

A Novel Statistical Model for Simulation of Arterial and Intracranial Pressure

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Abstract—We describe a novel statistical model of pressure signals that incorporates the effects of respiration on arterial (ABP) and intracranial pressure (ICP). This model can be used to synthesize pulsatile ABP and ICP signals with similar time, frequency, and variability characteristics of real pressure signals. These synthetic signals can be used during the development, simulation, or quantitative assessment of biomedical algorithms in a variety of applications.

Keywords—ABP modeling, ICP modeling, respiratory changes, pressure signal simulators.

I. INTRODUCTION

SYNTHETIC physiologic signal simulators and biomedical signal models are important tools in biomedical signal analysis. The ability to simulate physiologic signals with realistic characteristics enables researchers to develop and test their algorithms and methodology on synthetic signals prior to validation on experimental or clinical data. The main advantage of synthetic data over real data is that we know its statistical properties and other characteristics, and therefore there is a clear gold standard which can be used to quantitatively evaluate the performance of the algorithm.

There are three main settings where the availability of signal simulators is important: (1) quantitative assessment of biomedical noise enhancement algorithms, (2) validation of biomedical estimation algorithms, and (3) validation of detection algorithms. In the case of noise removal and parameter estimation algorithms we often cannot assess their performance quantitatively using real data, since the uncorrupted (clean) signal or the parameter to be estimated are not available. The availability of annotated synthetic signals with real characteristics also allows developers to test their detection algorithm extensively without the need of manual annotations. Furthermore, synthetic signals enable researchers to develop their algorithms and methodologies in situations where real data is yet unavailable or must be reserved for posterior prospective studies.

Although software tools have been developed to simulate ECG signals [1], [2], there are no pressure signal simulators available for pressure signals. The unavailability of synthetic pressure signal simulators has, in part, prevented researchers from conducting simulation studies and quantitative algorithm validation and assessment on synthetic arterial blood pressure (ABP) and intracranial pressure (ICP) signals.

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In this paper we present a simple mathematical model of physiologic pressure signals. The model incorporates the three primary effects of respiration on arterial and intracranial blood pressure (described later). Furthermore, we present a software implementation of the model that can be used to generate synthetic pressure waveforms with realistic characteristics.

II. MODEL DESCRIPTION

A. Modeling Pressure Signals: Respiratory Changes in Arterial and Intracranial Pressure

The simplest model of pulsatile pressure consists of a pure sinusoid with a DC component,

$$p(t) = \mu_p + P \cos(2\pi f_c t) \quad (1)$$

where μ_p corresponds to the mean pressure, P is the pulse amplitude, and f_c is the cardiac frequency.

A more realistic model of physiologic pressure signals must take into account the pulse morphology, its variability, and the effects of respiration on the pressure signal. There is abundant literature on respiration and respiratory modulation of human autonomic rhythms [3]–[7]. There are three primary effects of respiration on pressure signals: pulse amplitude variation (amplitude modulation), respiratory sinus arrhythmia (frequency modulation), and an additive effect.

B. Modeling Pulse Amplitude Changes with Respiration

The amplitude modulation accounts for part of the pulse pressure changes and we modeled as a double-sideband large carrier (DSB-LC) amplitude modulation (AM), also known as conventional AM,

$$\phi_{AM}(t) = A_c[1 + am_n(t)] \cdot \cos 2\pi f_c t \quad (2)$$

where $m_n(t)$, the normalized message signal, corresponds to respiration, and the carrier signal $\cos(2\pi f_c t)$ represents the pulsatile ABP or ICP components at the cardiac frequency. Since both ABP and ICP signals are composed of more than one harmonic and are not exactly periodic, the carrier signal $\cos(2\pi f_c t)$ does not provide an accurate model of ABP or ICP pulse pressure. However, we can model ABP and ICP as a modulation scheme where the carrier is not a pure sinusoid, but a periodic signal with more than one harmonic,

$$p(t) = A_c[1 + ar_n(t)] \cdot \sum_{n=-\infty}^{\infty} C_n e^{j2\pi f_c n t} \quad (3)$$

where $r_n(t)$ is the normalized respiratory signal (i.e. $|r_n(t)| \leq 1$), and the carrier signal is a periodic signal with an arbitrary pulse morphology that has a Fourier series representation.

The pressure signal model $p(t)$ given in (3) can be simplified by considering only the first two harmonics (i.e. f_c and $2f_c$), since most of the power in real pressure signals is contained in the first two harmonics and the DC component. The simplified model is then,

$$\begin{aligned} p(t) &= \mu_p + A_c[1 + ar_n(t)] \cdot (\alpha \cos 2\pi f_c t + \beta \cos(4\pi f_c t + \theta)) \\ r(t) &= \sigma \cos 2\pi f_r t, \quad r_n(t) = \cos 2\pi f_r t \end{aligned} \quad (4)$$

According to this model, the normalized respiratory signal $r_n(t)$ modulates a carrier signal with a respiratory frequency f_r . The carrier signal is given by $c(t) = \alpha \cos 2\pi f_c t + \beta \cos(4\pi f_c t + \theta)$ and has a fundamental frequency of f_c (i.e. the cardiac frequency) and a second harmonic at twice the fundamental. The term $[1 + ar_n(t)]$ is always greater than zero. This allows for envelope demodulation of respiration.

C. Modeling Respiratory Sinus Arrhythmia

The model for pressure signals given in (4) accounts for the amplitude modulation effect that respiration has on pulse pressure, the constant cardiac component, and a reflected wave due to the changes in impedance in the arteries. However, it does not incorporate the effect of respiratory sinus arrhythmia (i.e. frequency modulation of the cardiac frequency with respiration). The general form of frequency modulation is given by (5)

$$\phi_{FM}(t) = A_c \cos \left(2\pi f_c t + 2\pi k_f \int_{-\infty}^t m(\tau) d\tau \right). \quad (5)$$

Since in our model $m(t) = r(t) = \sigma \cos(2\pi f_r t)$, the integration is trivial. Incorporating the frequency modulation and the additive effect of respiration, the model becomes,

$$\begin{aligned} p(t) &= \mu_p + A_c[1 + ar_n(t)] \cdot \left[\alpha \cos(2\pi f_c t + \beta_f \sin 2\pi f_m t) + \right. \\ &\quad \left. + \beta \cos(4\pi f_c t + \beta_f \sin 2\pi f_m t + \theta) \right] + \kappa \cdot r(n) \end{aligned} \quad (6)$$

where $\beta_f = \frac{k_f a}{f_r}$ is the modulation index and k_f is the FM deviation constant.

D. Modeling Pulse Morphology Variability

The model for pressure signals presented in (6) is deterministic. Even though it can be used to generate synthetic pressure signals with realistic time morphology and frequency characteristics, and incorporates the amplitude, frequency and additive effects of respiration on pressure; it lacks the pulse morphology variability typical of real ABP or ICP signals. Variability is incorporated into the model by adding Gaussian

noise $n(t)$ to the deterministic pressure signal $p(t)$ given in (6), and passing it through a linear-time invariant (LTI) channel,

$$p_\nu(t) = \int_{-\infty}^{\infty} h(t - \tau)[p(\tau) + n(\tau)]d\tau. \quad (7)$$

Note that the channel model of (7) is not the classical communications linear filter channel; in this case the noise is added to the signal prior to sending it through the LTI filter. The impulse response of the channel can also be made time-variant $h(\tau; t)$, which enables to incorporate low-frequency variability by slowly updating the filter coefficients.

Noise such as motion artifacts and abrupt baseline drifts are not part of the model, since $p_\nu(t)$ is only intended to capture the physiologic characteristics of pressure signals and its variability, not artifacts.

E. Modeling Frequency Variability

As we already established, the simplest model of pulsatile pressure consists of a pure sinusoid with a DC component. We generalized this simple model by modeling the effects of pulse pressure variation as a conventional amplitude modulation (AM) of a multi-frequency pulse pressure carrier with respiration as the modulating signal, and an additive effect,

$$p(t) = u_p + [1 + ar_n(t)] \cdot \sum_{n=1}^N C_n e^{j2\pi f_c n t} + kr(t) \quad (8)$$

We refer to (8) as the *harmonic model*. In this model $r_n(t)$ is the normalized respiratory signal, a is the modulation index, and the carrier signal is a quasi-periodic signal with an arbitrary pulse morphology that can be approximated as a multi-harmonic periodic signal with a fundamental frequency of f_c , corresponding to the cardiac frequency. The respiratory signal $r_n(t)$ can also be modeled as a multi-harmonic signal with a fundamental frequency equal to the respiratory rate f_r . In the harmonic model the effect of respiratory sinus arrhythmia is modeled as a sinusoidal FM modulation of $p(t)$ with respiration.

The main limitation of the harmonic model is the lack of stochastic variability in the frequency domain, that is, the variation of the cardiac and respiratory frequencies is entirely deterministic. To overcome this limitation, we generalized this model by modeling f_c and f_r as a sum of two components: a constant carrier frequency \bar{f} and a stochastic frequency variation $\lambda(t)$,

$$\begin{aligned} f_r(t) &= \bar{f}_r + \lambda_r(t), \quad \lambda_r(t) = - \sum_{k=1}^{P_1} a_k \sigma_r(t - k) + w(t) \\ f_c(t) &= \bar{f}_c + \lambda_c(t), \quad \lambda_c(t) = - \sum_{k=1}^{P_2} b_k \lambda_c(t - k) + w(t) \\ &\quad + \sum_{k=0}^Q h_c(k) \lambda_r(t - k) \end{aligned} \quad (9)$$

where the cardiac $\lambda_c(t)$ and the respiratory $\lambda_r(t)$ stochastic frequency variations were modeled as two correlated autoregressive (AR) processes. The model is completed by passing

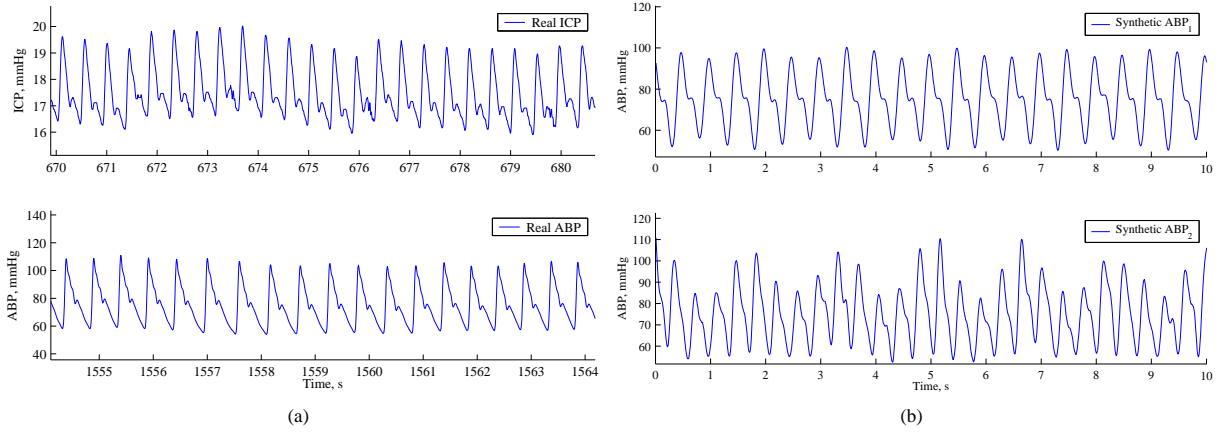


Fig. 1. (a) Example of two real pressure signals over a 10 s period. (b) Plot showing two synthetic pressure signal with distinct characteristics generated using the model presented.

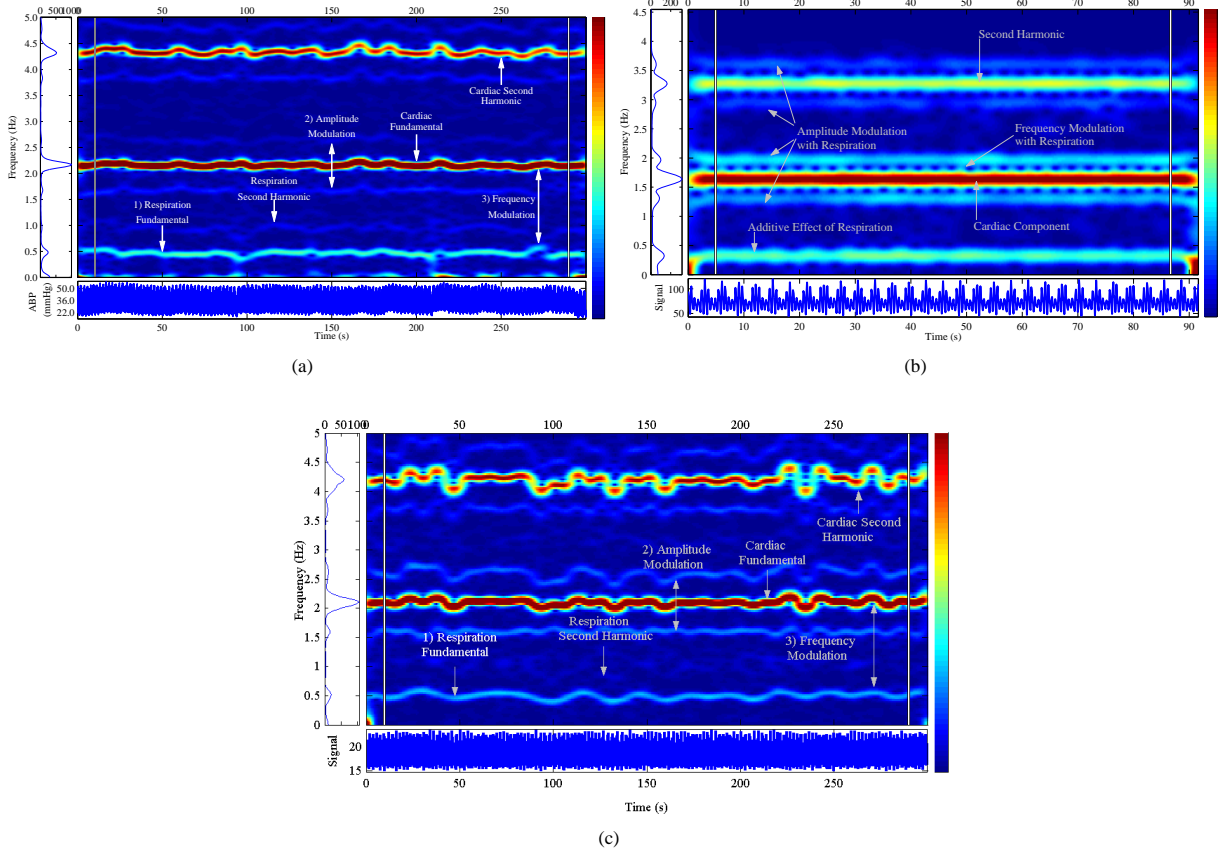


Fig. 2. (a) Spectrogram of a real pressure signal showing its frequency content. (b) Spectrogram of a synthetic pressure signal generated using the harmonic model. (c) Spectrogram of a synthetic pressure signal generated using the proposed statistical model.

$$p(t) = \sum_{k=0}^{K-1} h(k, t) \left\{ u_p + \left[1 + a \sum_{l=1}^K \gamma_l e^{j2\pi (\sum \bar{f}_r + \lambda_r(t)) l(t-k)} \right] \cdot \sum_{n=1}^N C_n e^{j2\pi (\sum \bar{f}_c \lambda_c(t)) n(t-k)} + \kappa r(t-k) \right\} \quad (10)$$

TABLE I
USER-SPECIFIED PARAMETERS OF THE MODEL.

Par.	Description	Par. Range
τ	Signal duration	—
α	Cardiac component amplitude	0–1
β	Second harmonic amplitude	0–1
σ	Respiratory component amplitude	1–5
κ	Additive respiration amplitude	1–5
f_r	Respiratory frequency	0.1–1
f_c	Cardiac frequency	1–2.5
θ	Phase of the second harmonic	$-\frac{\pi}{2} - -\frac{\pi}{2}$
a	Respiratory AM index	0.0 – 1
β_f	Respiratory FM index	0.0 – 0.5
μ_p	Mean pressure (i.e. $\bar{p}(t)$)	5–200
σ_p	Standard deviation of $p(t)$	1–15
σ_n	Standard deviation of Noise	0–15
N	Filter Order (MA coefficients)	3–10

the pressure signal through a fading multipath channel to incorporate the nonstationary pulse pressure variability typical of real ICP and ABP signals. Thus, the general model equation is given by (10).

F. Model Implementation

The model was implemented as a MATLAB function. It can be used as a random ABP and ICP signal generator or to simulate pressure signals with specific time and frequency characteristics. In addition to the model parameters shown in (6), the user specifies standard deviation (σ_p) of the pressure signal, and the noise power (σ_n^2). Table I shows the user-specified parameters.

III. RESULTS AND DISCUSSION

Fig. 1(a) and 1(b) show time domain plots of real and synthetic pressure signals. Each figure consist of two plots, and illustrate the capability of the model to generate synthetic pressure signals with similar time domain morphology, amplitude and frequency modulation, and frequency characteristics to those present in real pressure signals.

Fig. 1(a) shows an example of a real ICP and ABP signal a 10 s period. Comparing these real pressure signals with the synthetic signals generated using the model (Fig. 1(b)), we see that the simulated pressure signals seem to capture the variability and other characteristics of real pressure signals. It is difficult to differentiate between the real and synthetic signals based on time domain visual inspection of the signals only. This was confirmed by a brief expert study where a group of five experts were ask to individually classify 30 ICP signals as real or synthetic based on visual inspections of 10 s. time domain plots. The results of this survey showed that experts were unable to accurately distinguish between real and synthetic ICP signals.

The proposed harmonic model is capable of generating synthetic pressure signals with similar time domain characteristics to real pressure signals, as it was confirmed by the expert

study previously described. This is due to the fact that the model captures the pulse morphology and its variability in the time domain. However, visual inspection of a time–frequency decomposition such as the spectrogram of a synthetic signal reveals the shortcomings of the harmonic model. Comparing the spectrogram of a real pressure signal such as the one shown in Fig. 2(a) with the spectrogram obtained from a synthetic pressure signal generated using the harmonic model Fig. 2(b)), we can clearly see the main model limitation, namely, the lack of stochastic variability of the respiratory and cardiac frequency components. The frequency variability is completely deterministic, changing sinusoidally, as a consequence of the model harmonic structure. This may limit the applicability of the model in some situations, for instance, in the case of evaluation of detection algorithms. To overcome this limitation, we proposed to model the respiratory and cardiac components as correlated AR processes. Comparing the spectrograms of the real and synthetic pressure signal generated using this generalization (Fig. 2(c)), we can observe that the simulated pressure captures the frequency characteristics typical of real pulsatile pressure.

IV. CONCLUSION

We described a simple mathematical model of pressure signals that can be used to synthesize pulsatile ABP and ICP signals with similar time, frequency, and variability characteristics of real pressure signals. These synthetic signals can be used during development and assessment in a variety of applications. These include quantitative validation of noise removal algorithms, assessment of pressure component detection algorithms, estimation of respiration from ABP or ICP, and spectral estimation.

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