Echography of the inferior vena cava as a guide for estimation of "dry weight" in hemodialysis patients

Thesis
Submitted for Partial Fulfillment of Master Degree
In Internal Medicine

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1995

Acknowledgment

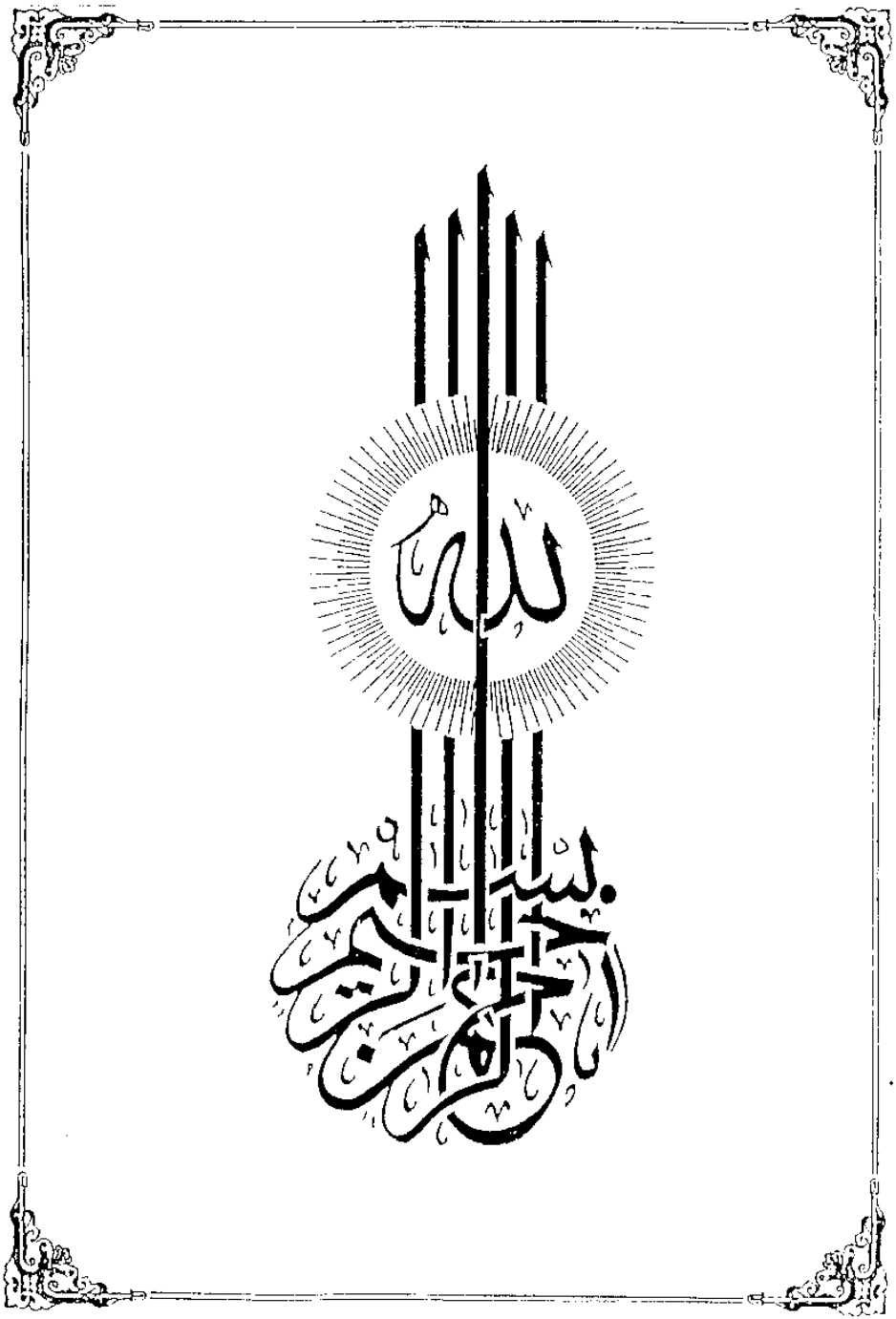
First of all, THANKS GOD..... for helping me to terminate this work

I would like to express my deep thanks and sincere gratitude to ***Prof. Dr. Mohamed Fayed****, Assistant Professor of Internal Medicine, Ain Shams University. I am indebted to him for his close supervision, valuable instructions, encouragement and thorough revision of this work.*

I would like also to express my sincere gratitude and respect to ***Prof. Dr. Mohamed Ali Ibrahim****, Assistant Professor of Internal Medicine, Ain Shams University for his guidance, valuable support, precious instructions and encouragement throughout this work.*

I would like to display my very indebtedness to ***Prod. Dr. Omar Hussien****, Assistant Professor of Radiology, Ain Shams University for his limitless help, valuable advise and kind encouragement.*

Nabil Mohamed Abd El-Aziem



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Introduction

Patients with end stage renal disease have an impaired volume regulation and the excess volume has to be removed during dialysis. Estimation of excess volume is dependent upon estimation of dry weight **(Cheriex et al., 1989).**

Dry weight for a given patient is frequently defined as the weight at the end of regular dialysis treatment below which the patient will become symptomatically hypotensive **(Henderson, 1980).** An incorrect estimation of dry weight will lead either to chronic fluid over load or chronic underhydration. In consequence, this will lead either to hypovolemia-induced hypotension or hypertension, left ventricular hypertrophy and pulmonary edema **(De Vries et al., 1993).**

Dry weight estimation is merely based on clinical grounds such as symptomatic hypotension, cramps and jugular venous pressure which are not very reliable methods **(Leunissen et al., 1993).** Other measures include; anthropometry **(Abd El Fattah et**

al., 1992) and chest X-ray parameters **(Donc et al., 1990).**

Invasive measurements of central venous pressure together with measurement of total blood volume and extracellular volume could give adequate information about fluid status, but these invasive techniques are not suitable for routine clinical practice. More accurate non-invasive method for dry weight assessment is therefore mandatory.

As the inferior vena cava size and dynamics vary with changes in central venous pressure and volume **(Moreno et al., 1984)** and its echographic imaging can be easily performed in most patients, its value as a potential tool in guiding fluid balance during hemodialysis will be investigated.

Aim of the work

Is to assess the value of the echography of the diameter of inferior vena cava in dialysis patients as a tool for estimation of dry weight.

Review of Literature

volemia with volume dependent hypertension, left ventricular hypertrophy and pulmonary edema.

Volume depletion in dialysis patients

During ultrafiltration, fluid is initially withdrawn from the intravascular compartment, so, ultrafiltration induced hypovolemia is a frequent complication **(Kouw et al., 1991)**. Some patients can tolerate aggressive fluid removal with little or no difficulty while others regularly have symptoms in the form of; symptomatic hypotension that yields complaints of anxiety, nausea, muscle cramps and dizziness. With more decline in blood pressure, the patient may exhibit vomiting, diaphoresis or frank syncope.

Relative hypovolemia represents the most common cause of symptomatic hypotensive episodes which in turn is the most commonly encountered complication during dialysis and is estimated to occur in about 25% of treatments **(Henderson, 1980)**.

To understand hypovolemic hypotension, we will begin by examining the normal compensatory mechanisms to hypovolemia. The focus will then shift to uremic patients and dialysis, how

functioning of the autonomic nervous system may be altered in uremia will be examined. The review will analyze how certain dialysis technique can adversely affect some of the compensatory hemodynamic responses to hypovolemia and how increased body temperature during dialysis and ingestion of food or glucose can have undesirable hemodynamic consequences. Finally a set of practical recommendations for optimizing blood pressure response during dialysis therapy will be presented.

Physiologic responses to hypovolemia

Compensatory responses to hypovolemia include: (a) Mechanisms that decrease venous capacity (which help maintain cardiac filling), (b) Mechanisms that increase cardiac contractility and rate (which optimize cardiac output under conditions of decreased filling) and (c) Mechanisms that increase vascular resistance (which decrease venous capacity by an indirect mechanisms which redistribute the cardiac output among the various vascular beds to ensure perfusion of vital organs) **(Daugridas, 1991)**.

(a) Decreased venous capacity

A substantial percentage of total blood volume is located in the venous system, the capacity of which can change markedly. Translocation of this

blood volume centrally during hypovolemia can result in an increase in rate of venous return **(Rothe, 1983).**

During subacute hemorrhage, in addition to redistribution of blood volume, actual refilling of plasma compartment with fluid and electrolytes from adjoining tissue spaces and with protein from thoracic lymph **(Zollinger, 1972)** can also help maintain venous return.

There are two mechanisms where by the capacity of regional circulatory bed can be reduced: active reflexly mediated venoconstriction and decreased regional filling. The extent to which active venoconstriction occurs in human during hypovolemia is controversial. A more important mechanism is decreased regional filling.

How decreased regional filling can increase venous return, when the resistance in vessels supplying a compliant vascular bed increased, flow is decreased as well as down stream distending pressure. Passive recoil of the venous bed occurs reducing its capacity. A portion of the blood volume previously in the venous bed is shifted back towards the heart, increasing cardiac filling. This

phenomenon is called DeJager Krogh phenomenon **(Rothe, 1983).**

The two vascular beds that participate most extensively in decreased venous capacity during hypovolemia are the splanchnic and cutaneous circulation.

The splanchnic bed is composed of the circulation to spleen, liver and intestine. Although the relative contribution of spleen, liver and intestine to splanchnic-mediated volume redistribution are not known, the fact that splanchnic capacity decreased during hypovolemia is well established **(Rowell, 1975).** Although the removed, perfused spleen is incapable of changing its volume **(Ayers et al., 1972)**, the in situ human spleen does appear to contract on change to upright position and during exercise, with the addition of previously sequestrated erythrocytes to the circulation **(Sandler et al., 1984).**

The cutaneous circulation is an extremely important blood reservoir. In man, the volume of which can increase markedly during heat stress and which can contract during hypovolemia.

During hypovolemia, flow as well as down stream distending pressure are reduced in several other circulatory beds which receives a substantial portion of cardiac output (that is muscle and kidney). However, the compliance of the muscle and kidney beds is such that their vascular capacity decreases only minimally during hypovolemia with minimal augmentation of venous return.

(b) Increased heart rate and contractility

Heart rate:

The normal cardiac response to hypovolemia is an early tachycardia followed by a reduction in heart rate back to normal or even a subnormal level **(Sander-Jensen et al., 1988).** It would seem that, as long as stroke volume is maintained, an increase in cardiac rate should result in an important increase in cardiac output and blood pressure. However, during hypovolemia, changes in cardiac rate have been shown to be of minor importance.

In a study in human in whom hypotension was induced by lower body negative pressure, blocking the late slowing of the heart rate with atropine had no effect on blood pressure response **(Sander-Jensen et al., 1988).**

Contractility:

Increase in cardiac contractility can increase cardiac output by increasing stroke volume. However, there is evidence that increase in cardiac inotropy are not very important during hypovolemic conditions. In conscious dogs, Beta adrenergic blokade **(Hintze et al., 1982)** has minimal effect on the hemodynamic response to hemorrhage. It appears therefore that in hypovolemia, cardiac output is determined primarily by the amount of cardiac filling. The heart can pump out only what flows in and optimizing pump performance under conditions of severely decreased filling is of very limited benefit **(Rothe, 1983).**

(c) Increased vascular resistance

Increased peripheral vascular resistance during hypovolemia has three effects: (1) increased resistance to splanchnic and cutaneous beds with relative increase in venous return, (2) reduced flow to renal and skeletal muscle beds which allows a large portion of the cardiac output to be directed to more critical regional circulation and (3) because overall resistance is increased, the decrease in pressure in the proximal arterial circulation due to decreased cardiac output is minimized **(Daugridas, 1991).**

Important cardiovascular reflexes during hypovolemia:

The two principal reflex arcs that affect vascular tone during hypovolemia involve the cardio-pulmonary receptors (located in the atria and in the region of main pulmonary veins) and the presso-receptors (located in the aortic arch and carotid bifurcation regions).

In the resting state, both sets of receptors exert a tonic inhibition of sympathetic outflow to resistance vessels in skeletal muscles and skin. The cardio-pulmonary receptors are believed to be a first line of defense against hypovolemia; a change in cardio-pulmonary receptors can be documented even at levels of hypovolemia, under conditions where a reduction in pulse pressure or mean arterial pressure is not observed.

When a cardio-pulmonary receptors sense a decrease in cardiac filling pressure and/or volumes, they become deactivated that is they cease their tonic inhibition of sympathetic outflow **(Victor and Leimbach, 1987)** and sympathetic tone to muscle and cutaneous arterioles is increased. As a result, cutaneous and skeletal muscle flow are reduced with redistribution of blood volume towards the heart