International Journal of Scientific Research Updates

Journal homepage: https://orionjournals.com/ijsru/

ISSN: 2783-0160 (Online)

(REVIEW ARTICLE)

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Assessment of liver blood flow regulation mechanism by ultrasound

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International Journal of Scientific Research Updates, 2022, 04(01), 150–154

Publication history: Received on 08 July 2022; revised on 11 August 2022; accepted on 13 August 2022

Article DOI: https://doi.org/10.53430/ijsru.2022.4.1.0110

Abstract

The constancy of hepatic blood flow is important for several homeostatic roles. This paper reviews studies related to liver blood flow regulation mechanism. The liver has mechanism maintain a constant hepatic blood flow and compensate for portal flow changes. A reduction in portal flow leads to reduced intrahepatic distending pressure, resulting the hepatic arterial buffer response causes dilation of the hepatic artery, thus buffering the Portal flow change. This mechanism is exclusive to this specific vascular bed and offer a great illustration of multiple integrative regulation of a significant homeostatic organ. Ultrasound provides noninvasive accurate information on the liver hemodynamic and assess the flow in the hepatic artery and the portal vein specially diameter of vessels.

Aim: The aim of this study was to evaluate the dual hepatic blood supply in normal individual and compared altered in the hepatic artery buffer response with chronic hepatic diseases.

Materials and methods: The current review focuses on researches that evaluate the mechanisms involved in maintaining a steady hepatic arterial buffer response (HBF), such as the impact of portal blood flow on hepatic artery flow, or assessing the intrinsic regulation of hepatic arterial blood flow by measure the hepatic arterial buffer response intraoperative in patients with or without cirrhosis using an ultrasound transit-time flowmetry**,** or by duplex ultrasound, Moreover studies to understand the role of hepatic flow in liver atrophy.

Keywords: Ultrasound; Liver; Hepatic arterial buffer response; Hepatic artery; Portal vein

1 Introduction

The liver receives three-quarters of the blood supply through the portal venous drainage which approximately 25% of the entire cardiac output. The liver has indirectly controlling portal blood flow through very significant effects to regulate portal blood flow via the hepatic arterial buffer response mechanism. Constancy of hepatic blood flow is important for the homeostatic of cardiovascular stability. There are multiple mechanisms that act to maintain a constant hepatic blood flow. (1)

According to Burton-Opitz reduced portal blood flow affect hepatic arterial flow. (2) and has since been examined directly in only a few cases. The relationship can frequently be seen in the literature, although the term hepatic arterial buffer response was not used until 1981(3)

Burton-Opitz, studied the influence of the portal blood-flow upon the flow in the hepatic artery he found the arterial blood flow in the liver can be altered by a variety of factors, including blood-borne factors reaching the arterial resistance sites and nerves. (2)

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Lautt, 1985 assess the mechanism and role of intrinsic regulation of hepatic arterial blood flow he found that the hepatic arterial blood flow response to altered portal blood flow is not dependent on hepatic metabolic demands. A hepatic arterial buffer response minimizes fluctuations in total liver blood flow by buffering the effects of altered portal flow on hepatic arterial blood flow. Teleologically, it is important for hepatic flow to be regulated so that short-term abrupt changes are minimized in order that the hepatic clearance rate of hormones, and possibly nutrients, is maintained relatively constant. Adenosine levels affect the mechanism of regulation of the hepatic arterial buffer response.

Tissue in-the area of the hepatic resistance vessels produces a constant level of adenosine that can either diffuse into portal blood and be washed away from the area of production, or it can also dilate the hepatic artery locally by acting on adenosine receptors. If portal flow is reduced less adenosine is washed away, resulting in local accumulation and elevated hepatic arterial blood flow.(3)

Ultrasound plays a crucial role in early diagnosing portal hypertension or occlusion to prevent complications as well as reduce costs and time spent on exams.(4)

2 Impact of intrinsic blood flow regulation in cirrhosis

Blood volume is effectively controlled by the hepatic arterial buffer response (HABR), however, the regulation of blood flow is poorly understood in cirrhosis. The portal vein is normally the major source of blood supply for the liver. Cirrhosis causes the ratio of portal venous to hepatic arterial blood flow to be shifted toward the hepatic artery, which may sustain oxygen delivery and protect organ function and integrity. Measurement was made for hepatic arterial and portal venous blood flow as well as mean arterial and portal venous blood pressure over experimental periods up to 6 h. Since portal venous blood flow is reduced in cirrhosis, maintaining hepatic arterial blood flow may counteract impaired nutrient blood supply to the cirrhotic liver. (5)

Doppler ultrasound is used to determine correlations between liver cirrhosis severity and the hemodynamic blood flow of the liver in patients with portal hypertension. Hemodynamic variables in this study obtained from a Doppler examination were correlated in different ways. According to the results, cirrhotic patients had a larger portal vein diameter than control subjects. A significant reduction in portal vein velocity and blood flow was observed in advanced forms of liver cirrhosis. Compared with controls, patients with cirrhosis had significantly higher portal vein congestion indexes, pulsatility indexes, and hepatic artery resistivity indexes. Patients with advanced cirrhosis had a lower liver vascular index. The study's conclusion states that ultrasound is an effective noninvasive tool to treat cirrhotic patients with portal hypertension. For cirrhotic patients with portal hypertension, standard 2D and Doppler ultrasounds are important, noninvasive screening tools. (6)

Hepatic fibrosis progression and hemodynamic disturbances can be detected by computational fluid dynamics analysis, as demonstrated by Du et al. They suggested that computational fluid dynamics may assist in the diagnosis of hepatic fibrosis, and changes in hemodynamics (7).

In Harvey's model, the arterial flow counteracts the changes in portal venous flow by an electrical analogy based on the hepatic arterial buffer response (HABR). To account for dynamic HABR effects, the model introduces nonlinear arterial and portal resistors, which simplify a previously published model. In this study, they calibrate the baseline model based on published hemodynamic data, and then simulate a virtual portal occlusion in which half or all of the portal vein is occluded. It is consistent with clinical reports and animal model data that increased arterial flow cannot fully compensate for lost portal perfusion in simulations. Hepatic blood flow is preserved by HABR, an important liver mechanism. In hepatic surgeries where both HA and PV contribution to total hepatic flow are frequently altered, it must be carefully monitored. Conclusion recommend establishing a model that agrees with physiological data and appropriately adapts to changes in inflow parameters will help scientists and clinicians better understand the pathophysiological consequences of changes in the hepatic blood flow. If the model is further adapted, it may be able to be used to study other hepatic circulation conditions.(8)

Achim also used Doppler measurement to study the hemodynamics of the hepatic arteries in children with extrahepatic portal vein obstructions. Using Doppler indices, he studied the Hepatic artery in children with extrahepatic portal hypertension. To determine the flow pattern within the hepatic artery, the resistive index, arterial acceleration time, and acceleration index were used. Compared with controls, children with extrahepatic portal hypertension had normal hepatic artery resistive indexes. There was a significant difference between cases and controls in the hepatic arterial early systolic acceleration index.

In children with chronic extrahepatic portal hypertension, hepatic arterial early systolic acceleration was significantly increased. Ternberg and Butcher reported in 1965 the relationship between portal vein and hepatic artery blood flow As a result of the obstruction in portal veins (slow flow), a rapid flowing arterial stream (hepatic artery) is mechanically interposed. (9)

In patients with or without cirrhosis, Aoki et al. measured the hepatic arterial buffer response intraoperatively. They state the hepatic arterial buffer response (HABR) is an intrinsic regulatory mechanism of the hepatic artery (HA) that makes up for decreases in portal venous blood flow. Because of wither this response is maintained in patients with cirrhosis is unclear. The aim of their study was to examine whether HABR is maintained in patients with liver cirrhosis using direct blood flow measurements. HABR was assessed in patients with or without liver cirrhosis at the time of surgery. Using ultrasonic transit-time flowmetry, which measures blood flow volume, the PV and HA blood flow in these individuals were assessed at the same time. Measurements were taken both before and after the PV was acutely clamped. In their conclusion, they note that in patients with liver cirrhosis, the baseline HABR appears to be continually activated; this characteristic most likely causes the acute HABR to be impaired.(10)

Iwao compared the hepatic artery hemodynamic response to changed portal blood flow in livers with normal and cirrhotic conditions in his study.

Before and after patients consumed a 500 kcal mixed-liquid meal and during an intravenous infusion of vasopressin at a rate of 0.3 U/mm, he evaluated the portal blood flow and hepatic artery pulsatility index by duplex ultrasonography. The ratio of the largest change from baseline in the hepatic artery pulsatility index to the maximum change from baseline in portal blood flow is known as the hepatic artery buffer index was also calculated. According to the findings, eating improved each subject's hepatic artery pulsatility index and portal blood flow. However, cirrhotic patients' hepatic artery buffer index was substantially lower than that of control subjects.

Vasopressin infusion reduced the hepatic artery pulsatility index and portal blood flow in all participants. Once more, cirrhotic participants' hepatic artery buffer index was much lower than that of control subjects. In conclusion, it was discovered that cirrhotic livers have decreased hepatic artery vascular reactivity to changed portal blood flow. (11)

3 Mechanism of hepatic artery dilation

A study of the literature demonstrates that hepatic vascular bed is special in numerous ways including it gets blood flow from the portal vein and hepatic blood vessel. These blood vessels travel as their terminal branches through a little space called the space of Mall. The hepatic and portal venule are drain into the hepatic sinusoids. Adenosine appears to be produced at a constant rate independent of oxygen supply and is secreted into the space of Moll, which acts as a powerful hepatic artery dilator.

According to this theory, a decrease in portal blood flow results in a decrease in adenosine exudation, and the accumulated adenosine concentration causes hepatic artery dilation, which compensating for the decrease in portal blood flow. The buffering capacity of this mechanism only partially compensates for changes in portal flow, but often completely compensates for diminished oxygen supply, even in the case of cirrhosis. (1)

Eipel et al. at their research of regulation of hepatic blood flow focus on the endogenous interrelationship between the hepatic arterial and portal venous inflow circuits in liver resection and transplantation, as well as inflammatory and chronic liver diseases and they found that the crucial importance of the HABR as a regulatory mechanism to maintain adequate liver function and metabolic homeostasis has been recognized. (12)

Decreased portal flow activates the hepatic artery buffering response (HABR) followed by decreased exudation of adenosine from the space of Moll before it is excreted into the hepatic sinusoidal syncytium. The rate of adenosine infiltration into portal blood regulates the concentration of potent vasodilators. By this mechanism, a decrease in portal blood flow results in adenosine accumulation and hepatic artery dilation, which helping to buffer changes in portal blood flow to total hepatic blood flow. This is the mechanism of HABR. The goal of this mechanism is to keep the entire liver perfusion within acceptable physiological limits.(8).

4 Conclusion

- This paper presents research on the hepatic arterial buffer response (HABR) in humans. In clinical practice, it is becoming increasingly apparent that the HABR plays a critical role in maintaining adequate hepatic perfusion.

There is no direct mechanism to control the liver's portal inflow. When portal flow changes, the portal and intra-hepatic pressure change, which passively acts against the compliant vascular bed, thereby regulating cardiac output through the liver.

- Reducing portal blood flow also activates the hepatic arterial buffer response by washing away less adenosine from the space of Mall surrounding the hepatic arteriole resistance vessels. In order to cause the hepatic arterial buffer response, adenosine vasodilates the hepatic artery.
- Although the buffer capacity of this mechanism results only in a partial compensation for changes in portal flow, it often results in a full compensation for the decrease in oxygen delivery even in cirrhotic livers.
- Multiple regulatory mechanisms interacting to maintain hepatic blood flow constancy, the hepatic arterial buffer response (HABR) is an intrinsic regulatory mechanism of the hepatic artery (HA) that compensates for reductions in portal venous (PV) blood flow.
- Hepatic arterial blood flow is altered by a variety of stimuli, including nerve and blood mediators, and can reach arterial resistance sites via arterial or portal blood.
- The hepatic arterial blood flow response to altered portal blood flow is not dependent on hepatic metabolic demands. But it occurs in a manner that buffers the impact of altered portal flow on total hepatic blood flow, that is, the hepatic arterial buffer response is regulated to minimize fluctuations in total liver blood flow.
- By the hepatic arterial buffer response (HABR) mechanism, reduced portal flow leads to adenosine accumulation and hepatic arterial dilation, thereby buffering the effects of changes in portal flow on total hepatic blood flow.
- HABR is meant to maintain a physiologically acceptable level of total hepatic perfusion.
- In patients with liver cirrhosis, the HABR is continuously activated, which probably impairs the acute HABR.

Recommendation

HABR plays a crucial role in maintaining adequate liver function and metabolic homeostasis. Efforts must be made to modulate altered hemodynamics in small-for-size livers, cirrhotics, and critically ill patients. To prevent potential problems arising from altered or impaired HABR, every effort should be made to diagnose it as soon as possible. The goal is to improve the outcome of patients suffering from liver disease with abnormal hepatic hemodynamics.

Compliance with ethical standards

Acknowledgments

We thank Prf. Mohammed El Mutasim Abd Albagi for his kind provision of the Knowledge and advice regarding ultrasound.

Disclosure of conflict of interest

No any conflicts of interest.

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