into four groups: (1) Acute blood loss; (2) skeletal trauma;

* We are grateful to Dr. Marvin Thompson of the Warner Institute for Therapeutic Research for liberal supplies of the dye T-1824.

(3) abdominal injuries; and (4) chest injuries. In the early part of this clinical study, we were called to see only patients in actual shock, but soon it became obvious that we should attempt to see and study all patients who had suffered severe traumatic injury, whether or not signs of shock were present. Thus, we are able to compare two groups of patients: (a) Those who had no signs of shock or only signs of mild shock; and (b) patients in moderate or severe shock. In general, the types of injury were the same in both groups. (See Tables I-IV)

The *acute blood loss* group is made up of those patients who suffered more or less severe loss of blood from lacerations of peripheral arteries and veins (usually as a result of knife or razor wounds). These wounds were not complicated by muscle trauma. The *skeletal trauma* group consists of all

CHART 3
Acute Blood Loss

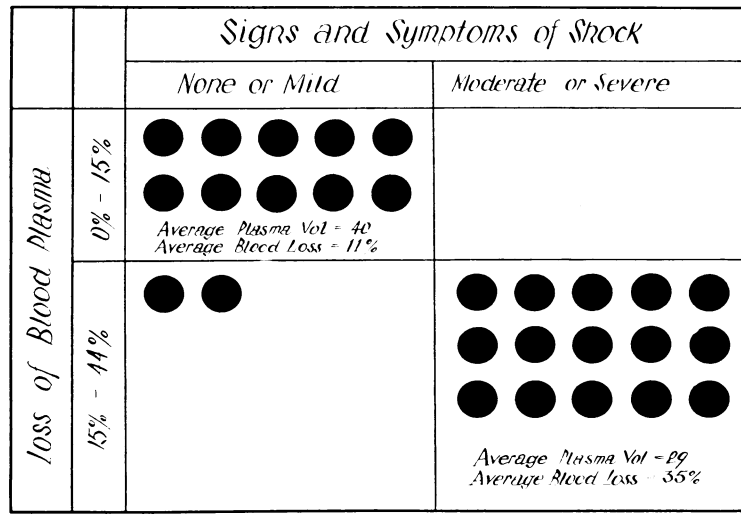

CHART 3.—Scattergram showing the relation of blood loss to severity of shock in simple acute blood loss.

patients who suffered any of the following fractures: Fractures, simple or compound, of the pelvis, femur, or tibia and fibula. The *abdominal injuries* consisted mainly of gunshot or stab wounds (perforating) of the abdominal cavity; also included are several patients with traumatic rupture of the small intestine. The *chest injury* group included all stab and gunshot wounds of the thorax, along with those patients with crushing injuries of the chest. The shock picture in this group is complicated many times by coexisting pneumothorax.

In Tables I, II, III and IV have been placed much of the collected data on this large group of 143 patients who have suffered traumatic injury. Space does not allow for an inclusion in these tables of all the clinical

TABLE III

CHEST INJURY

Shock—None or Mild

Patient	Diagnosis	B.P.	Pulse Rate	Pulse Quality	Venous Filling	Extremities Temp.	Extremities Sweat.	Plasma Volume	Hemato-crit	Plasma Protein
M. P.	Stab wound, hemothorax	88/58 to 120/70	102	Good	Good	Warm	Dry	41	37	5.9
G. B.	Stab wound	90/70 to 110/80	78	Good	Fair	Warm	Dry	44	33	6.7
M. P.	Bullet wound, hemothorax	132/80	80	Good	Good	Warm	Dry	38	35	6.5
A. H.	Bullet wound, chest	130/68	90	Good	Good	Warm	Dry	38	40	?
N. L.	Stab wound, hemothorax	80/60	92	Fair	Fair	Cold	Dry	38	39	?
J. B.	Stab wound	90/70 to 110/70	120	Fair	Fair	Warm	Dry	40	44	7.2
J. M.	Stab wound	90/60	120	Fair	Poor	Warm	Moderate	35	36	5.4
B. G.	Bullet wound	120/80	80	Good	Good	Warm	Dry	38	40	6.2

Shock—Moderate or Severe

C. G.	Stab wound, hemothorax	80/60	98	Good	Fair	Warm	Dry	29	43	7.0
B. B.	Stab wound, hemopneumothorax	60/20	88	Fair	Good	Warm	Marked	29	43	?
L. P.	Bullet wound, hemopneumothorax	50/28 to 80/58	96	Poor	Fair	Cold	Marked	29	43	6.1
W. C.	Stab wound, hemopneumothorax	60/40	95	Fair	Fair	Warm	Marked	33	44	5.7
*H. S.	Multiple rib fractures, axillary vein rupture	165/100 to 70/50	108	Fair	Poor	Cold	Marked	31	37	6.5
E. G.	Stab wound, hemopneumothorax	65/45	100	Poor	Poor	Warm	Marked	26	44	6.7
R. W.	Gunshot wound, chest and abdomen	50/?	160	Poor	Poor	Cold	Marked	31	45	6.8
D. F.	Crushed chest	110/80 to 70/50	70	Good	Fair	Cold	Dry	22	46	6.9
I. P.	Crushed chest	50/0	84	Poor	?	Cold	Dry	25	46	7.8
E. B.	Multiple fract., ribs	70/50	72	Poor	?	Cold	Dry	31	46	6.7
E. H.	Bullet wound, hemothorax	50/20	130	Poor	Fair	Warm	Dry	27	39	6.2
J. C.	Bullet wound, hemopneumothorax	70/40	128	Poor	Fair	Cold	Dry	26	36	5.8
E. W.	Stab wound, hemothorax	50/? to 80/60	120-102	?	?	Cold	Marked	29	49	7.0
W. R.	Shotgun wound, cardiac tamponade	60/40	96	Poor	Good	Cold	Marked	42	45	7.0
L. H.	Stab wound, hemothorax	Unobt.	73	Very poor	Fair	Cold	Dry	26	..	...
J. E.	Fract. ribs, massive hemothorax	40/?	150	Poor	Poor	Cold	Marked	25	45	?
E. M.	Bullet wound, aorta & heart, massive hemothorax	60/40	110	Poor	Poor	Cold	Marked	25	43	7.2
J. F.	Fract. of 5 ribs, hemopneumothorax	75/45	76	Poor	?	Cold	Dry	28	39	?
R. W.	Bullet wound, hemopneumothorax	50/?	160	Poor	Fair	Cold	Dry	32	44	6.8
W. J.	Stab wound, sucking pneumohemothorax	70/50	90	Fair	Fair	Warm	Dry	31	41	7.4
W. B.	Stab wound, hemopneumothorax	60/40	88	Poor	?	Cold	Marked	39	36	6.0
J. B.	Bullet wound, internal mammary artery	unobt.	100	Poor	Poor	Cold	Marked	20	44	6.9
*C. A.	Fract. sternum, contusion of heart	60/40	63	Poor	Good	Cool	Dry	45	44	7.2
H. F.	Contusion of heart	60/30	65	Poor	Poor	Cool	Dry	45	44	7.2
S. B.	Stab wound, internal mammary artery	65/45	68	Poor	Poor	Warm	Dry	28	43	6.9

data collected on the individuals of this group. The great majority of the patients were young or middle aged, and colored. References can be made to these tables for the pertinent shock data on these patients, such as early clinical signs of shock (which will be discussed below), plasma volume, hematocrit, and total plasma protein in per cent.

I. *Plasma Volume* in Acute Blood Loss.*—For purposes of ready analysis, plasma volume data on the individual groups have been arranged in scatter-

CHART 4
Skeletal Injury

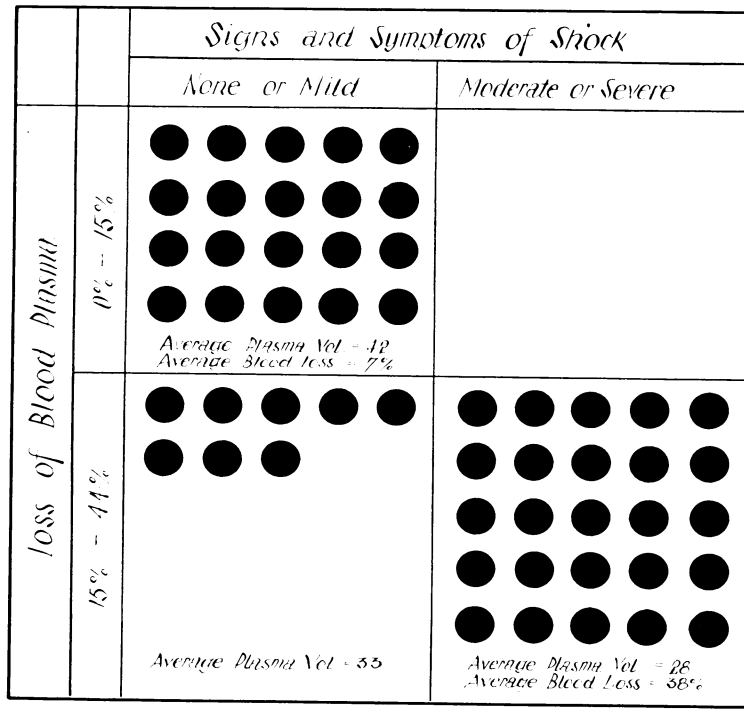

CHART 4.—Scattergram showing the relation of blood loss to the severity of shock in skeletal trauma.

gram fashion. In Chart 3 it will be seen that in the patients with acute blood loss in whom shock was absent, or only mild, the average plasma volume was 40 cc. Kg., representing an average blood loss of only 11 per cent. In the acute blood loss group where shock was moderate or severe, the average plasma volume was 29 cc. Kg., representing an average blood loss of 35 per cent.

II. *Plasma Volume in Skeletal Trauma.*—In the scattergram shown in Chart 4 that group of the skeletal trauma patients who showed no, or only

*We have accepted 45 cc. Kg. as the normal figure for plasma volume for adults. (Gregersen, and our unpublished data)

TABLE IV

ABDOMINAL INJURIES

Shock—None or Mild

Patient	Diagnosis	B.P.	Pulse Rate	Pulse Quality	Venous Filling	Extremities Temp.	Extremities Sweat.	Plasma Volume	Hemato-crit	Plasma Protein
J. M.	Gunshot wound	90/70	120	Fair	Good	Warm	Mild	39	44	6.0
J. K.	Gunshot wound	95/60 to 110/70	80	Good	Good	Warm	Dry	32	46	7.0
M. A.	Gunshot wound	130/70	90	Good	Good	Warm	Dry	38	..	...
S. P.	Gunshot wound	140/100	90	Good	Good	Warm	Dry	43	45	7.0
L. T.	Stab wound	128/80	114	Good	Good	Warm	Dry	39	43	6.2
E. G.	Gunshot wound	105/70 to 90/60	78	Good	Good	Cold	Dry	38	40	6.2
J. B.	Gunshot wound	132/90	96	Good	Good	Warm	Dry	43	54	7.2
B. A.	Gunshot wound	110/85	75	Good	Good	Warm	Dry	39	41	6.8
S. R.	Gunshot wound	120/80	80	Good	Good	Warm	Dry	35	48	6.2
L. F.	Gunshot wound	124/84	123	Good	Good	Warm	Dry	43	43	6.8
D. G.	Stab wound of liver	120/80 to 80/60	100	Fair	Good	Warm	Dry	39	43	6.8
E. P.	Gunshot wound	130/80	98	Good	Good	Warm	Dry	46	44	6.9
A. M.	Stab wound, late periton...	130/70	98	Good	Good	Warm	Dry	24	31	6.3
J. T.	Gunshot wound	140/100	95	Fair	Poor	Warm	Dry	28	49	6.4

Shock—Moderate or Severe

W. S.	Gunshot wound	84/60 to 64/35	120	Fair	Good	Warm	Dry	32	44	6.2
O. H.	Ruptured ileum (traumatic)	60/40	100	Poor	Fair	Warm	Dry	24	55	5.6
M. T.	Gunshot wound	60/40	95	Fair	Fair	Cool	Dry	28	43	6.8
A. M.	Rupt. jejunum (traumatic)	75/50	120	Poor	Poor	Cold	Marked	22	51	7.2
R. J.	Stab wound, liver	50/35	100	Poor	Poor	Cold	Dry	28	38	5.9
C. H.	Rupt. bladder, peritonitis...	140/120	155	Poor	Poor	Cool	Dry	25	57	?
M. J.	Stab wound	50/0	140	Poor	Poor	Cold	Marked	25	37	5.6
W. W.	Rupt. jejunum (traumatic)	90/40	160	Poor	Poor	Cold	Marked	18	60	7.3
H. G.	Gunshot wound	110/70 to 70/50	120	Poor	Poor	Cold	Dry	32	40	?
J. M.	Stab wound	120/80 to 60/40	100	Good	Good	Warm	Dry	34	41	6.9
G. M.	Peritonitis	Unobt.	Unobt.	Unobt.	Poor	Cold	Marked	25	44	5.5
A. C.	Stab wound	80/50	84	Good	Good	Warm	Moderate	32	45	7.2
M. B.	Rupt. uterus	130/70 to 60/30	78	Good	Good	Warm	Dry	29	32	6.0
L. A.	Gunshot wound	70/50	88	Fair	Fair	Warm	Mild	31	40	5.8
L. S.	Stab wound	84/45	70	Fair	Good	Cool	Dry	32	27	7.0
J. M.	Stab wound	105/70 to 80/60	96	Good	Good	Cold	Marked	30	36	5.8

mild, signs of clinical shock have an average plasma volume of 42 cc. Kg., which represents an average blood loss of only 7 per cent. On the other hand, in those skeletal trauma patients in whom shock was moderate or severe, the average plasma volume was 28 cc. Kg., which represents an average blood loss of 38 per cent.

III. *Plasma Volume in Abdominal Injuries.*—In the scattergram shown in Chart 5 it will be noted that the plasma volume of the "none or mild" shock group is 42 cc. Kg., representing an average blood loss of only 7 per cent, while in the moderate or severe group, the average plasma volume is 28 cc. Kg., or a represented blood loss of 38 per cent.

IV. *Plasma Volume in Chest Injuries.*—The study of shock in chest injuries is complicated by at least three important factors, other than blood

loss. Patients may receive heart wounds such that little or no external or internal blood loss occurs, but cardiac tamponade results. Secondly, direct trauma over the precordium may result in cardiac contusion, from which shock may result. Thirdly, the presence of a large pneumothorax, open or closed, complicates and makes more severe any shock, especially when the attendant large blood loss is great.

It will be seen (see Chart 6), therefore, that patients in the chest injury group showing little or no signs of shock have an average plasma volume of 40 cc. Kg., which represents a blood loss of only 11 per cent, while in the group showing signs of moderate or severe shock, there are two patients

CHART 5
Abdominal Wounds

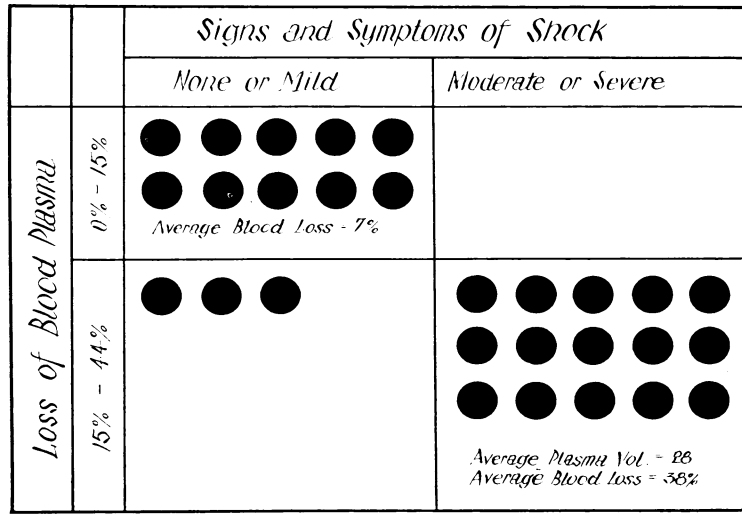

CHART 5.—Scattergram showing the relation of blood loss to the severity of shock in abdominal wounds.

with cardiac tamponade, and two with cardiac contusion, who lost very little blood. However, in all chest injury patients in whom blood loss was a real factor in the production on shock, the average plasma volume was 28 cc. Kg., which represents a blood loss of 38 per cent.

WHAT IS LOST FROM THE BLOOD STREAM IN SHOCK?

Chart 7 represents a diagram of the hematocrit values of these patients. A study of this chart will indicate to the reader that, in the main, the hematocrit values (the average of 3–6 individual readings taken during the plasma volume determination) show little evidence of hemoconcentration. Hence, it is readily evident that what is lost from the blood stream early in shock as a result of trauma must be *whole blood* and not its liquid component,

plasma. The protein figures (to be found for the individual patient in Tables I–V) indicate that if dilution of the blood is taking place by drawing in of fluids from the extravascular spaces to compensate for blood loss, the diluting fluid must closely simulate plasma.

Because of the hematocrit values obtained in this study, we believe we are justified in calculating from our plasma volume data an estimate of whole blood loss which, as has been seen in the various groups of shock

CHART 6
Chest Injury

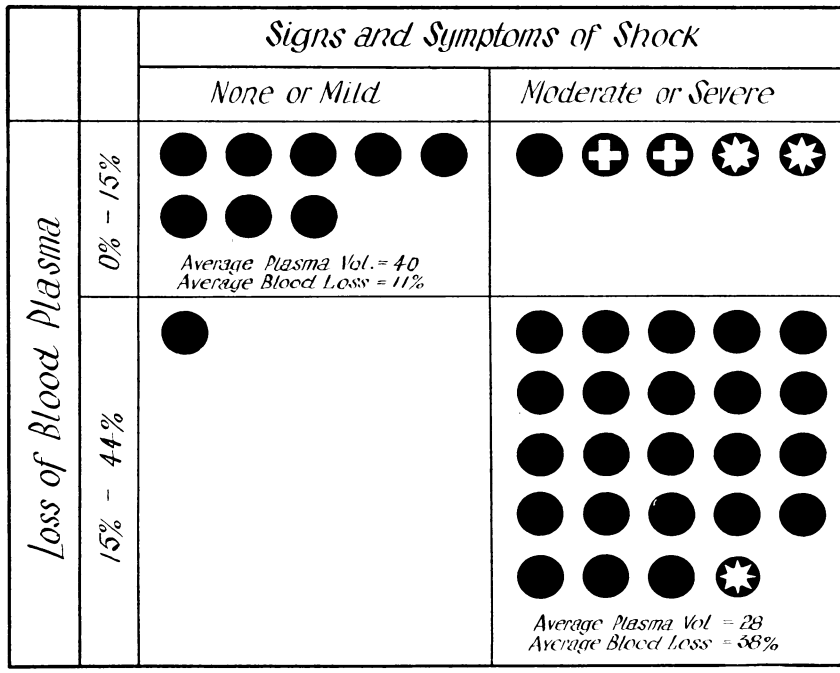

CHART 6.—Scattergram showing the relation of blood loss to the severity of shock in chest injuries.

patients studied, is for the "moderate and severe" groups, 35 per cent, 38 per cent, 38 per cent and 38 per cent, respectively.

It will be noted in Charts 3–6 that several patients, especially in the skeletal trauma group, showed no, or only mild, signs of shock even though the blood loss was greater than 15 per cent. In these patients it is evident that physiologic adjustment to blood loss (other than fluid replacement to the vascular system) was rapidly made. It is noteworthy, however, that only one patient of the entire series (in the chest injury group) showed signs of moderate or severe shock with a blood loss of less than 15 per cent.

In the abdominal injury group, it will be noted on Chart 7, that five patients

had hematocrit readings above 50 per cent. Reference to Table III will show that four of these patients had a ruptured viscus (three—ruptured small bowel; one—bladder). These studies suggest that if a patient is seen in shock with a gunshot wound of the abdomen and the hematocrit value is above 50, the chances are fair that there has been a perforation of a viscus, with peritonitis resulting.

THE EARLY CLINICAL SIGNS OF TRAUMATIC SHOCK

Because of the relative scarcity of information on the *early* clinical signs of shock we have endeavored to collect observations on these points.

(a) *Blood Pressure Readings.*—Blood pressure readings were made on the arm, using a pneumatic cuff and a standardized mercury manometer. At least three or four readings were made during the hour—sometimes more. It is emphasized that these readings are made with the patient in the moderate Trendelenburg head-down position (usually about 15°).

CHART 7

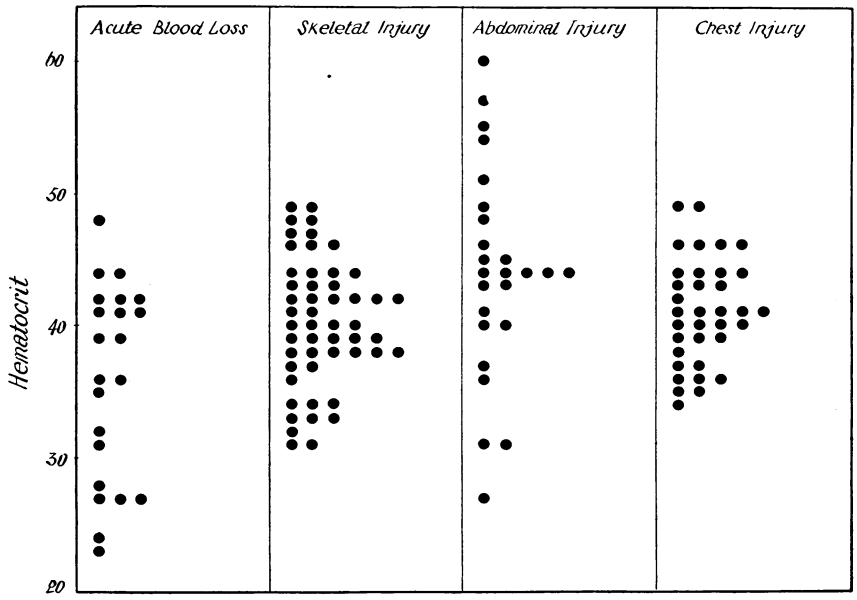

CHART 7.—Scattergram showing the average hematocrit readings in clinical shock produced by acute blood loss, skeletal trauma, abdominal wounds, and chest injuries. Except for the five patients in the abdominal injury group (mentioned in text), there is no indication of hemoconcentration early in the shock state. This suggests the loss of whole blood, rather than plasma alone, early in the shock state.

(b) *Pulse Rate and Pulse Quality.*—Pulse rate and quality were recorded from the radial pulse and usually checked (rate) at the precordium.

(c) *Venous Filling Time.*—This simple test, as employed by us, consists of emptying by pressure stroking of the finger one or more of the visible veins in the outstretched, ventral surface of the forearm and noting the time taken by the vein to refill. Although this is admittedly a crude indicator of peripheral blood flow to an extremity, this test has, at times, given us valuable information as to the severity of shock present, especially in

CHART 8
Acute Blood Loss

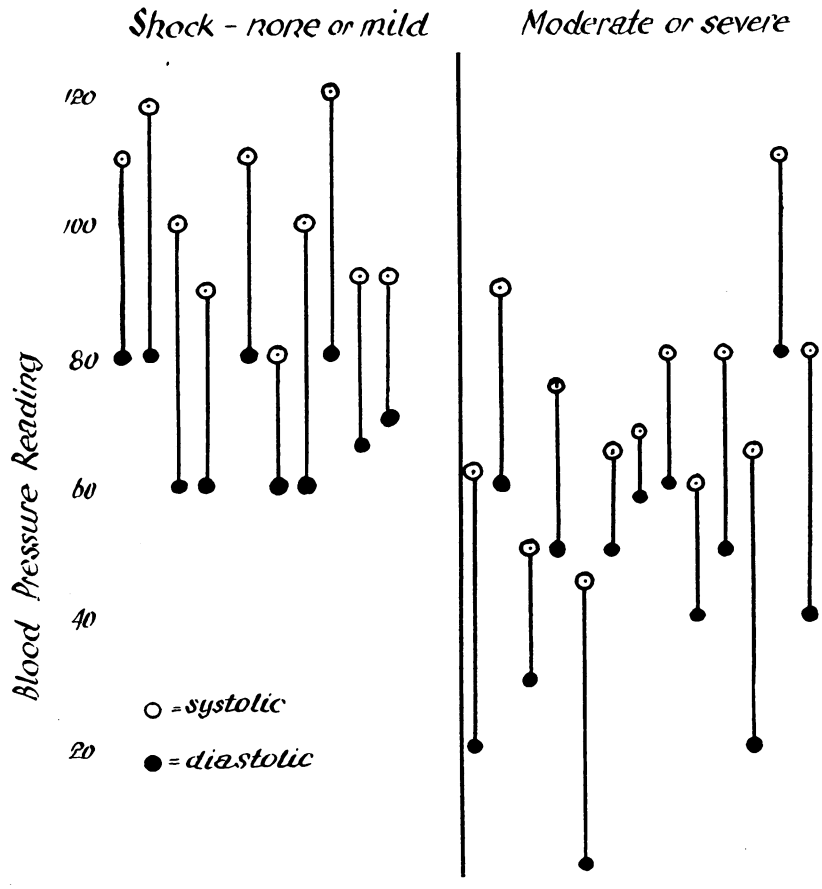

CHART 8.—Diagram illustrating the relation of blood pressure readings to the severity of shock in acute blood loss.

those individuals in whom we have found an initially relatively normal blood pressure but which was found later to be falling.

(d) *Sweating and Temperature of the Extremities.*—The sign of cold, clammy extremities has been accepted by many as being almost always present in shock, so we were interested in making observations on how soon this sign appeared and how well its presence or absence correlated with the blood volume estimation. No attempts were made to record temperature accurately; we simply tried to estimate, in a clinical fashion, whether the hands and feet were warm, cool or cold, and whether they were wet with perspiration or dry.

These are, in general, simple clinical tests that can be applied in the field and which, except in the case of blood pressure readings, employ no special apparatus.

BLOOD PRESSURE IN SHOCK

In Charts 8–11 the blood pressure readings for each group have been

placed together, according to the degree of shock manifested on clinical examination. The data on the blood pressure readings in relation to the severity of shock seem to justify the opinion that, no matter what the injury causing the shock, there is a fairly good correlation between the severity of shock and blood pressure readings. By reference to Tables I to IV, the reader will note that in most cases where the blood pressure was found to be relatively low the patient showed clinical signs of severe shock, *i.e.*, cold, wet extremities, *etc.* We have not tried to work out a correlation between

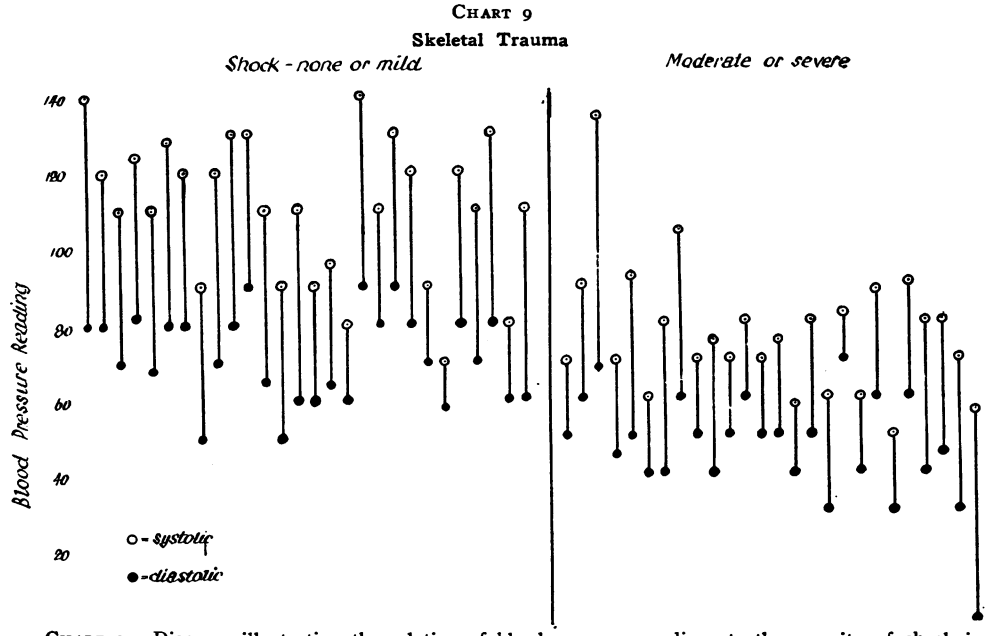

CHART 9.—Diagram illustrating the relation of blood pressure readings to the severity of shock in skeletal trauma.

the blood pressure readings and the degree of reduction of blood volume but it is readily evident from a study of the data in Tables I to IV that only rarely does one find a persistent low blood pressure when there has been little or no blood loss. Indeed, in most patients studied in this series there is a good correlation between the degree of reduction of blood volume and blood pressure readings.

Keith[2] found that cases of wound shock fell into three groups: Group 1. The *compensated* cases in which there was very little blood loss, the blood pressure tending to remain above 100 mm. mercury. In Group 2, the *partially compensated* cases, the blood loss was between 25 per cent and 35 per cent. In these cases the systolic blood pressure was usually between 70 and 80 mm. mercury and a rapid pulse was found. In Group 3, *uncompensated* cases, the blood volume reduction was greater than 35 per cent, and these patients had generally a systolic blood pressure around 60 mm. mercury, with a very rapid heart rate.

The relations between low blood pressure and reduced blood volume,

CHART 10
Abdominal Injuries

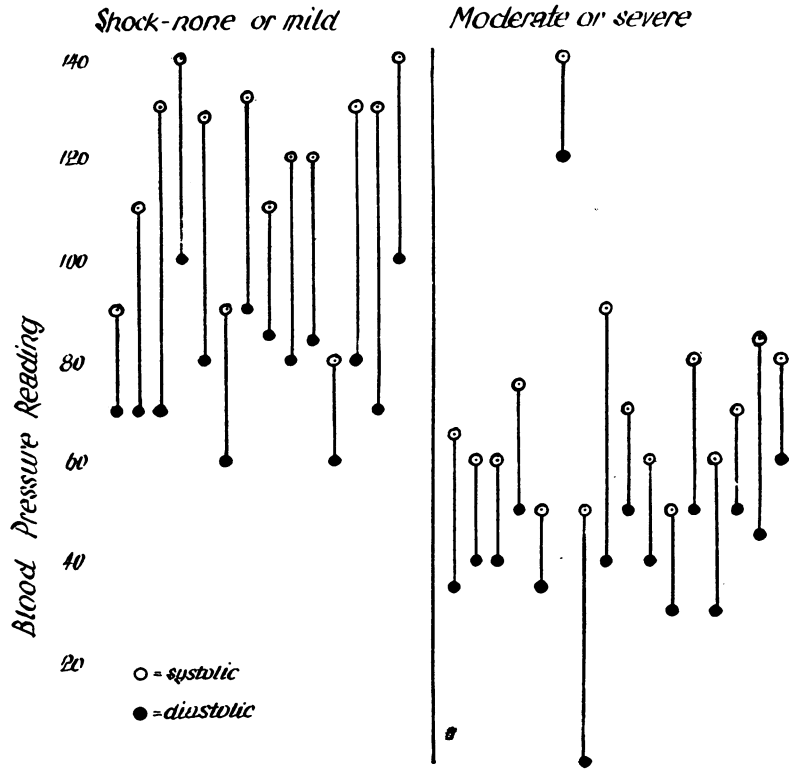

CHART 10.—Diagram illustrating the relation of blood pressure readings to the severity of shock in abdominal injuries.

reported by Keith, correspond to estimates that we have made with improved blood volume methods. An examination of our data leads us to the conclusion that if a patient has received trauma of the types studied by us and the blood pressure tends to remain below 90 mm. systolic, the chances are very great that there has been a considerable blood loss. If, on the other hand, the patient has received severe trauma and, in the Trendelenburg position, maintains a systolic blood pressure above 90 mm. mercury it is probable that he has either suffered little blood loss or has rapidly compensated by blood volume restoration for the amount of blood lost.

In this study, therefore, the blood pressure readings have given us a valuable index as to the severity of blood loss in traumatic shock and the degree of reduction of blood volume. This, likewise, was the conclusion of Kewick, *et al.*,[13] who studied 24 cases of secondary traumatic shock during the bombing of London in 1940.

We have not been impressed with the value of the pulse rate as an index of severe traumatic shock. Reference to Tables I to IV will show that there have been many cases where the blood pressure has been found to be at shock levels, the plasma volume estimation indicating a serious deple-

CHART 11
Chest Injuries

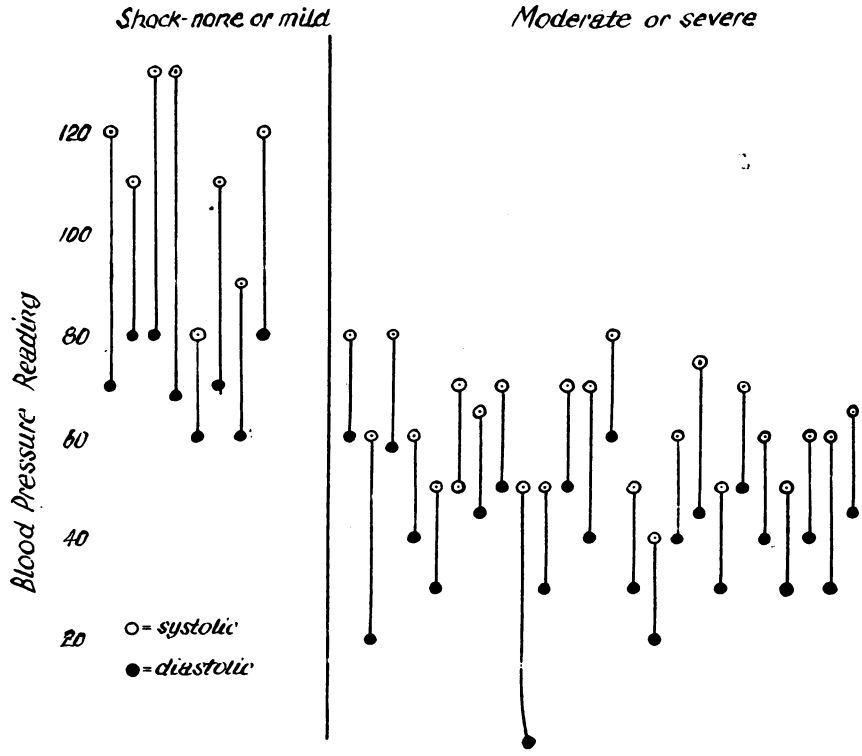

CHART 11.—Diagram illustrating the relation of blood pressure readings to the severity of shock in chest injuries.

tion of blood volume, while the pulse rate remained more or less within the normal range. We have seen no evidence in our study to indicate that in severe trauma a slow pulse necessarily indicates severe shock. In shock cases seen shortly after the trauma has been received, it is not at all uncommon to find a relatively slow pulse; this is especially true in severe chest injuries.

In individual shock cases an estimation of venous filling time has at times given us a valuable index as to the severity of shock. A study of this point in Tables I to IV indicates that in patients who have received severe trauma but who show little or no signs of shock, the venous filling time is within normal limits, whereas in those patients who have received severe trauma and show evidence of moderate or severe shock, the venous filling time may be considerably prolonged.

Likewise, when venous filling time has been found to be greatly retarded, the temperature of the extremities has been found to be considerably lowered from the normal. It was surprising to us, however, to find that in many patients in severe shock there was relatively little sweating of the extremities, so that we are under the impression that the temperature of the extremities is a far more important indicator of shock than whether or not sweating is present. Indeed, very often one finds in severe shock a cool or cold

extremity which remains dry. Dehydration may have been a factor in our series but we are inclined to doubt this because our patients were seen so soon after the trauma had been received.

PATCHY CYANOSIS

During this study we have had the opportunity to observe six patients who have suffered extreme trauma and who have exhibited the phenomenon that we have termed "patchy cyanosis." The picture may be described briefly as small areas of cyanosis (usually 1 to 2 cm. in diameter) scattered closely together on a background of pale, extremely ischemic skin. The phenomenon was seen best on the anterior chest and abdomen. The areas of patchy cyanosis resemble in some respects Bier's spots, except that they are larger and are on a background of ischemic skin. There appeared to be no tendency for these areas of cyanosis to coalesce.

In some regards these areas resemble the patchy ischemia produced in experimental animals by rapid depletion of blood volume, studied so extensively by Rous and Gilding.[14]

Our reason for calling attention to this sign is that, in our experience, its appearance in patients who have suffered severe trauma has portended in all cases, with the exception of one, an early fatal outcome. In one patient large amounts of blood and plasma were given rapidly; this and other signs of shock then disappeared, with the blood pressure assuming more normal levels. The patient died 48 hours later of an associated cerebral lesion. In the other five patients blood and plasma infusions were started soon after the patients were first seen but death ensued before any considerable increase of blood volume could be effected.

It is our impression that if this sign is seen in shock patients every effort should be made to restore blood volume as rapidly as possible. Even so, the outcome will likely be fatal.

EARLY HEMOCONCENTRATION IN TRAUMATIC SHOCK

Ever since the appearance of Scudder's[15] book on shock, it has been thought by many that the determination of specific gravity of whole blood or plasma would give valuable information in the early diagnosis of shock or impending shock in the patient who has suffered trauma. Indeed, Scudder stated in the final summary of his book (page 195) that the weight of a drop of peripheral blood may serve as a measure of this hemoconcentration, and is of more value than blood pressure determinations, as it heralds, by many hours, its ultimate fall. Moon[16] stated: "Experience with this criterion (hemoconcentration) has led me to the conviction that hemoconcentration is the earliest detectable manifestation of shock, as well as the most accurate index of its severity."

We are inclined to believe that determinations of specific gravity of whole blood (or hematocrit determinations) may be of distinct value in following the clinical course of patients with abdominal wounds or other

states in which plasma loss may be profound. It is evident from the analysis of the hematocrit data presented in Chart 7 that the estimation of specific gravity of whole blood would be of little if any aid in the early diagnosis of traumatic shock simply because there is no evidence of hemoconcentration early in traumatic shock. Unfortunately, as this study shows, it is whole blood that is lost in the initial stages of clinical traumatic shock.

From this study of the early clinical signs of shock, we are, therefore, impressed mainly with the value of blood pressure readings. As has been brought out by other writers, the blood pressure reading may be within normal limits when the patient is first seen but if readings are taken every 10 to 15 minutes, in most patients in severe shock (who on blood volume determination will show a considerable decrease in blood volume) it will be found, generally, that the blood pressure readings tend to become lower and lower with the passage of time if treatment of shock is not instituted early. As our group has seen more and more shock patients we have learned that a trained observer can often estimate, with surprising accuracy, the plasma volume simply by taking into account the blood pressure level, the injury, and the state of the patient.

DISCUSSION

It would be unfair to convey the impression that the group of investigators associated with Cannon,[1] and Bayliss,[17] did not appreciate the importance of the depletion of blood volume as a factor in the causation of wound shock. The demonstration by Keith[2] that the shocked man had a seriously reduced blood volume, whether shock was due primarily to hemorrhage or to a combination of hemorrhage and trauma, seems to have been readily accepted. What is more important from a therapeutic standpoint, restoration of blood volume by transfusion of whole blood or gum acacia solution was early advanced as the most efficient method to treat shock. Keith stated definitely that "recovery from wound shock is associated with an increase in blood volume" (page 16). Nevertheless, the question still puzzled many—what was the cause of the reduction of blood volume in wound shock?" ("However, that the reduction of blood volume is secondary to some still unknown primary cause seems evident." [Keith, page 15]).

Although Cannon and Bayliss[18] were convinced of the importance of blood loss as a factor in the production of shock, they entertained strongly the possibility that "the injured muscle would produce metabolites which, on being absorbed into the blood stream, would indicate their presence by a decrease in the blood pressure, with other signs of shock" (page 21).

It is not necessary to go into a long consideration of the many conflicting views regarding the importance of traumatic toxemia as a significant factor in the production of shock, since the evidence has been weighed so carefully in the communications of Parsons and Phemister,[19] and more at length in the excellent treatise of Blalock.[20] As Blalock has pointed out, it is un-

fortunate that so many of the experimental studies which have been carried out to test the correctness of the traumatic theory bear little relation to the clinical problem of shock (the implantation of muscle, *etc.*, into the peritoneal cavity, ligation of muscle masses, intravenous infusion of tissue extracts). In his search for the truth concerning traumatic toxemia, the reader may become bewildered by some of the writings on this subject.

We wish to emphasize here that we have seen the signs of moderate or severe shock appear in patients who have suffered any of four rather different types of body injury, yet the degree of reduction of circulating blood volume in each of the groups was approximately the same. This would indicate to us that in all studies involving an attempt to identify certain toxins as a causative factor in traumatic shock this common factor of blood loss, no matter what the injury, should be properly evaluated.

Certain recent studies have indicated the importance of the decrease in cardiac output as an initiating factor in the production of shock. We are inclined to the view from an analysis of our clinical shock material to believe that in clinical traumatic shock this follows an early reduction in blood volume, soon after the injury has been received. This is more evident in patients who have suffered lacerations of arteries or veins or stab and gunshot wounds of the chest and abdomen. Undoubtedly, in many patients there is a primary fall in blood pressure due to neurogenic causes.

In experimental studies, as Blalock and others have shown, it is possible to show that the decrease in cardiac output precedes a decrease in blood pressure levels. In clinical practice, however, it is our opinion that by the time most patients can be seen after severe trauma has been received, the blood pressure will have fallen to shock levels. From then on both cardiac output and blood volume remain low until efforts are made to restore blood volume by plasma or blood infusions. If restoration of blood volume and return of cardiac output to fairly normal levels cannot be effected, the patient passes more or less rapidly into generalized anoxia, in which state all the capillary walls become affected. When this stage is reached, as clinical experience has shown, blood or plasma infusions are no longer effective.

In our attempt to determine the most important factors in the cause of shock, we have not felt the need to consider toxemia as an initiating or contributing factor. Likewise, there has been no occasion to consider dehydration or exposure to cold as contributing factors since our patients in general were seen shortly after reception of the trauma.

The careful reader cannot fail to be impressed with three factors acting in the production of shock observed in wounded soldiers by Cannon, Bayliss, Robertson, Keith, and others during World War I. Their subjects were in the main (1) cold from exposure to wet, cold atmosphere; (2) apparently dehydrated; and (3) a long time getting back to a point "behind the lines" where shock could be treated properly. Should an active military campaign be pursued again in climates similar to that found in Flanders, exposure to

cold and rain should not be relegated to the background as a possible important factor in the causation of wound shock.

In conclusion, we wish to state that although we regard extreme blood loss at the site of injury as the most important single factor in the causation of traumatic shock, there is no evidence in our observations to exclude the possibility that toxic metabolites absorbed from the zone of injury in severe muscle trauma are not in part responsible for some of the shock picture. This would be especially probable should there be an associated infection in the wound and the patient is seen late. Further, in severe crushing chest injuries where the signs of shock come on rapidly and the patient responds poorly to intravenous infusion of large amounts of blood and plasma, we believe that some cause other than blood loss is responsible for the early fatal outcome in these patients. Indeed, in this group it would appear that there is a rapid and extreme disarrangement of the whole body mechanism. In this group we are inclined to consider seriously a neurogenic factor as being important in the production of shock.

SUMMARY

Using the Gregersen-Gibson method for the estimation of plasma volume in patients who have experienced various types of trauma, it has been found that signs of severe shock do not ordinarily appear unless the blood loss is greater than 15 per cent. The average blood loss in severe traumatic shock has been about 38 per cent, no matter what the nature of the trauma. From analysis of dye disappearance curves, we have found no evidence of increased generalized capillary permeability in traumatic shock. From hematocrit studies, it is evident that what is lost early in traumatic shock in the zone of injury is whole blood, not plasma.

Severe depletion of blood volume appears to be the most important single factor in the causation of traumatic shock.

A decline in blood pressure levels appears to be the most valuable clinical sign in the early diagnosis of clinical shock.

We wish to express our gratitude to our chief, Dr. I. A. Bigger, for arranging the facilities that made these clinical studies possible.

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