Direct Arterial Blood Pressure Monitoring
Angel M. Rivera CVT, VTS (ECC)
Animal Emergency Center
Glendale, WI
March 2003

Introduction

Direct measurement of arterial blood pressure is obtained via a peripheral artery catheter that is connected to a transducer and is a continuous display/recording device or monitor. Intra-arterial or direct blood pressure provides a continuous display of the arterial waveform along with measurements of mean arterial pressure, systolic and diastolic pressure.

Monitoring direct arterial pulse pressure provides valuable information in terms of pressure value or measurement, but it also provides valuable information in terms of its shape and contour. The arterial pulse wave can exhibit changes that are related to specific underlying pathology.

Indications for the use of direct arterial blood pressure monitoring include shock, critical illness, peripheral vasoconstriction, and intra-operative and post-operative monitoring of high-risk patients. In addition, arterial catheters allow for frequent monitoring of blood gas measurements.

Basic Physiology

Arterial blood pressure is generated by the ejection of blood from the left ventricle (LV) into the arterial vasculature. The amount of pressure generated is determined by the volume of blood ejected as well as the resistance to ejection within the systemic vascular network. In other words:

PRESSURE= Flow X Resistance

As blood is ejected into the proximal portion of the aorta, the vessel wall stretches and distends to accommodate the increase in volume. This stretching of the aortic segment is transmitted peripherally along adjacent segments of the aorta, thus producing a pulse wave that is transmitted through the arterial circulation and felt peripherally as a pulse.

The rate at which the pulse travels down the aorta is determined by the compliance and distensibility (vasodilation) of the arterial system. Decreases in compliance (hypertension or vasoconstriction) results in a rapid transmission of pulse wave. As the pulse travels peripherally from the central aorta, it changes shape as well as value. This occurs as a result of the reflected waves that summate on the primary systolic wave.

The arterial pressure wave is divided into two phases that correspond to the cardiac cycle: systole and diastole. Arterial systole begins with the opening of the aortic valve and the rapid ejection of blood into the aorta. This is followed by "run-off" of blood from the proximal aorta to the peripheral arteries. On the arterial pressure waveform, this appears as a sharp rise in pressure followed by a decline in pressure. As pressure falls, the aortic valve snaps shut, causing a small change in arterial pressure that appears as a dip on the down slope. This is

termed the "incisure" in central aortic pressures and the "dicrotic notch" in peripheral pressures. This marks the end of the ejection period.

The "*peak systolic pressure*" is a reflection of maximum pressure generated by the LV and is normally 100-140 mmHg. Systolic pressure is frequently used to monitor ventricular "afterload". Afterload is defined as LV wall tension during systole. The two principal determinants are systolic pressure and the radius of the left ventricle (related to LV volume) is assumed to remain relatively constant, and therefore systolic pressure is the parameter used to clinically monitor after loads.

Diastole follows closure of the aortic valve and continues until next systole. During this time, "run-off" to the peripheral arteries occurs without further flow from the LV. On the peripheral arterial waveform this is seen as a gradual decrease in pressure. The lowest point on diastole (end-diastole) is referred to as arterial diastolic pressure and is normally 60-80mm Hg.

The diastolic arterial blood pressure reflects both the velocity of "run-off" and the elasticity of the arterial system. The elastic properties of the vessels affects the arterial system's ability to change luminal dimensions as blood volumes change. Heart rate also affects the diastolic pressure because it determines the duration of diastole in the cardiac cycle. The longer the period of time and the shorter the diastolic period (with tachycardia), the higher the diastolic pressure.

Diastolic pressure is also important in determining coronary perfusion, especially to the left ventricle, where most flow occurs during diastole. Normal coronary artery perfusion pressure is 60 to 80 mm Hg. Perfusion pressures below 50 mmHg threaten myocardial perfusion.

The arterial pressure differs in both contour and value depending on location of the artery. The systolic pressure tends to be higher in the femoral artery than in the radial or brachial artery by as much as 25 to 50 mmHg. Generally, the diastolic and mean arterial pressure (MAP) remains nearly the same.

Normal arterial blood pressure is approximately 120/80 mmHg in the healthy dog and cat. Systolic pressure greater than 180mmHg and diastolic pressures greater than 120mmHg are suggestive of hypertension. MAP is defined as the sum of diastolic pressure plus one third of the pulse pressure. It can also be mathematically calculated as:

$$MAP = \frac{Systolic\ pressure + (Diastolic\ pressure \times 2)}{3}$$

MAP represents the average pressure within the arterial system. Since diastole typically lasts approximately two thirds of the entire cardiac cycle, the mean arterial pressure value is closer to diastolic value than to the systolic value. Even though, the arterial pressure pulse is amplified as it travel away from the heart, the MAP remains relatively unchanged, thus its importance as a reflection of overall perfusion. Normal MAP is 70-90 mm Hg. MAP dependent on two factors:

1. Cardiac Output (CO): The blood flow through the vessel.
2. Systemic Vascular Resistance (SVR): The elasticity of resistance of the vessels.

The above interrelationship can be expressed as:

MAP= Cardiac output X Systemic vascular resistance.

The pulse is the difference between the systolic and diastolic pressure. In other words:

Pulse Pressure = Systolic Pressure - Diastolic pressure

The pulse pressure is largely determined by stroke volume (amount of blood ejected with each contraction), the compliance or distensibility of the arterial network and the character of ejection from the heart during systole. Thus, it is said that pulse pressure is reflective of stroke volume and arterial compliance. Any factor that increases stroke volume, such as decrease in pulse rate or an increase in CO, also increases pulse pressure. A decrease in compliance or distensibility of the arterial network also tends to increase pulse pressure. In the emergency and critically ill patient, wide pulse pressures are associated with large stroke volumes, and narrow pulse pressures are seen with low stroke volume.

The pulse pressure and not the systolic blood pressure largely determine the strength of a pulse on palpation. A weak peripheral pulse is generally associated with a stroke volume that is lower than normal. This could be due to a rapid pulse rate or decreased cardiac output. However, it is important to remember that, weak peripheral pulses at the extremities can also be due to arteries and arterioles severely constricted.

Pulse pressures changes reflect alterations in cardiac output much more accurately than systolic blood pressure as shock develops. In hypovolemic shock, major decreases in stroke volume and pulse pressure as often occur long before there is significant fall in the systolic pressure. Diastolic pressure often rises initially with hemorrhage due to sympathadrenal stimulation, particularly if there is pain or tissue damage. Since vasoconstriction can increase only to a certain maximum, continued blood loss will eventually result in a fall in both systolic and diastolic pressures.

Therefore, in an individual patient, changes in pulse pressure can often correlate quite well with changes in stroke volume. An example of this can be of a patient's blood pressure changing from 120/80 to 110/90. In this particular case, the pulse pressure has fallen from 40 mm Hg to 20 mm Hg. A 50% decrease in pulse pressure, could also signify a 50% decrease in stroke volume.

Intra-arterial or direct arterial pressure measurement

The three components or characteristics of intravascular pressures measured through a fluid- filled catheter are:

- Residual or static pressure inside the fluid-filled vessel
- Dynamic pressure, which is caused by the imparted kinetic energy of the moving fluid (similar to the encountered in arterial pressures when the catheter tip is directly facing the flow of fluid).
- Hydrostatic pressure, which results from the difference between the ends of the fluid-filled tubes) the tip of the catheter and the air-reference port of the transducer).

The most desired measurement during hemodynamic monitoring is residual or static, pressure within the vessel or chamber being assessed. To achieve this goal, both dynamic and hydrostatic pressure components need to be eliminated. As stated above, dynamic pressures are encountered only in high or frequent flow rates or when the catheter tip is directed into the flow. This component is greatly reduced when assessing arterial blood pressure by the use of dampening services.

The hydrostatic pressure is the component to most consistently interfere with the accuracy of pressure measurements obtained through fluid-filled systems. Placement of the air-reference port at the level of the right atrium eliminates the effects of hydrostatic pressure within the fluid-filled column and provides accurate measurement within the peripheral artery.

All biomedical devices consist of three basic components:
1. Transducer or device that detects the physiological event.
2. An amplifier that increases the magnitude of the signal from the transducer.
3. A recorder, meter, oscilloscope or monitor to display the signal.

TRANSDUCERS

The measurement of pressures requires the transformation of a biophysical event into an electrical event or signal, which can then be displayed, recorded and quantified. This transformation is done with the transducer.

Depending on its application, transducers can sense changes in flow, color, temperature, concentration, pressure, displacement, light intensity, frequency, sound and other physiological changes. They are classified according to the type of energy to which they are sensitive and the method of energy coupling used. The pressure transducer most commonly used in biomedical instrumentation is the mechanical or displacement. Mechanical or displacement transducers consist of a mechanical element that is displaced as a result of changes in pressure. The most commonly used type of displacement transducer is the strain gauge transducer.

Strain gauge transducers are sensitive to temperature changes (typically 0.1 mm Hg/1° C) and require occasional checking of the zero-pressure baseline for drift. The room in which they are being used should be maintained at a fairly constant temperature. Solutions flushed through the transducers can also cause temperature variance and baseline drifts to occur.

AMPLIFIERS

Amplifiers are used to boost the size of a signal by a factor of 1,000 before it can be displayed. They can be straightforward electronic devices that simply amplify the signal from the transducer, or they can be highly complex devices that process, filter and amplify the signal to display on the oscilloscope and record. The signal can be fed into a computer for further digital processing and analysis.

Amplifiers should filter out other physiological signals and undesired environmental noise detected by the transducer. The amplifier's ability to increase the signal level and its accuracy in reproducing signals from input to output should not add significant distortion (termed linear response). Calibration, stability, and temperature drifts should minimally affect the signal.

ZERO DIFFERENCE

The weight of air or atmospheric pressure must be eliminated from pressure measurements dependent on altitude and environmental factors. Atmospheric pressure is about 760 mm Hg at standard temperature and pressure at sea level. This is achieved by opening the transducer to air, and adjusting the display system to read zero. By doing so, all pressure contributions from the atmosphere are negated, and only pressure values that exist in the vessel will be measured.

CALIBRATION

Accurate quantitation of recorded pressure measurements requires precise calibration of both transducer and the amplifying electrical components. Most monitors have a built-in electrical calibration that yields a known pressure value. This calibration should be checked before hemodynamic monitoring is initiated. To avoid electronic drifts, the instrument should be allowed to warm up for approximately 15 minutes.
Calibration steps are:

1. Depress the "CAL" button on the monitor.
2. After the readout stabilizes, use the control knob to adjust and obtain the appropriate readout value (e.g.160, 200mm Hg).
3. Release the "CAL' button and make sure the reading returns to zero.

NOTE: This procedure assesses the electrical calibration of the system. It DOES NOT check the calibration of the transducer.

TRANSDUCER CALIBRATION

All disposable transducers are pre-calibrated and coded by the manufacturer and are considered to be accurate at the time of use. However, as with any sensitive instrument they can be easily damaged. The calibration of the transducer can be checked in the following manner:

1. Attach a 3 or 4 foot long extension tubing to the side port of an in-line stopcock.
2. Turn the stopcock open between the infusion bag and the attached extension tubing and completely fill the tubing with fluid.
3. Vertically hold or mount the fluid filled extension tubing above the transducer.
4. Turn the stopcock open between the fluid filled extension tubing and the transducer.
5. Check the digital readout on the monitor.
   a. A 3-foot fluid filled tubing should exert a pressure of 67mm Hg (+/-1).
   b. A 4-foot fluid filled tubing should exert a pressure of 90mm Hg (+/-1).

Equipment
1. Catheter of choice.
2. Pressure transducer.
3. Electronic monitor or oscilloscope.
4. Plastic IV heparinized fluid bag.
5. Pediatric IV.
6. Three way stopcocks.
7. Continuous flush device.

8.   Pressurized IV cuff or pump.

Equipment Set Up

1.   Plug transducer cable into the monitor.
2.   Select appropriate scale on monitor.
3.   Turn monitor power switch on.
4.   Add heparin 1 unit per each ml of fluid. And label bag.
       • Using a sterile needle, remove all air from the IV heparin flush bag via the
          medication port.
5.   Connect IV pediatric set to heparin flush bag.
       • Lightly squeeze the drip chamber.
       • DO NOT fill chamber more than 0.5 cm, since the fluid level will increase when pressure
          is applied to the bag.
       • DO NOT prime the IV tubing at this point.
6.   Close the IV line roller clamp, insert the IV bag into the pressure administration
       cuff, and hang the pressure cuff.
       • DO NOT pressurize bag at this point.
7.   Replace all caps on the stopcock with injection caps on the sideports of all
       stopcocks.
8.   Attach the IV set to the designated end of the continuous flush device.
9.   Attach a venting stopcock to one port of the transducer. (NOTE: Some transducer may
       come preassembled).
10.   Attach a luer LUER LOCK stopcock the remaining transducer dome (if applicable).
11. Attach the female end of the continuous flush device to the LUER LOCK stopcock on the
       transducer dome.
12. Open the transducer- venting stopcock to air.
13. Open the IV roller clamp.
       • Pull the fast flush device and allow the IV solution to fill the tubing, the
          stopcocks, the flushing device and the transducer dome.
       • NOTE: It may be necessary to rotate the transducer dome while fasting-flushing to
          purge all air bubbles from dome.
14. Close the transducer-venting stopcock.
15. Remove dead-ender cap at patient end of the connecting tubing.
       • Attach a LUER-Lock stopcock (if not pre-assembled) and close off all its side ports.
16. Pull fast flush device and slowly flush entire monitoring line (using only gravity).
       • Take special care that no air is trapped in any stopcocks.
17. Pressurize cuff on IV flush bag to 300 mm Hg.
       • Make sure drip chamber DOES NOT completely fill.
       • Set drip rate between 1 to 5 mls/hr.
18. Allow catheter hub to fill with blood.
       • While holding the fast flush device open, connect IV line to the catheter,
       • Reassess drip rate to to ensure a rate of 1 to 5 mls/hr.
19. Check all connecting sites.
20. Place transducer air- reference stopcock at level to patients thoratic inlet and secure.
       • This positioning should be approximate to the level of the right atrium.
21. Open the air reference stopcock to air.
22. Turn the ZERO control knob on the monitor to obtain a zero reading.

23. Push the calibration knob and recheck the monitor calibration.

- Adjust if necessary

24. Close the transducer-venting stopcock to air.