Research Article

Understanding the Pathophysiology of Portosystemic Shunt by Simulation Using an Electric Circuit

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Portosystemic shunt (PSS) without a definable cause is a rare condition, and most of the studies on this topic are small series or based on case reports. Moreover, no firm agreement has been reached on the definition and classification of various forms of PSS, which makes it difficult to compare and analyze the management. The blood flow can be seen very similar to an electric current, governed by Ohm’s law. The simulation of PSS using an electric circuit, combined with the interpretation of reported management results, can provide intuitive insights into the underlying mechanism of PSS development. In this article, we have built a model of PSS using electric circuit symbols and explained clinical manifestations as well as the possible mechanisms underlying a PSS formation.

1. Introduction

Portosystemic shunt (PSS) is a common condition and usually follows portal hypertension or liver trauma, including iatrogenic injury [1–3]. However, congenital or spontaneous PSS can also occur and presents diagnostic along with management challenges [3]. The definition and classification of PSS are in a chaotic status in respect to its cause, location, and anatomical inflow/outflow vessels. This situation probably arose because of lacking consensus, due to most of the relevant literature being composed of case reports or small series [4, 5]. The blood flow is basically similar to an electric current, in that it is determined by pressure difference and resistance, governed by Ohm’s law [6]. In this article, we tried to develop a model of PSS using electric circuit symbols and applied it to the interpretation of the reported management results of PSS. Also, we suggested that PSS can be classified according to two distinct underlying mechanisms.

2. Materials and Methods

2.1. Developing an Electric Circuit Model of PSS. The schematic diagram of the splanchnic circulation is presented in Figure 1. By representing the blood flow as an electric current and the vascular resistance of intraabdominal organs as resistors, the intraabdominal vascular system can be further simplified using electric symbols (Figure 2). We assumed that the aortic pressure ($V_{AO}$) and mesenteric vascular resistance ($R_M$) are constant and the systemic venous pressure ($V_{IVC}$) is approaching zero (grounded).

2.2. Literature Review. We have reviewed the English literature articles that were published between 1999 and 2014 and searched case reports or series which presented the management results of PSS, with special focuses on the site of shunt blockade and the postoperative evolution of PSS. The occlusion site was divided according to the location of the blockade with respect to the shunt flow, that is, the inflow, shunt per se, and the outflow. The management results were classified according to whether PSS disappeared or not after the shunt occlusion. The former was described as collapsed and the latter as persistent.

2.3. Understanding the Pathophysiology of PSS. The possible explanations regarding the pathogenesis of PSS were deduced by applying the circuit theory to the reported management results, including our own case reported elsewhere [7].
3. Results

3.1. Clinical Application of Electric Circuit Model. In normal condition, \( R_S \) is sufficiently high and the shunt flow \( (I_S) \) is negligible, and the basic configuration of PSS model is essentially two resistors connected in series. It is a voltage (=pressure) divider with \( R_M \) and portal venous resistance \( (R_P) \), and the portal pressure \( (P_{PV}) \) can be calculated by the formula \( P_{PV} = V_{AO}/(R_M + R_L) \). In other words, portal pressure is directly proportional to portal venous resistance. When a disease process increases the portal venous resistance, such as in liver cirrhosis, portal pressure will increase as well. Therefore the pressure difference across the shunt increases. By Ohm's law, the shunt flow is defined as \( I_S = P_{PV}/R_S \). If \( P_{PV} \) becomes sufficiently high, \( I_S \) can become greater than zero, resulting in PSS formation. The other way for \( I_S \) to increase is for \( R_S \) to decrease at a fixed \( P_{PV} \). A clinical example is aneurysmal dilatation of the collateral channel, whether intrahepatic or extrhepatic. Once \( R_S \) has decreased, \( P_{PV} \) also decreases because \( R_L \) and \( R_S \) are connected in parallel. The portal venous flow \( (I_P) \) decreases consequently, implicating the portal flow to bypass the liver.

3.2. Literature Review. The reported management results of a PSS are presented in Table 1. Most articles described the result as the improvement in the encephalopathic symptoms, not as the morphologic change of the PSS. In 10 cases out of 49 reviewed (20.4%), the morphologic evolution of the PSS was identified. PSS had disappeared or collapsed in 7 cases, whilst in 3 cases, PSS persisted or thrombosed after the occlusion of the shunt by various modalities. Of note, there was no case in which PSS persisted after inflow occlusion, while there were two reported cases in which PSS had collapsed after outflow occlusion.

3.3. Understanding the Pathophysiology of a PSS. The cause of a PSS can be deduced by combining the shunt blockade site and the treatment results (Table 2). When a PSS was formed by the increase in \( P_{PV} \), the evolution of PSS after treatment would vary according to the occlusion site. If the inflow

| Authors          | Liver cirrhosis | Shunt location | Block site (modality) | Result        |
|------------------|-----------------|----------------|-----------------------|---------------|
| Hiraoka et al. [8] | No              | Intrahepatic   | Inflow (embolization) | Collapsed     |
| Lee et al. [9]    | No              | Intrahepatic   | Inflow (embolization) | Collapsed     |
| Chagnon et al. [10]| No              | Intrahepatic   | Shunt per se (resection) | Collapsed    |
| Lee et al. [9]    | No              | Intrahepatic   | Shunt per se (embolization) | Collapsed    |
| Shimoda et al. [11]| Yes             | Extrahepatic   | Shunt per se (surgical closure) | Collapsed    |
| Cauchy et al. [12]  | Yes             | Extrahepatic   | Shunt per se (surgical closure) | Persistent    |
| Machida et al. [13]| No              | Intrahepatic   | Outflow (graft insertion) | Collapsed    |
| Kwon et al. [7]   | No              | Intrahepatic   | Outflow (surgical closure) | Collapsed    |
| Seman et al. [14]  | Yes             | Extrahepatic   | Outflow (surgical closure) | Persistent    |
| Hara et al. [15]  | No              | Intrahepatic (patent ductus venosus) | Outflow (surgical closure) | Persistent    |
assuming that the systemic venous pressure is \( \sim R \).

Representative clinical conditions in which collateral vessels dilate or new shunt channel appears [2].

The draining vein can be a hepatic vein, ductus venosus, an umbilical or paraumbilical vein, or other systemic veins [2, 17]. A shunt implies flow and can be simulated using an electric circuit just like other flow systems [18, 19]. The inflow can originate from portal venous systems including the intrahepatic portion of the left portal vein [2, 3]. Many authors have tried to define types of PSS with different schemes [3, 5, 31]. One of the most confusing terms is “spontaneous,” because it is controversial whether a portal hypertensive PSS should be included in spontaneous PSS or not [30, 32]. It is clear from the electric circuit PSS model that there are two mechanisms underlying a PSS formation, and we suggest the PSS should be classified as portal hypertensive (increase in \( V_{PV} \)) and spontaneous (decrease in \( R_S \)), seem plausible, and published evidence supports both scenarios [2, 3].

### Table 2: The relationship between the location of shunt blockade and the expected fate of portosystemic shunt according to the cause of shunt formation.

| Cause                              | Location of blockade | Inflow | Outflow |
|------------------------------------|----------------------|--------|---------|
| Increase in portal pressure        | Collapse             | Persistent |
| Decrease in shunt resistance       | Collapse             | Collapse |

(\( \oplus \)) in Figures 1 and 2) is blocked, PSS will collapse because the pressure difference across PSS is zero. On the other hand, if the outflow (\( \oplus \)) in Figures 1 and 2) is blocked, PSS will persist because the pressure across the PSS is \( V_{PV} \). When a shunt occlusion is made within the shunt channel (\( \ominus \) in Figures 1 and 2), the PSS portion proximal to the blockade will persist, whilst that distal to the blockade will collapse. However, when a shunt was formed by the decrease in \( R_S \), the PSS would collapse after the shunt blockade. This is irrespective of the occlusion site because \( R_S \) becomes infinite.

### 3.4. Suggestions to PSS Classification.

PSS can be classified by its underlying causes. The PSS formed by the increase in \( V_{PV} \) can be classified as portal hypertensive, and the PSS formed by the decrease in \( R_S \) can be classified as spontaneous; the shunt channel was opened without the increase in \( V_{PV} \).

### 4. Discussion

PSS is defined as a condition whereby the gut venous system flows directly to a systemic vein, thus bypassing the liver [16]. The inflow can originate from portal venous systems including the intrahepatic portion of the left portal vein [2, 3]. The draining vein can be a hepatic vein, ductus venosus, an umbilical or paraumbilical vein, or other systemic veins [2, 17]. A shunt implies flow and can be simulated using an electric circuit just like other flow systems [18, 19]. The shunt flow is determined by the formula \( I_S = V_{PV}/R_S \), where \( V_{PV} \) is portal pressure or the portosystemic pressure gradient, assuming that the systemic venous pressure is \( \sim 0 \) mmHg, and \( R_S \) is shunt resistance, which is inversely proportional to the area of the shunt vessel [6]. For a PSS to form, either \( V_{PV} \) has to increase or \( R_S \) has to decrease, or both. When a PSS is formed by an increase in \( V_{PV} \) as a consequence of increased hepatic resistance \( R_L \), \( V_{PV} \) will continue to increase until collateral vessels dilate or new shunt channel appears [2]. Representative clinical conditions in which \( R_L \) is increased are liver cirrhosis and Budd-Chiari syndrome [6, 20]. An extreme case would be congenital absence of portal vein, where \( R_L = \infty \), \( I_P = 0 \), and \( I_M = I_S \) [21]. \( R_L \) and \( R_S \) are inversely related at fixed \( I_M(= I_P + I_S) \), meaning that an increase in \( R_S \) by occluding the PSS will result in the increase in \( V_{PV} \), which in turn increases \( I_P \), portal flow through the liver [22]. This can be understood by the same mechanism as the formation of a PSS, but in the reverse direction. Alternatively, for \( R_S \) to decrease, either shunt vascular diameter must be increased or multiple shunt channels must be opened [23]. \( R_S \) can decrease until \( I_S = I_M \), with resultant total steal of portal flow though the shunt (\( I_P = 0 \)). Congenital PSS with or without an aneurysm is a representative clinical condition [24, 25]. Whatever the initiating event may be, either the increase in \( V_{PV} \) or decrease in \( R_S \), once the shunt flow is established the shunt channel can be dilated and even form an aneurysm according to Laplace’s law [26].

The electric circuit PSS model can be used to interpret other clinical conditions. For example, we had assumed that the mesenteric vascular resistance \( R_M \) was constant. However, there are diseases in which \( R_M \) is decreased, such as mesenteric arteriovenous malformation or fistula. Being a pressure divider with \( R_S \) and \( R_L \), the decrease in \( R_M \) has the same effect as the increase in \( R_L \), and portal hypertension ensues [27, 28].

Unfortunately, the evolution of a PSS after blockade was not always available in the literature. Two cases have been issued on intrahepatic PSS managed by outflow occlusion, both of which reported the disappearance of PSS [7, 13]. The patients had no liver cirrhosis. On the other hand, one patient who had extraportal PSS and liver cirrhosis was managed by outflow occlusion; PSS persisted [14]. Another patient without portal hypertension had patent ductus venosus, and the shunt thrombosed but did not collapse after shunt blockade, probably because the anomaly persisted even when the shunt was blocked [15]. These findings support the notion that intrahepatic PSS occurs in patients without portal hypertension and that it can be congenital or spontaneous in origin, whereas extraportal PSS develops as a consequence of portal hypertension [2, 29]. Even in patients who have portal hypertension and intrahepatic PSS together, one condition may provoke the other, because the probability of them to occur simultaneously is low [30]. Also, the reported cases comply with our inference that the cause of a PSS can be deduced after outflow occlusion. At present, both proposed scenarios pertaining to the cause of PSS formation, namely pressure-first (increase in \( V_{PV} \)) and shunt-first (decrease in \( R_S \)), seem plausible, and published evidence supports both scenarios [2, 3].

5. Conclusions

By simulating PSS using an electric circuit, we found that similarities between the two “flow” systems provide valuable insight to the mechanisms underlying PSS formation. The simulation is simple, easy to understand, and readily applicable to various clinical situations which are seemingly...
complicated. The shunt blockade site should be selected according to the cause of the PSS because serious complications can occur. Further clinical experiences are required to refine the PSS classification scheme.

**Competing Interests**

The authors declare that there is no conflict of interests regarding the publication of this paper.

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