Impact of congestive heart failure on voice and speech production: A pilot study

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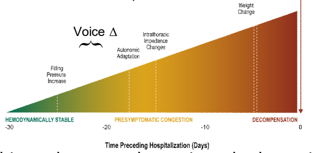
Motivation

Noninvasive identification of volume overload is critical to maintaining stability of chronic heart failure (HF) patients. Current methods (e.g. weight monitoring) have limited reliability and only reflect changes that occur shortly before the onset of symptoms.

Time Course of Changes in Physiologic Markers of Heart Failure Decompensation



The goal of this study was to determine whether voice and speech changes in chronic HF patients hospitalized for acute decompensation during diuresis are correlated with measures of volume status such as weight, NT-proBNP, and symptoms.

Hypotheses

- vocal fold edema lowers pitch and increases acoustic perturbation
- volume overload increases the frequency of breaths during continuous speech

Methods

Ten HF patients with acute decompensation were studied. The following voice and cardiac-related assessments were performed:

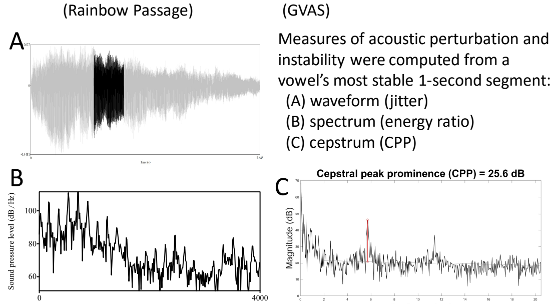
Daily

- Physical exam
- Sustained vowels
- Standard reading passage (Rainbow Passage)

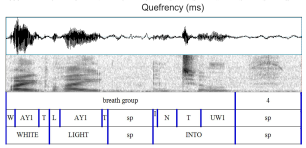
Admission and discharge

- Plasma NTproBNP
- Dyspnea visual analog scale (DVAS)
- Global symptoms visual analog scale (GVAS)

Measures of acoustic perturbation and instability were computed from a vowel's most stable 1-second segment:
(A) waveform (jitter)
(B) spectrum (energy ratio)
(C) cepstrum (CPP)



Passages were transcribed (right) and speech was segmented into breath groups to yield measures of respiratory stress.



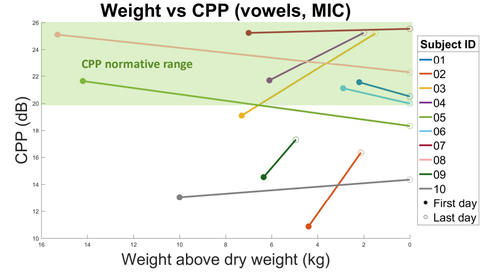
Results

	1	2	3	4	5	6	7	8	9	10	Mean ± SD
	Patient number										
Age (yr)/Sex	82/F	76/M	47/M	65/F	69/M	58/M	58/M	70/M	86/M	84/M	**69.5 ± 12.8**
Baseline BNP (pg/mL)	3712	2201	3766	1919	2900	11521	6866	4939	2616	31601	**7204 ± 9040**
Baseline weight (kg)	88.0	79.0	116.6	61.4	122.5	104.9	119.7	127.7	78.5	90.6	**98.8 ± 22.5**

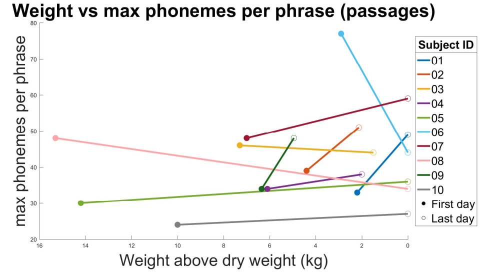
Total changes in selected measures from admission to discharge

	Δ Measure	1	2	3	4	5	6	7	8	9	10	# improved
Cardiac	**ΔWeight (kg)**	−5.9	−2.13	−7.3	−6.1	−14.2	−2.9	−7	−15.3	−14.4	−10	**10 of 10**
	ΔNT-proBNP (pg/ml)	−623		−2303	233	−47	−8497	−2982	−465	2617	−348	**7 of 9**
	ΔDVAS	0.14	−0.15	0.14	−0.27	0.07	0.15	0.16	0.43			**6 of 8**
	ΔGVAS	0.52	−0.11	0.42	−0.16	0.07	0.26	0.09	0.68			**6 of 8**
Acoustic	**ΔPitch stdev (Hz)**	−0.73	−0.20	−0.12	−0.47	−0.08	−0.01	1.15	0.13	−2.64	−6.47	**8 of 10**
	ΔJitter (pts)	−0.12	−1.53	−0.06	−0.02	0.24	−0.04	−0.06	0.25	−0.76	−0.39	**8 of 10**
	ΔCPP (dB)	−1.05	5.46	6.09	3.55	−3.30	−1.13	0.30	−2.82	2.76	1.30	**6 of 10**
Respiratory	**ΔTalk time (seconds)**	−3.21	1.50	−4.24	−0.53	1.02	−1.21	0.62	−2.60	−0.31	−9.75	**7 of 10**
	ΔPhonemes per phrase (maximum)	16	12	−2	4	6	−33	11	−14	14	3	**7 of 10**

Weight vs CPP (vowels, MIC)



Weight vs max phonemes per phrase (passages)



Discussion

Measures of voice stability and respiratory capacity correlate with improvements in HF patients after diuresis:

- Overall acoustic irregularity (CPP)
- Pitch instability (pitch standard deviation, jitter)
- Respiratory stress (phonemes per phrase)

Inter-subject variation and considerations

- Changes in voice may not be directly related to the amount of vocal fold edema. Compensatory mechanisms allow talkers to produce similar voices under different physiological conditions.
- Older patients can have loss of vocal fold vibratory tissue that negatively impacts voice quality. HF-related edema may improve these patients' voices.

Future Work

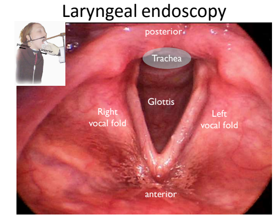
Current data analysis:

- Analyze measures from neck-surface accelerometer
- During-stay changes in weight vs. voice measures
- Computation of additional voice and speech measures

Future data collection:

- Laryngeal endoscopy to image HF impact on vocal fold tissue
- Enroll healthy controls matched for age and co-morbidities
- Additional speech and respiratory assessment tasks

Laryngeal endoscopy



Acknowledgments

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References

Adamson, P. B. (2009). Pathophysiology of the transition from chronic compensated and acute decompensated heart failure: New insights from continuous monitoring devices. Current Heart Failure Reports, 6(4):287–292.
Awan, S. N., Roy, N., Jette, M. E., Meltzner, G. S., & Hillman, R. E. (2010). Quantifying dysphonia severity using a spectral/cepstral-based acoustic index: Comparisons with auditory-perceptual judgements from the CAPE-V. Clinical Linguistics and Phonetics, 24:742-758.
Yuan, J. and Liberman, M. (2008). Speaker identification on the SCOTUS corpus. Proceedings of Acoustics '08.
Zeitels, S. M., and Healy, G. B. (2003). Laryngology and phonosurgery, New England Journal of Medicine, 349(9):882–892.