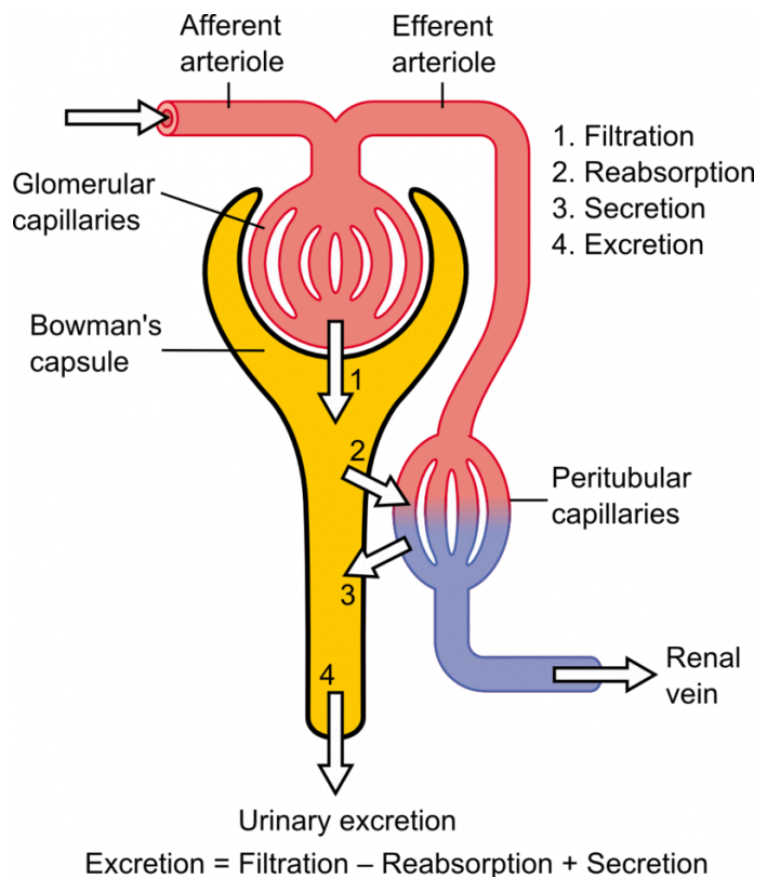


## 7.2.1

# Filtration

As we discussed as we studied the anatomy of the renal corpuscle in the previous section, filtration is the first step in urine formation. Blood enters the kidneys to be filtered. Blood travels to the afferent arterioles and enters into the glomerulus where it can be filtered through the fenestrated endothelium, the basement membrane and the visceral layer and podocytes. The filtrate then spills into Bowman's capsule and from there is taken on its journey through the rest of the nephron.



Author: By Madhero88 (Own work) Link: <https://books.byui.edu/-UHwZ>

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## Glomerular Filtration Rate (GFR) and Net Filtration Pressure (NFP)

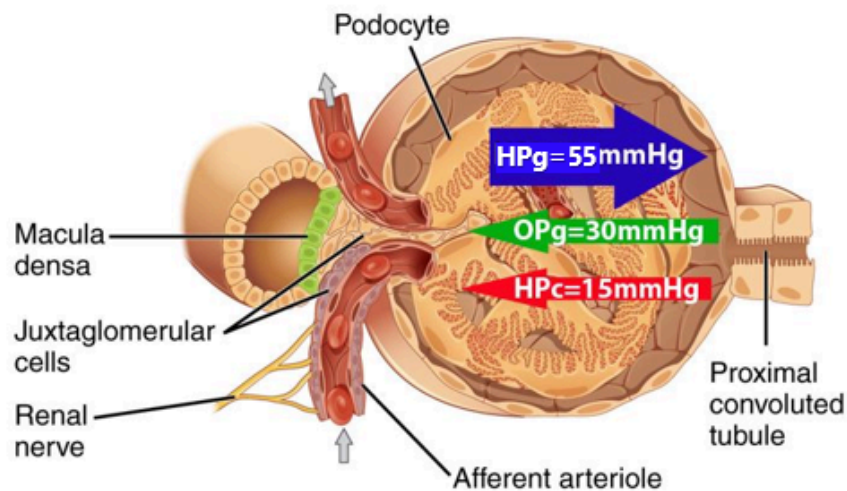
Although the filtration barrier is very important for the selection of substances, filtration would not occur without pressure. To move from the glomerulus to Bowman's capsule, pressure must be higher in the glomerulus than in Bowman's capsule.

The kidneys put special emphasis on glomerular pressure. The rate at which the kidneys filter blood is called the **glomerular filtration rate (GFR)**. The normal GFR is around 125 ml plasma/min. If systemic pressure were to drop suddenly, the glomerular pressure would also drop, resulting in a decrease in the **glomerular filtration rate, which would** result in imbalances in glomerular filtration. To compensate for the drop in blood pressure, the **afferent** arteriole can dilate, allowing more blood to enter the glomerulus and thus maintaining the pressure. An alternative method would be to constrict the **efferent** arteriole, causing a "back-up" in blood and increase in the pressure. In contrast, if the systemic blood pressure were to increase, the afferent arteriole would be stimulated to constrict, reducing blood flow, or the efferent arteriole could dilate. These mechanisms of constriction or dilation are part of a regulatory system called **autoregulation**.

Not only is filtration pressure regulated by the afferent and efferent arterioles, but also from the forces within the glomerulus. Just like we learned when studying capillary exchange in the blood, there are hydrostatic and osmotic pressures at work in the glomerulus. The main *outward pressure* forcing blood through the filters of the glomerulus is the **Hydrostatic Pressure of the glomerulus (HPg)** created by the blood flow regulated by the afferent and efferent arterioles of the glomerular capillaries. The HPg is carefully regulated between 50 to 55 mmHg. This outward pressure is resisted or counteracted by the *inward pressure* from the hydrostatic pressure of the fluid already within the nephron or **Hydrostatic Pressure of Bowman's capsule (HPc)** which is typically 15 mmHg. Another *inward* force drawing fluid back into the blood is created by the proteins that remain in the capillaries, or blood colloidal osmotic pressure, also known as the **Oncotic Pressure of the glomerular capillaries (OPg)** which is roughly 30 mmHg. The **Net Filtration Pressure (NFP)** is the outward pressure minus the inward pressure.

**Net Filtration Pressure** = Hydrostatic Pressure of glomerulus (HPg) minus the sum of Hydrostatic Pressure of the capsule (HPc) and the Oncotic Pressure of the glomerular capillaries (OPg).

$$\text{NFP} = 55 \text{ mmHg} - (30\text{mmHg} + 15\text{mmHg}) = 10 \text{ mmHg}$$



HPg = (Hydrostatic Pressure of the Glomerular Capillaries)

OPg = (Oncotic Pressure of the Glomerular Capillaries)

HPc = (Hydrostatic Pressure of the Capsule)

**NFP=Net filtration pressure**

**NFP=all outward pressures - all inward pressures**

**NFP=HPg - (OPg + HPc)**

**NFP= 55 - (15 + 30)**

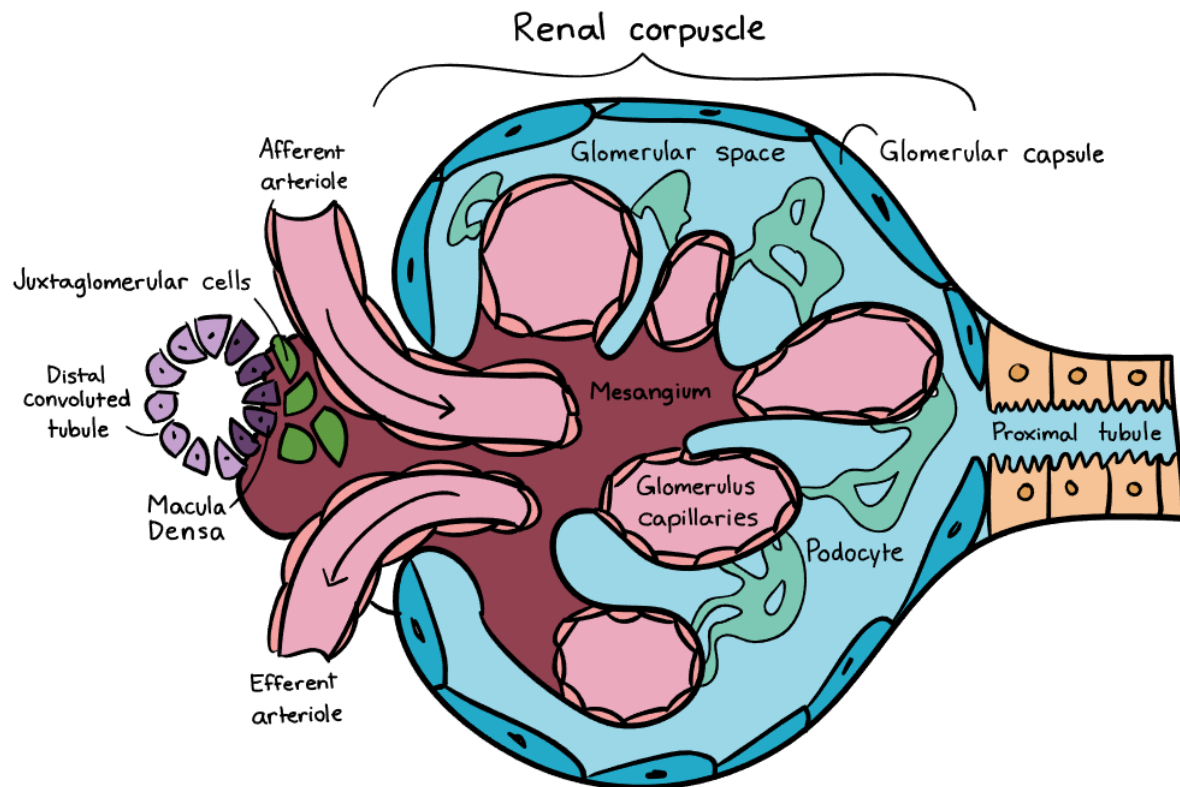
**NFP= 10 mmhg**

**Net Filtration Pressure.**

Adapted from Title: File: Juxtaglomerular Apparatus and Glomerulus.jpg; Author: OpenStax College;  
<https://books.byui.edu/-hGnz>;

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Most capillary beds in the body have a maximum pressure of about 25mmHg under normal conditions, but the kidney's glomerular capillaries have a pressure of about 55mmHg. This high pressure is necessary for filtration to occur but also presents some interesting problems that the kidney must compensate for. Since capillaries are very thin walled, they are not designed to withstand high pressure for long periods of time. To help compensate, the renal corpuscle contains a population of cells called **mesangial** cells.



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 <https://ka-perseus-images.s3.amazonaws.com/f001c2f927f6d65690a1b3c6812c2d596d5e7e76.svg>

### Diagram of the Renal Corpuscle Structure

File: Renal corpuscle.svg; Author: Khan Academy Site: <https://books.byui.edu/-xgcP>

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Similar to the podocyte cells, the mesangial cells surround the glomerular capillaries and provide additional structural support and may even have some role in regulating filtration. These cells are also phagocytic and may serve important immune type functions. Because the pressure in the glomerulus is so important for filtration, the kidneys go to great lengths to ensure that the pressure stays as close to 55mmHg as possible.

## Regulation of GFR

How do the kidneys "know" when to constrict or dilate the arterioles to maintain GFR? The kidney nephrons have the ability to measure the amount of NaCl in the filtrate. If the NaCl concentration of the filtrate begins to increase, specialized cells in the nephron distal tubule called **macula densa** cells (shown in previous images of Bowman's capsule and the renal corpuscle) will detect the increase, and release paracrine signals that will cause the afferent arteriole to constrict. This constriction decreases the GFR and reduces the amount of NaCl entering the nephron. This is

an example of negative feedback. The cells can also respond to a decrease in the NaCl by causing a dilation of the afferent arteriole. This mechanism can occur because of the close association of the macula densa cells with the afferent arteriole. Together they form a structure known as the **juxtaglomerular apparatus**.

High blood pressure in the glomerulus can also be compensated by the intrinsic properties of smooth muscle. Smooth muscle cells constrict in response to stretch. Thus, an increase in blood pressure (increase in stretch) will cause the smooth muscle cells in the afferent arteriole wall to constrict, thus reducing the amount of blood and associated pressure increase. Afferent arterioles also have baroreceptors that respond to stretch and cause the release of renin from the juxtaglomerular cells (discussed later). In addition to the auto regulatory mechanisms, the kidneys can rely on input from sympathetic nerve fibers which innervate both the afferent and the efferent arterioles.



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