Published in final edited form as:

Proc SPIE Int Soc Opt Eng. 2016 February ; 9708: . doi:10.1117/12.2212803.

Instrument to detect syncope and the onset of shock

Daniel R. McAdams^a, Noah J. Kolodziejewski^a, Christopher J. Stapels^a, Daniel E. Fernandez^a, Matthew J. Podolsky^a, Dana Farkas^a, James F. Christian^a, Michael J. Joyner^b, Christopher P. Johnson^b, and Norman A. Paradis^c

^aRadiation Monitoring Devices, Inc. 44 Hunt St., Watertown, MA 02472

^bDepartment of Anesthesiology, Mayo Clinic, 200 First St. SW, Rochester, MN 55905

^cSection of Emergency Medicine, Dartmouth-Hitchcock Medical Center, Lebanon, NH 03756

Abstract

Currently the diagnosis of hemorrhagic shock is essentially clinical, relying on the expertise of nurses and doctors. One of the first measurable physiological changes that marks the onset of hemorrhagic shock is a decrease in capillary blood flow. Diffuse correlation spectroscopy (DCS) quantifies this decrease. DCS collects and analyzes multiply scattered, coherent, near infrared light to assess relative blood flow. This work presents a preliminary study using a DCS instrument with human subjects undergoing a lower body negative pressure (LBPN) protocol. This work builds on previous successful DCS instrumentation development and we believe it represents progress toward understanding how DCS can be used in a clinical setting.

Keywords

Diffuse correlation spectroscopy; speckle; hemorrhagic shock; multiple-scattering; capillary blood flow monitoring; microcirculation

1. INTRODUCTION

Detecting when a patient is experiencing shock is currently left to a doctor or nurse's clinical expertise. Subjective markers like clammy skin and patient behavior are typical of the only clues utilized. Syncope (loss of consciousness) is a symptom of the brain not receiving enough oxygen and often occurs as a patient goes into shock. The development of a non-invasive measurement that aids in the early detection of shock is of utmost importance. Recently the availability of medical data like heart rate and blood pressure have led to the development of various "shock indices" [1] [2]. These have shown some predictive value, but lack diagnostic robustness. Central venous oxygen saturation (ScvO₂) under 65% is correlated with large blood loss as well but requires an invasive and dangerous catheter insertion for measurement [3].

Hemorrhage is classified in stages, and it is desirable to detect the hemorrhage in Stage I before vital signs like heart rate and blood pressure show large changes [4]. Some redistribution of blood flow away from less important systems of the body may occur during Stage I and it would be desirable to detect non-invasively. These redistribution mechanisms can keep heart rate and blood pressure unchanged, masking the underlying problem [5].

Stroke volume consistently decreases with blood loss, but during Stage I would likely not change more than 10% from baseline [6]. Measuring changes in peripheral perfusion has been suggested as a way to detect ongoing hemorrhage earlier than is currently possible [7]. There is evidence that peripheral perfusion can change by much more than 10% during an episode of hemorrhage [8]. Of particular importance is determining how deep below the skin changes in perfusion must be monitored. Existing commercial technologies like laser Doppler flowmetry can only measure perfusion to a depth of about 1 mm [9]. Deeper measurements at multiple sites must be done to determine the optimum locations to monitor.

An optical technique called diffuse correlation spectroscopy (DCS) can provide interrogation of perfusion on the order of centimeter depth [10] [11]. We have been developing DCS blood monitoring technology for several years [12] [13] [8]. DCS injects near infrared light into a material and measures the autocorrelation of signals returning to a detector. The prototype described here measures blood flow at multiple points on a patient's body simultaneously, giving a practitioner a record of changes with time from when the patient is admitted.

Current competing technologies for blood flow monitoring include cumbersome and expensive methods like positron emission tomography (PET), Xenon enhanced computed tomography (XeCT) and arterial spin labeling – MRI. More closely related technologies include Doppler ultrasound which is limited to large diameter blood vessels and scanning laser Doppler flowmetry which is used for blood flow monitoring less than 1 mm below the tissue surface [9]. RMD has been developing a DCS-based blood perfusion monitor for several years, and it is also being tested for use in detecting free flap viability after maxillofacial surgeries [14].

DCS technology has already been tested on animals that were bled in a controlled manner, and this work describes human subject testing using a lower body negative pressure (LBNP) protocol. LBNP has been shown to cause a similar physiological response as that of acute hemorrhage in humans [15]. This work presents results from the first four subjects surveyed to prepare for a 30 subject trial. The integration time over which the DCS data was taken, was reduced from 30 seconds [8] to 10 seconds to check for higher frequency changes in the body's response to LBNP. Evidence of these fast responses were found. The goal is to develop a product that will allow patients to be continuously monitored for shock without the need for a doctor or nurse to be present in the room, saving the cost of the practitioner's time and freeing them to see to patients with more critical needs.

2. BACKGROUND

2.1 Diffuse Correlation Spectroscopy

DCS is a method to measure the speed of an ensemble of scatterers by measuring a time series of photon detection events. The method described here uses multiple scattering and monitors a single speckle cell with a single-mode optical fiber (see Figure 1). The light is delivered through a multimode source fiber and the detection is done with a single photon counting module (SPCM) from Excelitas (SPCM-AQRH). The SPCM is an APD operated in

Geiger mode, in which a single photon triggers an avalanche of charge. The operating voltage must be dropped below breakdown to reset before another detection can be made.

The DCS signal is the normalized intensity autocorrelation, $g_2(\tau)$, of the time series of detection events. The autocorrelation is calculated with a multi-tau method [16]. The normalized intensity autocorrelation function for single-scattering is [9]:

$$g_2(\tau) = \frac{\langle I(t)I(t+\tau) \rangle}{\langle I(t) \rangle^2} \quad (1)$$

where τ is the lag time after t , t is time and I is the intensity of detected light. The extension to multiple-scattering is detailed in [9]. The autocorrelation function calculated by the DCS system integrates the effect of all scattering events within the interrogation volume. In addition to the movement of the scattering particles, the autocorrelation signal also contains information about the optical properties of the tissue. The optical properties related to the autocorrelation signal are include the absorption coefficient, μ_a , the reduced scattering coefficient, μ'_s , the fraction of moving scatterers, α , and the mean square displacement of scatterers, $\langle r^2(\tau) \rangle$. This results from the fact that the autocorrelation can be thought to diffuse through the tissue in a similar way as the photons diffusing through the tissue [9]. As blood oxygenation and blood flow vary, it is possible that each of the four optical properties mentioned above can vary with time. RMD is currently developing a combination diffuse reflectance spectroscopy (DRS)/DCS system to isolate the blood flow measurement from the changes in static tissue scattering (μ_a and μ'_s).

2.2 Lower Body Negative Pressure Protocol

To study the effects of shock in humans in a controlled way, it is necessary to use protocols that do not involve actual blood loss. Hypovolemic shock, or hemorrhagic shock, occurs when the body loses a significant volume of blood or fluid, causing a decrease in perfusion. Other types of shock include cardiogenic shock and vasogenic shock due to inability of the heart to supply blood to the body and failure of the vasculature, respectively [17]. LBNP is a method to simulate the effect of shock by pulling blood away from the core and into the legs, creating a state of central hypovolemia [6]. The anticipated effects of LBNP are a decrease in several hemodynamic variables including central venous pressure, pulse pressure, systolic arterial pressure, stroke volume and cardiac output along with an increase in heart rate [6]. The legs of the volunteer are placed in a vacuum chamber that is sealed at the waist and air is progressively removed (see Figure 2). The amount of negative pressure applied roughly corresponds to the amount of blood removed from circulation. LBNP causes a decrease in stroke volume and cardiac output in the same way that hemorrhage does, and there is a roughly linear relationship between negative pressure and total blood loss (~25 ml/mmHg) [15]. Another study resulted in a slope of about 17 ml of blood loss per mmHg of LBNP for a 70 kg body weight [6]. LBNP causes a decrease in splanchnic volume which is a similar vascular response which occurs during hemorrhage [18].

However, the relationship between LBNP and the body's response to actual shock is complex. The amount of blood that is pulled into the legs depends on many factors including

leg compliance, size and vascular compliance [15]. Therefore there is significant variation in response subject to subject. Other factors that effect LBNP response are age, body size, physical conditioning, hydration and gender [15].

Symptoms of presyncope include nausea, continued systolic blood pressure of <80 mmHg, and/or bradycardia accompanied by a reduction in the difference between diastolic and systolic blood pressure [19]. In some reports, the act of standing can induce up to a 40% reduction in stroke volume and may be associated with a presyncopal state [18].

Hemorrhage and LBNP do not tend to correspond with respect to changes in hemoglobin, hematocrit, and central venous oxygen saturation [6]. This may complicate validating optical absorbance- and reflectance-based methods for shock monitoring with LBNP. It is suggested in [6] that CVP and pulse pressure are good early indicators of blood loss and these changes are also visible in LBNP. Pulse pressure can be measured non-invasively and is a good candidate for combination with DCS in future studies.

During LBNP, the reduction in pressure around the legs pulls fluid into the interstitial spaces of the tissue, pulling plasma out of circulation [6]. It is well known that microcirculation and macrocirculation may be decoupled during progression toward shock meaning that both should be monitored [20].

2.3 DCS with LBNP

It is important for shock monitoring instruments to be developed because a doctor or nurse's time is very limited and they cannot monitor many patients in person at once. This work indicates that DCS may detect the subtle reactions of the body to blood loss before catastrophic effects occur. We use a multichannel system to determine the optimum source/detector spacing, patch placement location, and physiological signatures to assess syncope from LBNP measurements. It has been suggested that simply measuring the capillary blood flow of skin on extremities may be complicated by environmental conditions and therefore a different location should be found for monitoring. The liver could be an important place to monitor for perfusion because blood flow reduction is greater there than in other vascular beds during the body's compensatory reaction to blood loss [21]. The need to monitor multiple sites must be balanced with the fact that there are limited places on the body that do not already have medical equipment attached in a standard hospital room. This work probed blood flow at multiple depths on multiple body parts to find the one that best corresponded with the onset of shock.

The change in some hemodynamic parameters is relatively small even at large LBNPs of -50 mmHg. For example Taneja *et al.* found less than 5% change in systolic and diastolic blood pressure, 10% change in heart rate and about 12% change in pulse pressure in a group of 10 volunteers [18]. We have found larger changes in DCS-measured peripheral blood flow.

3. METHODS AND MATERIALS

DCS requires a laser source with a long coherence length. It is important that the scattering volume is smaller than the coherence volume of the incident radiation [22]. In other words the coherence length of the source must be longer than the width of the distribution of detected photon path lengths through the scattering medium such that all detected photons will interfere coherently [22]. We have found that relatively inexpensive diode lasers (Thorlabs LD785-SH300) have coherence lengths long enough for DCS in human tissue. The laser light is delivered through a step index optical fiber (Thorlabs FG200LEA) with a core diameter of 200 μm . The scattered light is collected by a single-mode fiber (Thorlabs 780HP with a 4.4 μm core diameter). The source and detector fibers are integrated into a skin patch that includes a stiff epoxy chuck held in a flexible PDMS base (see Figure 3). The chuck can hold multiple detector fibers allowing for multiple depths to be monitored at the same time. We use an 8-channel box with integrated light sources, detectors, and programmable systems on a chip (PSOCs). The PSOC is described in detail in [23]. This system sends data to software developed at RMD to analyze a series of time-stamps corresponding to each recorded event to derive an autocorrelation curve. The rate of events and calculated time constant are also displayed by the software in real time (see Figure 4).

In the experiment described here, it is assumed that tissue optical properties are constant except for changes in scatterer velocity. The time constant associated with the decay of the autocorrelation signal is proportional to the movement of the blood cells in the vasculature. We use a triple exponential fit to account for the possibility of multiple blood flow components in the interrogation volume. The triple exponential fits to the normalized intensity autocorrelation, g_2 , are of the form:

$$y = A_1 \exp\left(-\frac{\tau}{t_1}\right) + A_2 \exp\left(-\frac{\tau}{t_2}\right) + A_3 \exp\left(-\frac{\tau}{t_3}\right) + y_0 \quad (2)$$

where y is the correlation, τ is lag time, y_0 is a constant offset, and the factors, A_n correspond to the amount of decay due to each time constant, t_n (see Figure 5).

In general the shallowest interrogation depths showed content in three exponentials while the deeper probes showed only one or two components. It is hypothesized that the shallow region probed by small source/detector separations can extend into areas with larger vasculature while the probes with larger source/detector separation show signals that are dominated by deeper tissue with little content from the surface capillaries.

The system was validated by several rounds of testing that included measuring the coherence length of the laser diodes, characterizing the throughput of each source and detector fiber, and measuring autocorrelation signals on flow phantoms in the lab. The instrument also showed the expected reduction and return to baseline when placed on the thumb during an arm-raising and lowering experiment.

The data was collected at the Mayo Clinic in Rochester, MN on Nov. 9–10, 2015. Four male volunteers underwent the LBNP protocol. The patches containing the source/detector pairs

were placed on each volunteer, and the largest spacing was adjusted such that the minimum rate of detected events was greater than about 20 kHz. This provides a large enough signal-to-noise ratio. Each volunteer spent at least 5 minutes at zero negative pressure, and then approximately 5 minutes at -15, -30, -45 and -60 mmHg unless they felt uncomfortable. If a patient did not wish to continue, the protocol was ended and they were immediately returned to zero negative pressure (recovery).

Medical data was taken at a rate of 1 kHz during the LBNP protocol. The medical measurements included ECG, blood pressure, SpO₂ (oxygen saturation), plethysmograph oxygen saturation (Pleth SpO₂), stroke volume, cardiac output, and respiration (see Figure 6 for example data). The DCS measurements were taken at four locations: thenar eminence (1 mm and 5 mm source/detector spacing), bicep (1 mm, 10 mm, and ~50 mm), forehead (1 mm and ~40 mm), and abdomen proximal to the hepatic system (~50 mm). The spacing between the source and detector fiber determines the depth at which perfusion is measured. For human tissue, the probed depth is about half the source/detector spacing [24]. These locations are considered to be physiologically distinct. It is hypothesized that the compartments of the body will react to shock in a certain order. The compartments being monitored are 1.) skin, 2.) skeletal muscle), and 3.) visceral organ.

4. EXPERIMENTAL RESULTS AND DISCUSSION

The DCS data was collected in 10 second intervals over the course of the protocol (except Subject 1 which had 30 second integration times). Previous research focused on the average value of the time constant over a long period of constant LBNP [8]. The higher time resolution presented here shows some interesting aspects that will be investigated further while still being consistent with previous data. Each patient showed a reduction in stroke volume vs. LBNP consistent with other studies [6]. One of the four patients (Subject 1) did not reach the end of the protocol due to the onset of uncomfortable symptoms. This is consistent with previous reports that 1/3 of people are unable to tolerate -60 mmHg of LBNP [6].

The decay constant associated with each integration time was then calculated with a fit done in Origin 2015. Batch processing allowed for most of the data to be analyzed automatically. There is some debate as to the best fitting method. In Subject 1 a shallow interrogation depth showed a similar increase in time constant as previous results (see Figure 7, bottom) [8]. Several deeper regions of the body also showed the same qualitative response to LBNP. If the surface of the skin is damaged or effected by the environment in some other way, the ability to probe deeper will still allow for blood flow monitoring and the information it provides about the patient's hemodynamic status. Also deeper probing may show a time dependence of the reaction to blood loss of multiple compartments of the body increasing the robustness of an algorithm that predicts shock based on multiple measurements. Figure 7 shows that the muscle compartment reacts similarly at both a 5 mm (10 mm source/detector separation) and 25 mm interrogation depth (50 mm source/detector separation), while the visceral organ compartment shows a less extreme but qualitatively similar response. The brain shows the opposite reaction again potentially improving the accuracy of a shock detection algorithm.

The data from Subject 1 is also interesting because the protocol was cut short due to patient duress. The point where Subject 1 ended the protocol stands out in a plot of three consecutive autocorrelation integration times (see Figure 8). The noise in the autocorrelation data increased as blood flow slowed and cardiac collapse was approached as in [8]. These observations are less valuable for early shock detection because they would occur with other obvious symptoms of cardiac collapse but demonstrate the functionality of the device.

Another notable aspect of this preliminary survey is the high frequency response on multiple patients across multiple parts of the body (see Figure 9 and Figure 10). Figure 9 shows what appears to be an oscillation in the blood flow while LBNP is held constant. The fact that the error in the fits (shown as error bars) is much smaller than the amplitude of the oscillation suggests it could be real. This will be investigated further in the larger trial. Figure 10 shows a sudden slowing in blood flow at several LBNP transitions only to settle back to a faster flow in less than 30 seconds. This may be a difference between LBNP and actual gradual hemorrhage and points toward a need to investigate a more gradual LBNP protocol. The right side of Figure 10 indicates that the measured slowing is not an artifact of the fits done by batch processing in Origin 2015.

Table 1 shows which locations on which volunteer exhibited any qualitative correspondence between decay constant and LBNP. Across all volunteers, the deeper bicep locations and the hepatic region showed relatively consistent reduction of blood flow until the recovery phase. On the other hand, the deeper palm depth showed an increase in blood flow while the LBNP protocol progressed (see Figure 10). The instances of a decrease in time constant (increase in flow) that occur before recovery could be due to capillary recruitment in which more capillaries open in response to hemorrhage [4]. This is associated with Stage II of hemorrhage and therefore an increase in blood flow in this case would not indicate the success of a particular intervention.

When combined with medical data that is routinely collected, the accuracy of shock diagnosis could improve further [8]. This experimental protocol will be performed on at least 20 more patients in the near future to increase the statistical significance of the results and confirm any preliminary patterns noted here.

5. SUMMARY AND CONCLUSION

This work indicates that a DCS instrument may be useful in detecting the onset of shock at various locations on the body. By testing multiple locations at multiple depths, the instrument was shown to give results consistent with previous work while extending to deeper regions below the skin. Increased DCS time resolution points toward potential physiological reactions to LBNP that require further investigation. Development of the RMD DCS blood perfusion monitor will free up doctors and nurses to spend time on patients that require their direct care and intervention, leaving at-risk patient monitoring to instrumentation.

Acknowledgments

This work was sponsored under the SBIR program (NIH contract #2R44HL106851).

References

1. Liu, Y-c, Liu, J-h, Fang, ZA., Shan, G-l, Xu, J., Qi, Z-w, Zhu, H-d, Wang, Z., Yu, X-s. Modified shock index and mortality rate of emergency patients. *World J Emerg Med.* 2012; 3(2):114–117. [PubMed: 25215048]
2. Cannon CM, Braxton CC, Kling-Smith M, Mahnken JD, Carlton E, Moncure M. Utility of the Shock Index in Predicting Mortality in Traumatically Injured Patients. *J Trauma.* 2009; 67(6):1426–1430. [PubMed: 20009697]
3. Scalea TM, Harnett RW, Duncan AO, Atweh NA, Phillips TF, Sclafani SJA, Fuortes M, Shaftan GW. Central venous oxygen saturation: A useful clinical tool in trauma patients. *The Journal of Trauma.* 1990; 30(12):1539–1543. [PubMed: 2258969]
4. Gutierrez G, Reines HD, Wulf-Gutierrez ME. Clinical Review: Hemorrhagic Shock. *Critical Care.* 2004; 8:373–381. [PubMed: 15469601]
5. Poeze M, Solberg BCJ, Greve JWM, Ramsay G. Monitoring global volume-related hemodynamic or regional variables after initial resuscitation: What is a better predictor of outcome in critically ill septic patients? *Crit Care Med.* 2005; 33(11):2494–2500. [PubMed: 16276172]
6. Hinojosa-Laborde C, Shade RE, Muniz GW, Bauer C, Goei KA, Pidcoke HF, Chung KK, Cap AP, Convertino VA. Validation of lower body negative pressure as an experimental model of hemorrhage. *J Appl Physiol.* 2014; 116:406–415. [PubMed: 24356525]
7. Genderen, MEv, Bartels, SA., Lima, A., Bezemer, R., Ince, C., Bakker, J., Bommel, Jv. Peripheral Perfusion Index as an Early Predictor for Central Hypovolemia in Awake Healthy Volunteers. *Anesthesia & Analgesia.* 2013; 116(2):351–356. [PubMed: 23302972]
8. Gurjar RS, Riccardi SL, Johnson BD, Johnson CP, Paradis NA, Joyner MJ, Wolf DE. Point-of-care Tool to Detect Early Stage of Hemorrhage and Shock," in. *Optical Diagnostics and Sensing XIV: Toward Point-of-Care Diagnostics.* 2014
9. Yu, G., Durduran, T., Zhou, C., Cheng, R., Yodh, AG. Near-Infrared Diffuse Correlation Spectroscopy for Assessment of Tissue Blood Flow. In: Boas, DA, Pitris, C., Ramanujam, N., editors. *Handbook of Biomedical Optics.* CRC Press; 2011. p. 195-216.
10. Boas D, Campbell L, Yodh AG. Scattering and Imaging with Diffusing Temporal Field Correlations. *Physical Review Letters.* 1995; 75(9):1855–1858. [PubMed: 10060408]
11. Boas D, Yodh AG. Spatially varying dynamical properties of turbid media probed with diffusing temporal light correlation. *J Opt Soc Am A.* 1997; 14(1):192–215.
12. Wolf DE, Seetamraju M, Gurjar RS, Kuo S, Fasi A, Feinberg S. Non-invasive monitoring of vascularization of grafted engineered human oral mucosa," in. *Dynamics and Fluctuations in Biomedical Photonics IX.* 2012
13. Vishwanath K, Gurjar R, Kuo S, Fasi A, Kim R, Riccardi S, Feinberg SE, Wolf DE. Sensing vascularization of ex-vivo produced oral mucosal equivalent (EVPOME) skin grafts in nude mice using optical spectroscopy," in. *Photonic Therapeutics and Diagnostics X.* 2014
14. Kolodziejewski, NJ., Stapels, CJ., McAdams, DR., Fernandez, DE., Podolsky, MJ., Farkas, D., Christian, JF., Ward, BB., Vartarian, M., Feinberg, SE., Lee, SY., Parikh, U., Mycek, M-A. A compact instrument to measure perfusion of vasculature in transplanted maxillofacial free flaps," in. *Optical Diagnostics and Sensing XVI: Toward Point-of-Care Diagnostics;* San Francisco. 2016;
15. Cooke WH, Ryan KL, Convertino VA. Lower body negative pressure as a model to study the progression of acute hemorrhagic shock in humans. *J Appl Physiol.* 2004; 96:1249–1261. [PubMed: 15016789]
16. Magatti D, Ferri F. Fast multi-tau real-time software correlator for dynamic light scattering. *Applied Optics.* 2001; 40(24):4011–4021. [PubMed: 18360435]
17. Tuggle, D. AACN Advanced Critical Care Nursing. Elsevier; 2008. Optimizing hemodynamics: strategies for fluid and medication titration in shock.
18. Taneja I, Moran C, Medow MS, Glover JL, Montgomery LD, Stewart JM. Differential effects of lower body negative pressure and upright tilt on splanchnic blood volume. *Am J Physiol Heart Circ Physiol.* 2007:H1420–H1426. [PubMed: 17085534]
19. Ganio MS, Overgaard M, Seifert T, Secher NH, Johansson PI, Meyer MAS, Crandall CG. Effect of heat stress on cardiac output and systemic vascular conductance during simulated hemorrhage to

presyncope in young men. *Am J Physiol Heart circ Physiol*. 2012;H1756–H1761. [PubMed: 22367508]

20. Lima A, Bommel Jv, Sikorska K, Genderen Mv, Klijn E, Lesaffre E, Ince C, Bakker J. The relation of near-infrared spectroscopy with changes in peripheral circulation in critically ill patients. *Crit Care Med*. 2011; 39(7):1649–1654. [PubMed: 21685739]
21. Brinkmann A, Calzia E, Trager K, Radermacher P. Monitoring the hepato-splanchnic region in the critically ill patient. *Intensive Care Med*. 1998; 24:542–556. [PubMed: 9681775]
22. Bellini T, Glaser M, Clark N, Degiorgio V. Effects of finite laser coherence in quasielastic multiple scattering. *Physical Review A*. 1991; 44(8):5215–5223.
23. Stapels, CJ., Kolodziejski, NJ., McAdams, D., Podolsky, MJ., Fernandez, DE., Farkas, D., Christian, JF. Advanced Biomedical and Clinical Diagnostic and Surgical Guidance Systems XIV. San Francisco: 2016. A scalable correlator for multichannel diffuse correlation spectroscopy," in.
24. Mozina H, Podbregar M. Near Infrared spectroscopy for evaluation of skeletal muscle tissue oxygenation in different types of shock. *Signa Vitae*. 2015; 10(1):10–24.

Figure 1.
Schematic of DCS

Figure 2.
Volunteer undergoing LBNP protocol with DCS blood flow measurements.

Figure 3.
Combination of skin patch with single detector fiber to increase source/detector separation and therefore probing depth.

Figure 4. Screenshot from RMD DCS GUI. The plots show count rate (top left), decay constant associated with single exponential fit to the autocorrelation function (top right) and the autocorrelation function (bottom).

Figure 5.

Representative sample of autocorrelation curves from shallow (1 mm source/detector separation) and deeper (5 mm source/detector separation) DCS probes. Note the multiple components of the curve from a shallow depth. (Subject 1, bicep, 1 and 5 mm source/detector spacing, 0 mmHg LBNP)

Figure 6.

Left) Stroke volume vs. LBNP; Right) Cardiac output vs. LBNP (Subject 1).

Figure 7.

Time constants from single exponential fits from subject 1. The labels above each graph show the LBNP applied during that period. BL, baseline; 15, -15 mmHg, R, recovery, etc. Upper left) bicep at 50 mm source/detector separation; Upper right) bicep at 10 mm; Middle left) Abdomen (near liver) 50 mm; Middle Right) Forehead (near brain) 40 mm; Bottom) Thenar eminence 1 mm. Each file corresponds to an integration time period (30 seconds). Curves in Figure 8 correspond to the circled points.

Figure 8.

Three consecutive integration times as Subject 1 corresponding to circled time constants in Figure 7 (bicep, 10 mm source/detector separation ended the protocol early due to presyncope).

Figure 9.

Time constants increase with LBNP until recovery. There appear to be high frequency oscillations in the blood flow during each LBNP level. The fluctuations are greater than the error in the fits suggesting they are real (Subject 2, bicep, 10 mm source/detector separation).

Figure 10.

Left) Time constants for single exponential fit to data. Note that at each LBNP transition, there is a fast response that then settles. Also the time constants start decreasing before recovery, possibly indicating capillary recruitment (Subject 4, palm, 5 mm source/detector separation). Right) Autocorrelation curves showing quick response to LBNP change before settling corresponding to circled points in plot to the left (Subject 4, palm, 5 mm source/detector separation).

Table 1

Qualitative behavior of blood flow in each region of each patient during LBNP protocol. "x" means that blood flow slows until recovery. "x*" means blood flow increased before recovery.

Patient	Bicep (1 mm)	Bicep (10 mm)	Bicep (50 mm)	Palm (1 mm)	Palm (5 mm)	Forehead (1 mm)	Forehead (40 mm)	Abdomen (50 mm)
1				x	x			
2		x	x		x*		x	x
3		x*	x*	x*	x*			x
4	x	x	x	x*	x*			x*