BLUNT ABDOMINAL INJURIES IN CHILDREN

Essay

SUBMITTED IN PARTIAL FULFILLMENT

OF THE MASTER DEGREE OF GENERAL SURGERY

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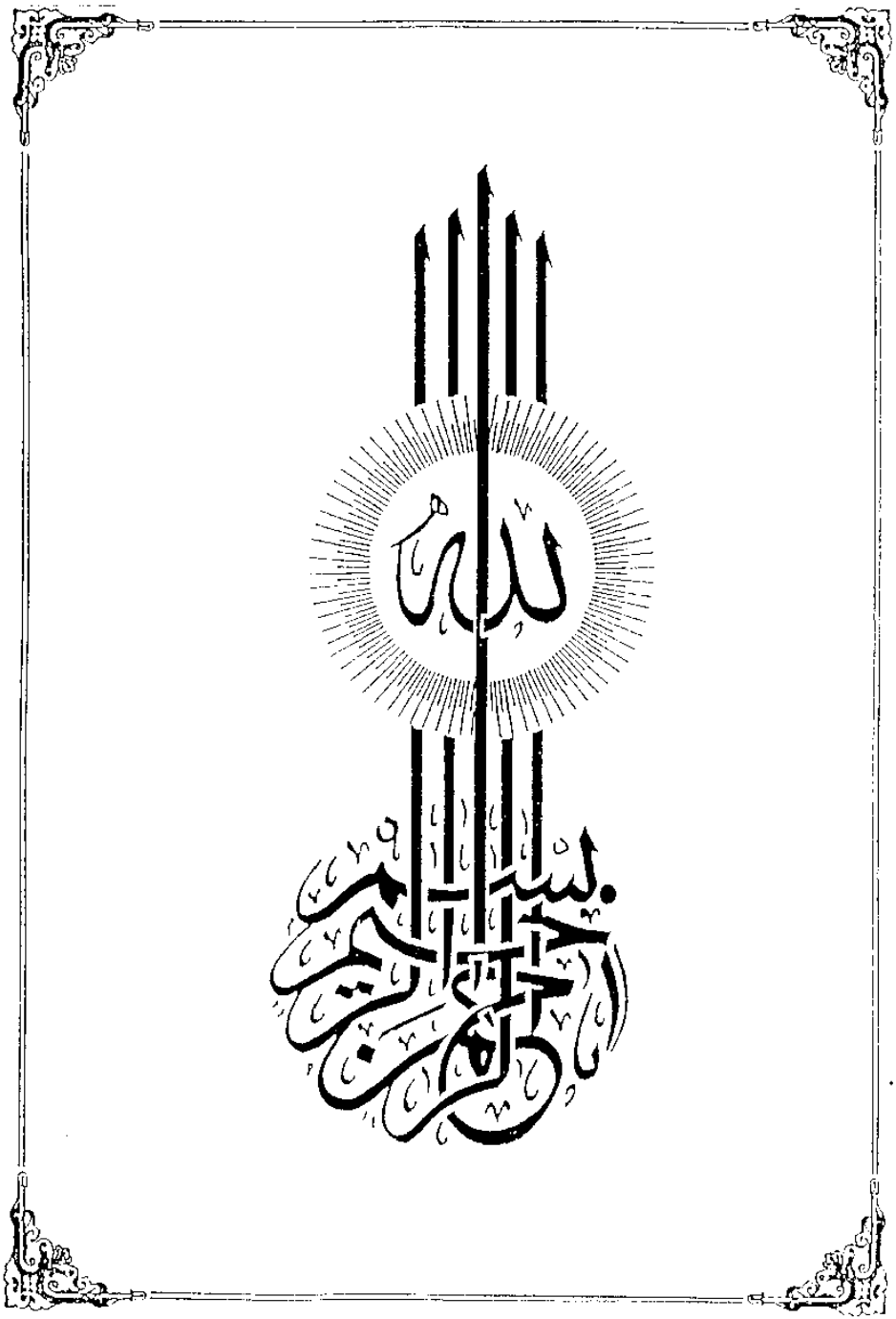

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INTRODUTION

Injuries have been recognized as the leading cause of childhood deaths. While the death rate from natural causes and pediatric diseases has decreased, injuries are responsible for:

* Half of the deaths in children from 1-4 yaears.
* More than half the deaths in children from 5-14 years.

(David E. W., et al, 1989)

More disturbing is the morbidity and long term sequelae of the crippling injuries.

Abdominal injuries in children (which are mostly blunt) contribute to considerable percentage of these injuries.

Generally speaking the thin margin of error, the smaller total blood volume, the age variation for pulse - respiratory rate - blood pressure - and medication dossage and the relative unexperience of primary care providers make the condition more critical.

A scientific study is attempted here including some physiological aspects, etiology, and clinical picture of the patient. Management of these cases will be discussed here in details.

PHYSIOLOGY

* Physiology of shock .
* Splanchnic circulation :

  splenic circulation - functions of the spleen -

  hepatic circulation

SHOCK

The term shock has been recognized for over one hundred years, as more seriously ill patients are presented, the symptom complex of shock is more frequently encountered by the physician. Many attempts had been made over the years to define this complex.

In 1940 Blalock offered the definition " shock is a peripheral circulatory failure resulting from a discrepancy between the size of the vascular bed and the volume of the intra vascular fluids ".
Shock of all forms appears to be invariably related to inadequate tissue perfusion .

An etiologic classification offered by Blalock in 1934 is still useful and functional up till now, he suggested four categories :

* Hematogenic (oligemic or hypovolemic)
* Neurogenic (caused primarily by nervous influences)
* Vasogenic (initially decreased vascular resistance & increased vascular capacity)
* Cardiogenic : - failure of the heart as a pump
- unclassified category including diminished cardiac output from various causes (Tom G. Shires, et al, 1984)

It is now clear that shock invariably results from one or more of four separate but interrelated dysfunctions involving the pump (heart), fluid pumped (blood volume), the arteriolar resistance vessels (mainly the medium sized vessels

), the capacity of the venous side of the circulation .

CLINICAL MANIFESTATIONS

The signs & symptoms of hypovolemic shock when are well established are classic, and usually easy to recognize. One must assume that an injured child who presents with shock (hypovolemic type) has lost at least 20 ml/kg.B.W. of blood (*James A. O.,1986*).

Most of the signs of clinical shock are characteristic of low peripheral blood flow and increased adreno-sympathetic activity. According to the severity of shock, it is classified into four grades :

degree of shock	blood press.	pulse quality	SKIN			thirst
			temp.	colour	cap. circ.	
None	N	N	N	N	N	No
Slight	↓ to 20%	N	Cool	Pale	Slow	N
Moderate	↓20-40%	↓ Volume	Cool	Pale	SLow	Definite
Severe	↓ >40%	Very Weak	Cold	Ashen to cyanotic mottling	Very Slugg-ish	Severe

(Tom G. Shires, et al, 1984)

On inspection of the shock patients (when seen early) they appear to be restless, anxious and give the appearance of great fear, sweaty with collapsed neck viens. With the advance

of cases, this will give the way to a picture of apathy, weakness, and the patient looks sleepy. Finally, the condition will progress to a state of coma and even death .This could be explained by the progressive decrease in the cerebral blood flow and brain hypoxia.

Another characteristic symptom is the thirst sensation which is produced whenever there is increased osmolarity of the extra-cellular fluid (E.C.F.) at the capillary site. A large volume of water moves from E.C.F. to the intra vascular fluid in a trial to compensate the decreased intra vascular volume. This leads to stimulation of certain centers in the anterior hypothalamus which in turn sends impulses to:

* Limbic lobe leading to thirst sensation .
* Posterior pituitary leading to increased secretion of (A.D.H.) decreased urine formation (oligurea)

This may also occur in patients with intense adreno-medullary stimulation from trauma, not necessarily accompanied with shock. So caution must be taken in allowing water intake to avoid dangerous water intoxication .

Another characteristic finding in patients with hemorrhagic shock is the low peripheral venous pressure manifested by small empty viens in the limbs. The veins act as a large reservoir of blood under normal condition. So venoconstriction mediated by intense adreno-medullary stimulation increase the venous return (V.R.) to the heart

increased cardiac output (C.O) in a trial to maintain sufficient blood flow to vital organs .

Another classical finding in hypovolemic shock is the fall in body (Core) temperature. This may be due to lowered metabolic rate or to lower perfusion in areas where body temperature is measured.

Nausea and vomiting from hypovolemic shock are common. It is true that other causes should be thought for (e.g. head injury) but shock alone may be first manifested in this manner.

PHYSIOLOGICAL CHANGES

BLOOD PRESSURE:

Arterial blood pressure (A.B.P) is normally maintained by the following equations:

$$A.B.P \propto C.O \times P.R$$

$$C.O = S.V \times H.R$$

$$= (E.D.V - E.S.V) \times H.R$$

where * C.O ----> the Cardiac Output = the amount of blood pumped by each ventricle/beat/min.

* P.R ----> peripheral resistance

* S.V ----> stroke volume = the amount of blood pumped by each ventricle/beat

* E.D.V.--> end diastolic volume = the amount of blood found at the end of diastole (in the ventricle).
* E.S.V --> end systolic volume = the amount of blood at the end of systole
* H.R.----> heart rate.

So, A.B.P is normally maintained by C.O. and P.R.
When C.O. is reduced by decreased intravascular volume, A.B.P may remain normal so long as P.R. can be increased to compensate for the reduction in C.O. Also, the increased sympathetic discharge will increase H.R. and contractility of the cardiac muscles to compensate for the decreased C.O.
Consequently the B.P. may not fall until the reduction in cardiac output is so great that the adaptive mechanisms can no longer compensate. As the deficit continues there will be progressive hypotension.

PULSE RATE :

Characteristically reduction in the intravascular volume is associated with tachycardia . Any decrease in blood volume will stimulate certain volume receptors present in the media of large sized vessels , which in turn stimulate the cardio-acceleratory center (sympathetic) and inhibit the cardio-inhibitory center (vagal-parasympathetic) ---> increased heart rate .

Also the excessive adrenaline release from the suprarenal

will have a direct stimulatory effect on the S.A.NODE (cardiac pace maker). So according to the equation

C.O = S.V X H.R

= (E.D.V - E.S.V) X H.R

The increased heart rate will elevate C.O towards the normal but we must notice that :

* Excessive tachycardia ---> decrease time for cardiac filling (diastolic time) ---> decreased C.O ---> aggravate the stage of shock .
* Many other stimuli (e.g fear, anxiety) also cause increase heart rate .

VASOCONSTRICTION (V.C) :

The increase in peripheral vascular resistance by production of peripheral V.C rapidly becomes maximal in an effort to compensate for the decreased C.O .The vascular resistance varies in different organs depending upon the degree of V.C or V.D in the vascular bed of this organ .

For example, in case of shock the great increase in peripheral resistance in the skin and kidney causes significant reduction in the flow through these organs , while providing a life saving diversion to the C.O to the more vital organs as the heart and brain .

HEMODILUTION :

All the responses to reduction of intravascular volume eventually result in decreased flow to tissues and the initiation of compensatory mechanisms directed at correction of the low flow state . One of them is movement of fluid into the circulation resulting in hemodilution . This fluid commonly known as extra-cellular fluid (E.C.F) has the same composition of plasma but with lower protein content.

So in hemorrhagic hypovolemia , there will be progressive hemodilution and fall in hematocrite value . The degree of fall will increase with the severity of shock stage . The mechanism of hemodilution is that the reduction of hydrostatic pressure in capillaries because of arterial & arteriolar V.C will result in a shift of pressure gradient to favour the passage of fluid from the extra-cellular space to inside the capillary bed .

* The children have a very good compensatory mechanisms e.g. they have a very good power of myocardial contractility a very good elasticity and peripheral resistance so we can say that the compensatory mechanisms in children is better than those in adult.

* On the other hand loss of 400 c.c blood in an adult person will create small problem as this volume equals less than 1/10 th of his total blood volume . But this is not the picture for a child who weight

about 15 k.g. because these 400 c.c. represent about 1/3rd of his total blood volume.

* Generally speaking the child can compensate for a blood loss up to 20 - 25 % of his blood volume without changing the blood pressure and the only significant change is tachycardia. With the blood loss increase, the child begins to suffer from hypotension .....etc.

SPLANCHNIC CIRCULATION

The splanchnic circulation may be represented to be consisting of three parts :

* The mesentric bed supplying G.I.T.
* The splenic bed
* The hepatic bed

A unique feature of this circulating system is that the combined outflow from the two components (mesentric & splenic) constitutes the major portion (about 70%) of the inflow of the third one (hepatic) (*David K. D. , 1979*) . Such anatomic arrangement allows most of the absorbed substances from the G.I.T. to pass first to the liver via the portal vein where most of the metabolic processes take place before reaching the general circulation. Under resting condition (basal) the splanchnic blood flow is about 1500 cc/min
(about 30% of the cardiac output) (*Ganong W.F. , 1987*) .

SPLENIC CIRCULATION

The nature of the intra-splenic circulation has long been controversial especially the manner of connection between arterial & venous capillaries. Several theories are employed here :-

A) Closed circulation :- blood is passing through preformed channels lined throughout by an endothelium. Anatomically, two types of these channels present :

* venous sinuses which are freely permeable to