A SYSTEM FOR DETERMINATION OF BLOOD PRESSURES AND HEMODYNAMICS FROM OSCILLOMETRIC WAVEFORMS

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Abstract: An experimental system (Carditor) for noninvasive determination of blood pressures and hemodynamics is presented. The system consists of a wrist cuff, a battery powered module with pneumatic and electronic circuits, and a notebook computer. The computer controls cuff inflation, deflation and data acquisition functions during gradual cuff deflation. Proprietary software is used to analyze oscillometric waveforms obtained from radial artery. Systolic, mean and diastolic blood pressurers and stroke volume are computed first. Stroke volume is then adjusted for body area and the adjusted value is used to compute cardiac output. Total peripheral resistance and systemic arterial compliance are computed from arterial pressures, cardiac output and stroke volume. The computed hemodynamic variables are displayed on the computer screen in a "quadrant" graphic form. Preliminary hemodynamic data from from a group of 41 men and women (age 17-76) were used for qualitative comparison with published data (deSimone, n=544). Carditor results: stroke volume=76 ml, heart rate=70 bpm, cardiac output=5.3 l/min. DeSimone results: stroke volume=81 ml, heart rate=68 bpm, cardiac output=5.5 l/min. Examples of test results and clinical applications of the system are discussed.

Introduction

Cardiovascular disease (CVD) is the leading cause of death in many countries. An important contributor to CVD is hypertension (HTN) [1]. It is now the most frequent reason for visiting a physician. The incidence and prevalence of HTN is very high despite efforts focused on its detection, evaluation and treatment [2]. Clinical hypertension can be characterized [3] by an elevated mean arterial pressure (MAP), which is equal to the product of cardiac output (CO) and total peripheral resistance (TPR).

$MAP=CO$. TPR/80 [mmHg, $l.min^{-1}$,dyn.sec.cm⁻⁵] (1)

Elevation of MAP can be caused either by increased CO or by increased TPR. This relationship relates to the steady phenomena and it does not take into account the fact that blood pressure fluctuates about the MAP during the cardiac cycle [4]. More complete approach takes into account systemic arterial compliance (SAC) [5]. SAC influences systolic pressure (SBP), diastolic pressure (DBP) and pulse pressure (PP):

$$
PP = SBP - DBP \qquad [mmHg] \tag{2}
$$

In the arterial system with decreased SAC, PP is higher for the same stroke volume (SV). The importance of arterial compliance has been recognized and its surrogate PP has been found [6] to be a significant risk factor of mortality in older people. The described hemodynamic variables determine SBP, DBP and MAP. The most common essential HTN is characterized by increased TPR. When CO is increased to abnormally high level while TPR stays the same, the result is hyperkinetic HTN Hyperkinetic HTN occurs mostly, but not exclusively, in younger individuals. Isolated systolic hypertension (ISH) is characterized by increased SBP and normal or decreased DBP due to decreased SAC and increased TPR [7]. ISH occurs mostly in older individuals.

The types of HTN described above make diagnosis and treatment difficult without knowledge of underlying hemodynamics. Improved treatment of HTN assisted by noninvasively obtained hemodynamics using bioimpedance has been recently described by several investigators [8, 9, 10].

An inexpensive system capable of noninvasive determination of BP and hemodynamics could help to improve diagnosis and treatment of HTN.

Most noninvasive blood pressure monitors use oscillometric methods to determine systolic (SBP), diastolic (DBP) and mean (MAP) arterial pressures [11]. The system described in the present paper determines BPs and hemodynamics from oscillometric waveforms (OMWs) during gradual cuff deflation.

Materials and methods

The system (Carditor, Carditech, Culver City, CA, USA) consists of a battery powered module, a wrist cuff, and a notebook computer. The module contains pneumatic and electronic components necessary to inflate and deflate the cuff, to perform analog and digital functions, and to send formatted data to the notebook computer. Block diagram of the system is shown in Figure 2.

Figure 1: The system with cuff, module and notebook

The notebook computer controls system functions via microcontroller (87C51). The microcontroller controls pneumatic functions and analog-to-digital conversion of cuff pressure (CP) and OMW data. The operation starts with cuff inflation to about 30 mmHg above the expected SBP. The valve is closed during cuff inflation. Slow deflation of the cuff from supersystolic to supradiastolic CP is controlled by the valve in a linear fashion. When the CP reached predetermined supradiastolic value, the valve quickly deflates the cuff to zero pressure.

Figure 2: Block diagram of the system.

The valve is controlled by a digital-to-current converter and the control current is adjusted in 300 mS intervals. CP is converted to analog voltage by a piezoresistive pressure sensor with a range 0 - 300 mmHg. The analog voltage is amplified by an instrumentation amplifier and filtered by a band-pass filter (0.5 - 35 Hz). A 2 channel 12-bit serial A/D converter (MAX1247) is used to digitize CP and OMW. Sampling rate is 11.8 mS. Digitized data is transmitted to the computer via RS232C serial interface.

Blood pressures and hemodynamics are computed by proprietary software developed at Carditech. Oscillometric MAP is computed first. Mean pressure in the cuff corresponds to the largest amplitude of pressure pulses [11]. Heart rate (HR) is computed from peak -to peak intervals between individual OMWs. SBP and DBP are computed next. Algorithmic procedure based on the rate of change of pulse waveform amplitudes is used. The acquired OMWs are analyzed to obtain stroke volume (SV). The computed SV is adjusted for body surface area (BSA). Height, weight and age of the tested subject are manually entered.

 $BSA = (weight + height - 60)/100 [m^2, kg, cm]$ (3)

Cardiac output (CO) is obtained by multiplying SV by HR.

$$
CO = SV . HR [1.min-1, ml, beats.min-1] (4)
$$

Total peripheral resistance (TPR) is obtained by dividing MAP by CO.

 $TPR = 80$. MAP/CO $\left[\text{dyn/sec.cm}^{-5}, \text{mmHg, l.min}^{-1}\right]$ (5)

An estimation of systemic arterial compliance (SAC) [5] is computed by dividing SV by pulse pressure (SBP - DBP).

$$
SAC = SV / PP [ml.mmHg-1, ml, mmHg]
$$
 (6)

The computed blood pressure and hemodynamic variables are displayed on the computer screen as numeric values and as a "quadrant" graphic format (Figure 3). The quadrant shows the relationships of CO, TPR and SAC. TPR and SAC are graphically represented by small rectangles that move together on the vertical axis according to the value of CO. TPR and SAC rectangles are positioned on the horizontal axis according to their values. Left ventricular ejection time (LVET) is presented only numerically because it does not have a direct relationship to the other hemodynamic variables.

Figure 3: Test results of a normotensive individual

The values of hemodynamic variables displayed in Figure 3 are typical normal values. Normal SV values are usually between 60 and 90 ml and CO values between 4 and 7 liters per minute. TPR value for of 1319 dyn is also normal for corresponding MAP and CO. The value of 2.1 ml for SAC indicates good arterial compliance [5]. The values of TPR and SAC are displayed graphically in the right-hand "normal" side of the quadrant.

Hemodynamics of test results displayed in Figure 4 are abnormal. The SBP and DBP values indicate moderate essential HTN. Essential HTN is characterized mainly by increased TPR. The TPR value of 1742 dyn is increased mainly because of increased MAP. Normal value of SAC indicates that the hemodynamic variables

are most likely those of a younger individual.

Normal value of CO (5.7 l.min^{-1}) indicates that the main source of pressure increase is the TPR.

Hypertension characterized by high TPR and low SAC is usually found in older individuals. Test results of a 76 years old woman are in Figure 5.

Figure 4: Test results of a 45 years old hypertensive individual

TPR value of 2173 dyn and SAC value of 1ml are a clear indication of HTN with abnormally low arterial compliance.

Figure 5: Test results of an elderly hypertensive individual.

Figure 6: Test results of a normotensive pregnant woman.

Normal pregnancy is characterized by 30 - 40 % increase of CO and a decrease in TPR [12]. The increase of CO is caused by increased HR and SV. Many pregnant women also have decreased BP. Figure 6 shows test results of a normotensive pregnant woman (gestational age=36 weeks). When compared with test results of a normotensive, nonpregnat individual in Figure 3, the CO is clearly increased and the TPR is decreased.

Last weeks of a hypertensive pregnancy is usually characterized by CO lower than that of normal pregnancy and TPR is increased [13]. Figure 7 shows test results of a hypertensive woman (gestational age=37 weeks). CO is abnormally low and TPR is abnormally high. The pregnancy resulted in a low birth=weight baby that was probably caused by decreased CO.

Figure 7: Test results of a hypertensive pregnant woman.

Obesity-induced HTN is increasingly common. It is hemodynamically characterized by hyperdynamic circulation (increased SV, HR, CO) and increased blood volume [14]. Test results of obesity-induced mild HTN are in Figure 8.

Figure 8: Test results of of an obese woman.

CO is elevated due to increased HR and SV. TPR is decreased as compared to a typical value in HTN. This decrease is likely caused by increased CO. Systemic arterial compliance (SAC) is normal. High SAC and high SV both indicate increased blood volume.

The woman whose test results are shown in Figure 8 lost weight in excess of 60 kg. Test results after the weight loss are in Figure 9. HR, SV and CO are decreased and this decrease in CO caused blood pressures to return to normal values. TPR increased to the value expected for the measured value of MAP and CO.

Figure 9: Test results after weight loss of 60 kg.

Comparative results

Data from the system's developmental database were used to compute and compare hemodynamic values estimated by the system with values obtained from a study conducted by de Simone et al [15]. Data from a group of 41 male and female volunteers (age 17 - 76) were computed. The comparative values are displayed in table 1.

Table 1. Comparison of hemodynamic variables

Discussion

The system's noninvasive nature and ease of use makes it well suited for clinical situations where monitoring of blood pressures and hemodynamic variables is desirable. Examples presented in this paper were related to HTN but congestive heart failure, post myocardial infarction, kidney failure, and high risk pregnancy could benefit from the convenience of this simple, inexpensive test. Although the use of hemodynamic measurements in HTN is not new [16], application in diagnosis and management of HTN has been limited by a lack of simple, noninvasive methods.

The bioimpedance method requires application of 8 electrodes to the torso and the neck. Blood pressures must be measured separately and the values entered manually. The needs to apply electrodes and relatively high costs of a bioimpedance monitor and a BP monitor have prevented widespread use of hemodynamics for management of HTN.

The results of published studies [8, 9, 10] show that combined knowledge of BPs and hemodynamics could guide antihypertensive therapy more effectively than clinical judgment alone.

The data used in examples and in Table 1 are presented for qualitative comparison only. A quantitative comparative study with an established method is being prepared. Further software development may be necessary to establish accuracy of BP and SV determination.

Conclusion

The described experimental system could be useful in many applications where other methods of measuring hemodynamics may not be justified in terms of cost and complexity of operation.

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References

- [1] LENFANT, C. (1996): 'High Blood Pressure: Some Answers, New questions, Continuing Challenges', *JAMA,* **275**, pp. 1604.
- [2] PERRY, H. M. ET AL. (1992): 'Difficulties in diagnosing hypertension: Implications and alternatives', *J. Hypertension,* **10**, pp. 887-96
- [3] LARAGH, J.H., BRENNER, B.M. (1995): 'Hypertension: Pathophysiology, Diagnosis and Management', (Raven Press).
- [4] SAFAR, M.E. ET AL. (1996): 'Artertial alterations in hypertension with disproportionate increase in systolic over diastolic blood pressure', *J Hypertension,* **14**, pp. 78-79.
- [5] DE SIMONE, M.J. ET AL. (1999): 'Stroke Volume/Pulse Pressure Ratio And Cardiovascular Risk in Arterial Hypertension', *Hypertension,* **33**, pp. 800-05
- [6] GLYNN, R.J. ET AL. (2000): 'Pulse Pressure and Mortality in Older People', Arch Intern Med., **160**, pp. 276-572.
- [7] BERGER, D.S. (1990): 'Concurrent Compliance Reduction and Increased Peripheral Resistance In the Manifestation of Isolated Systolic Hypertension', *Am J. Cardiology,* **65**, pp. 67-71.
- [8] TALER S.J. ET AL., (2002): 'Resistant Hypertension: Comparing Hemodynamic Management to Specialist Care', *Hypertension,* **39**, pp. 982-88.
- [9] VENTURA H.O, TALER S.J, STROBEK J.E. (2005): 'Hypertension as a Hemodynamic Disease: The Role of Impedance Cardiography in Diagnostic, Prognostic, and Therapeutic Decision Making', *Amer. Jour. Heart*, **18**, pp. 26-43.
- [10] SANFORD T, TREISTER N, PETERS C. (2005): ' Use of Noninvasive Hemodynamics in Hypertension Management', *Amer. Jour. Heart*, **18**, pp. 87-91.
- [11] JILEK J, FUKUSHIMA T. (2005): 'Oscillometric Blood Pressure Measurement: The Methodology, Some Observations, and Suggestions', *Biomed Instr & Technol,* **39**, pp. 237-41.
- [12] MABIE W.C, DISESSA T.G, CROCKER L.G, SIBAI B.M, ARHEART K.L. (1994): 'A longitudinal study of cardiac output in normal human pregnancy', *Am. Journal of Obstet Gynecol*, **170**,

pp. 849-56.

- [13] NISELL H., LUNEL N., LINDE B. (1988): 'Maternal Hrmodynamics and Impaired Fetal Growth in Pregnancy - Induced Hypertension', Obstet Gynecol, **71**, pp. 163-66.
- [14] ROCCHINI A. P. (1992): 'Cardiovascular Regulation in Obesity-Induced Hypertension', *Hypertension*, **19**, Suppl. I- pp. I56-I61.
- [15] DESIMONE G, ET AL. (1997): 'Stroke Volume and Cardiac Output in Normotensive Children and Adults', *Circulation*, **95**, pp. 1837-43.
- [16] TARAZI R. C. (1983): 'The hemodynamics of hypertension, Physiopathology and Treatment', (McGraw -Hill, New York), pp. 15-42.