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# Temporal structure of variability decreases in upper extremity movements post stroke

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1                   **Temporal structure of variability decreases in upper extremity**  
2                   **movements post stroke**

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45 **Abstract**

46 *Background:* The objective of this study was to determine movement variability in  
47 the more-affected upper-extremity in chronic stroke survivors. We investigated  
48 two hypotheses: (1) individuals with stroke will have increased amount of  
49 variability and altered structure of variability in upper-extremity joint movement  
50 patterns as compared to age-matched controls; and (2) the degree of motor  
51 impairment and joint kinematics will be correlated with the temporal structure of  
52 variability.

53 *Methods:* Sixteen participants with chronic stroke and nine age-matched controls  
54 performed three trials of functional reach-to-grasp. The amount of variability was  
55 quantified by computing the standard deviation of shoulder, elbow, wrist and  
56 index finger flexion/extension joint angles. The temporal structure of variability  
57 was determined by calculating approximate entropy in shoulder, elbow, wrist and  
58 index finger flexion/extension joint angles.

59 *Findings:* Individuals with stroke demonstrated greater standard deviations and  
60 significantly reduced approximate entropy values as compared to controls.  
61 Furthermore, motor impairments and kinematics demonstrated moderate to  
62 strong correlations with temporal structure of variability.

63 *Interpretation:* Changes in the temporal structure of variability in upper-extremity  
64 joint angles suggest that movement patterns used by stroke survivors are less  
65 adaptable. This knowledge may yield additional insights into the impaired motor

66 system and suggest better interventions that can enhance upper-extremity  
67 movement adaptability.

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69 *Keywords:* Time-dependent structure, Motor skills, Complexity, Kinematics,  
70 Upper extremity

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## 84 **1. Introduction**

85           Stroke is a leading cause of disability in the United States affecting over  
86 795,000 individuals every year (American Heart Association, 2010). Up to 85% of  
87 individuals with stroke exhibit hemiparesis resulting in upper-extremity (UE)  
88 impairments (Olsen, 1990). Unfortunately, despite the development of various  
89 rehabilitation techniques, residual UE impairments remain (Duncan et al., 2000;  
90 Nakayama et al., 1994). Thus, a more thorough understanding of UE  
91 impairments is needed to develop effective treatments maximizing motor ability  
92 post-stroke.

93           Among the constellation of UE impairments, individuals with post-stroke  
94 hemiparesis often exhibit atypical movement patterns characterized by mass and  
95 whole limb movements with limited dissociation between joints (Cirstea and  
96 Levin, 2000). These aberrant movement patterns exhibit high variability in terms  
97 of increased standard deviation (SD) and/or coefficient of variation (CV) in  
98 several kinematic measures: UE joint range of motion, peak velocity, movement  
99 time and trajectory accuracy as compared to healthy controls (Cirstea and Levin,  
100 2000; Woodbury et al., 2009). SD and CV are linear measures of variability and  
101 quantify the amount of variability, or movement error, around a central point  
102 (Newell, 1976); however, they cannot capture the fine adjustments of the limbs  
103 that occur during the course of motor performance (Harbourne and Stergiou,  
104 2009). UE movements involve continuous adjustments to successfully reach and  
105 grasp objects of various sizes and shapes. For instance, individuals make  
106 continuous fine adjustments to maintain their grip on a glass, if they perceive that

107 the glass may slip from their hands. These fine adjustments or variations made  
108 during continuous movements over time are referred as temporal structure of  
109 variability (Harbourne and Stergiou, 2009). Temporal structure of variability  
110 allows individuals to adapt their movement patterns to overcome perturbations  
111 encountered during daily tasks. Temporal structure of variability can be quantified  
112 using nonlinear measures such as approximate entropy (ApEn) (Harbourne and  
113 Stergiou, 2009). Unlike linear measures of variability, which compute variability  
114 around the mean of a movement parameter, ApEn examines the variability by  
115 evaluating all values of a movement parameter over the entire time series. Non-  
116 linear measures capture the temporal structure of variability that occurs over time  
117 reflecting the adaptability of the motor system. There is limited evidence of the  
118 application of non-linear measures in UE motor impairments post stroke.  
119 Therefore, the application of non-linear measures to characterize the temporal  
120 structure of variability in UE movement may yield additional insights into impaired  
121 motor control post-stroke.

122         Stergiou, Harbourne and Cavanaugh (2006) proposed that an optimal state  
123 of variability is associated with a healthy motor system. This model suggests that  
124 healthy states are associated with optimal movement variability and this  
125 variability reflects the adaptability of the underlying control system. The principle  
126 of optimality is demonstrated by an inverted U-shape relationship exhibited  
127 between complexity and predictability. At an optimal state of movement  
128 variability, the largest complexity lies in the intermediate region between  
129 maximum predictability and no predictability and is representative of a “healthy”

130 state. For a detailed description of the optimal variability model refer to figure 2 in  
131 Stergiou, Harbourne and Cavanaugh (2006). Complexity signifies the presence  
132 of chaotic temporal variations in the steady state output of a healthy biological  
133 system and represents the underlying physiologic capability to adapt to everyday  
134 stresses placed on the human body (Lipsitz and Goldberger, 1992; Lipsitz,  
135 2002). Decrease or loss of the optimal state of variability renders the system  
136 more predictable and rigid exhibiting a robotic type of motor behavior. For  
137 example, individuals with stroke often exhibit UE movements with limited  
138 dissociation between joints resulting in predictable or stereotypical movements  
139 referred as abnormal synergies. Conversely, increases beyond optimal variability  
140 render the system more noisy and unpredictable. For instance, individuals with  
141 movement disorders such as ataxia or athetosis, often demonstrate jerky,  
142 uncontrolled and less predictable movements of extremities. Both situations  
143 reveal decreased complexity, flexibility and adaptability to perturbations and are  
144 associated with impairments in ability to engage UE in meaningful tasks.

145 Movement adaptability is an innate and fundamental feature of a healthy  
146 nervous system (Lipsitz and Goldberger, 1992; Stergiou, Harbourne and  
147 Cavanaugh, 2006). Everyday functional tasks involve continuous adaptations of  
148 reach and grasp movements to meet the dynamic demands of the tasks.  
149 Temporal structure of variability allows individuals to adapt their movement  
150 patterns to overcome perturbations encountered during daily tasks. Several  
151 changes associated with stroke, including spasticity, decreased range of motion  
152 (Cirstea and Levin, 2000), difficulty dealing with the interaction torques produced



153 by muscle contractions, and abnormal motor recruitment patterns, (Dewald et al.,  
154 1995) might alter the temporal structure of variability in UE joints. Consequently,  
155 altered temporal structure of variability should be reflected in the altered  
156 adaptability of UE movement.

157       Examining variability in reaching movements post-stroke provides a window  
158 to understand the impaired motor system and suggest better interventions that  
159 enhance UE movement adaptability. Therefore, the primary aim of this study was  
160 to compare the amount and the temporal structure of variability of the shoulder,  
161 elbow, wrist and proximal interphalangeal (PIP of index finger) flexion/extension  
162 joint angles during reach-to-grasp movements between healthy individuals and  
163 individuals with stroke. We hypothesized that the amount of variability of  
164 shoulder, elbow, wrist, and PIP angles would be significantly greater and the  
165 temporal structure of variability of shoulder, elbow, wrist and PIP joint angle  
166 movement patterns would be significantly reduced in individuals post- stroke as  
167 compared to in healthy individuals.

## 168 **2. Methods**

### 169 *2.1. Participants*

170       The participants were 16 individuals diagnosed with stroke and nine  
171 healthy controls. The mean years of age for the participants with stroke was 67.6  
172 (SD 8.1) and for the healthy controls 57.2 (SD 6.7). Demographic information as  
173 well as lesion location and severity of stroke based upon the UE Fugl-Meyer  
174 subscale for individuals with stroke are presented in Table 1. The participants  
175 were part of a larger study investigating upper-extremity motor rehabilitation.

176 Participants were included if they: (1) were between the ages of 18-90 years; (2)  
177 had a single ischemic stroke at least 6 months prior to enrollment; (3) were able  
178 to follow two-step commands; (4) had no history of more than minor head  
179 trauma, subarachnoid hemorrhage, dementia or other neural  
180 disorder/dysfunction, drug or alcohol abuse, schizophrenia, serious medical  
181 illness, or refractory depression. A sample of convenience comprised of eight  
182 right hand dominant females and one left hand dominant male were recruited  
183 from the staff of the Brain Rehabilitation Research Center to serve as healthy  
184 age-matched controls.

## 185 *2.2. Procedures*

186 Eligible participants provided written informed consent approved by the  
187 University of Florida Institutional Review Board and North Florida/South Georgia  
188 Veterans Health System's Research and Development Committee. Each  
189 participant was evaluated once at the Human Motor Performance Laboratory  
190 located within the Brain Rehabilitation Research Center.

191 Individuals with stroke reached to grasp a soda can (56 mm in diameter;  
192 208 mm circumference) with the paretic UE. Healthy controls reached with their  
193 non-dominant hand. Sixty-seven reflective markers were secured to various  
194 landmarks of the upper body as illustrated in Figure 1. Marker placements were  
195 determined using a marker set described by the Plug-In-UE marker set defined  
196 by our laboratory (Patterson et al., 2011). All participants wore dark colored  
197 sleeveless shirts and were seated on an adjustable, backless bench with knees  
198 bent at 90° flexion and feet flat on the floor. The hands were placed palm down

199 on a table in front of them and supported in 90° of elbow flexion by arm rests  
200 positioned flush with the table. This position was the starting position for all the  
201 trials.

202 A soda can was placed at 80% arm's length (Michaelson et al., 2004) on  
203 the table directly in front of the respective shoulder of the participant. This  
204 distance has been referred to as the "critical boundary" (Mark et al., 1997).  
205 Healthy individuals use UE joints alone to reach for objects within this  
206 workspace; to obtain objects beyond this boundary; they might involve the trunk  
207 by leaning forward (Mark et al., 1997). All participants were instructed to reach for  
208 the can, lift it off the table, and put it back down as fast as possible and return to  
209 the starting position. All participants performed four trials with the first serving as  
210 a practice trial. Each trial was cued with a "go" command.

### 211 *2.3. Data analysis*

212 Kinematics of reaching were recorded using two different 12-camera  
213 VICON motion capture systems (Vicon 612; Oxford Metrics In., Oxford, UK). All  
214 controls and 11 individuals post-stroke were tested using a 12MX camera system  
215 and Vicon Workstation v4.6 software at a sampling frequency of 100Hz. The  
216 remaining five individuals post-stroke were tested using 12 T40 Vicon cameras  
217 and Vicon Nexus 1.5.2 software with data sampled at 200 Hz. Data collected  
218 using VICON Nexus were down sampled from 200 to 100Hz to construct  
219 comparable time series and enable appropriate comparisons.

220 Data analysis was performed on the last three trials. The data were the 3D  
221 positional coordinates of each marker with respect to a laboratory coordinate

222 system throughout the movement series. The data were manually labeled and  
223 reconstructed using Vicon software, and then modeled using SIMM (4.2, Santa  
224 Rosa, CA) to calculate the shoulder, elbow, wrist and PIP angles. The start of  
225 reach was identified as the time point at which the velocity of the index finger  
226 marker exceeded 5% peak velocity and the termination of reach as the time point  
227 at which velocity of this marker fell below 5% peak velocity. One degree of  
228 freedom in the sagittal plane (flexion/extension) was used to determine shoulder,  
229 elbow, wrist and PIP joint angle. To retain the inherent temporal structure of the  
230 variability present, the kinematic data were not filtered prior to analysis (Rapp,  
231 Albano, Schmah, and Farwell, 1993).

#### 232 *2.4 Variability of UE kinematics*

233 To measure the amount of variability, SDs of three trials of the shoulder,  
234 elbow, wrist and PIP joint angle range of motion were computed. The temporal  
235 structure of variability of shoulder, elbow, wrist and PIP joint angle time series  
236 was determined by computing approximate entropy (ApEn) with the MATLAB  
237 code (R2009a, Natick, MA) developed by Kaplan and Staffin (1996) utilizing the  
238 algorithm provided by Pincus, Gladstone, and Ehrenkranz (1991). Each joint  
239 angle time series was analyzed from the start of the reach through the entire  
240 length of the respective time series including the pauses between the three trials.  
241 This approach was adopted because ApEn is effectively a measure of  
242 probability, developed to identify whether small patterns of a time series repeat  
243 later in the entire time series. These small patterns might not be repeated in a  
244 single trial of reach-to-grasp movement. Overall, four time series were obtained

245 (one for each joint). The most common method employed in the computation of  
246 ApEn is to identify repeating vectors of length  $m$  across the entire time series  
247 (figure 2). Biomechanical data analysis conventionally utilizes  $r = 0.2$  times the  
248 standard deviation of the time series,  $\text{lag} = 1$  and  $m = 2$  (Slifkin and Newell,  
249 1999). Because the length of the data could affect ApEn values, we normalized  
250 the ApEn values of each participant to the length of their time series and then  
251 multiplied the ratio with a constant equal to 100. A more detailed description of  
252 the computation of ApEn can be reviewed in the Appendix of Slifkin and Newell  
253 (1999). Generally, a vector of shorter length repeats more often than a longer  
254 one within a time series, thus the lowest possible ApEn value can be the natural  
255 logarithm of 1, which is 0. ApEn values range from 0 to 2. In a highly periodic  
256 time series, values of  $C_m(r)$  can be similar to  $C_{m+1}(r)$  producing  $\text{ApEn} = 0$ .  
257 Hence, smaller values characterize a more regular time series where similar  
258 patterns are more likely to follow one another. In contrast, high ApEn values,  
259 suggest a highly irregular time series, where the predictability of subsequent  
260 patterns is low and ApEn could be close to 2 (Stergiou et al., 2004).

261 We also computed the percentage contribution of each joint to the total  
262 ApEn of UE. Total ApEn was computed by adding the ApEn from shoulder,  
263 elbow, wrist and PIP for each participant. Thereafter, the percentage contribution  
264 from each joint was obtained by multiplying the ratio of the individual joint ApEn  
265 to total ApEn by 100. Such analyses would reveal the distribution of ApEn across  
266 UE joints.

267

## 268 2.6. *Surrogate analysis*

269 A surrogation procedure was applied prior to computing ApEn utilizing the  
270 Theiler et al. (1992) first algorithm. Surrogation procedure is a critical step to  
271 perform prior to computing ApEn to verify whether the kinematic data were  
272 deterministic in nature and not a source of noise. Theiler's first algorithm (1992)  
273 utilizes a phase randomization technique which removes the deterministic  
274 structure from the original shoulder, elbow, wrist and PIP joint angle time series  
275 creating 20 surrogate time series of each trial with the same mean, variance, and  
276 power spectrum as the original time series. ApEn was then computed on the  
277 original as well as each of the 20 surrogate time series. Significant differences in  
278 ApEn between the original and 19 of 20 surrogate time series confirm the  
279 deterministic nature of the original data.

## 280 2.7 *Statistical Analysis*

281 Dependent one-tailed t-tests were conducted to compare ApEn shoulder,  
282 elbow, wrist and PIP values between the original and surrogate time series using  
283 SPSS (17.0, Chicago, IL). For the remaining analyses non-parametric statistics  
284 were employed due to the violation of assumptions of normality using SPSS  
285 (17.0, Chicago,IL). Mann-Whitney U tests were employed to investigate the  
286 differences in SD and ApEn shoulder, elbow, wrist and PIP between individuals  
287 with stroke and healthy controls. Mann-Whitney U tests were also employed to  
288 compare the percent contribution of each joint's ApEn to total ApEn between  
289 healthy controls and individuals with stroke. Data were analyzed with statistical  
290 significance set at  $P < 0.05$ . Holm's step-down procedure was used to correct for

291 multiple comparisons (Holm, 1979).

292

### 293 **3. Results**

#### 294 *3.1 Determinism in joint angle time series using surrogate analysis*

295 Determinism in the joint angle time series was confirmed in both control ( $P$   
296 = 0.001) and stroke ( $P = 0.000$ ) groups which revealed significantly greater  
297 shoulder, elbow, wrist and PIP ApEn values in surrogate time series. These  
298 findings suggest that the data were deterministic in nature and not a source of  
299 noise.

#### 300 *3.2 Amount of variability in joint angle time series*

301 Individuals with stroke had larger SDs for shoulder, elbow, wrist and PIP  
302 angles than for healthy controls. However, these differences did not reach  
303 statistical significance ( $P > 0.05$ ) (Table 3).

#### 304 *3.3 Temporal structure of variability in joint angle time series*

305 Individuals with stroke exhibited significantly less ( $P < 0.05$ ) ApEn values  
306 across all UE joints than controls (Table 2). Additionally, the contribution of ApEn  
307 of movement at each joint to the total ApEn differed between the groups. The  
308 percent contribution of ApEn PIP joint to total ApEn was significantly greater ( $P =$   
309 0.002) for controls than for individuals with stroke (Table 2). In contrast,  
310 individuals with stroke demonstrated a significantly greater percent contribution  
311 of ApEn elbow ( $P = 0.002$ ) and wrist ( $P = 0.014$ ) joints to total ApEn than controls  
312 (Table 3). However, the difference in percent contribution of ApEn shoulder joint  
313 to total ApEn was not significantly different ( $P = 0.803$ ) between controls and

314 individuals with stroke (Table 2).

315

#### 316 **4. Discussion**

317       The primary purpose of the study was to compare the differences between  
318 the amount and temporal structure of variability in UE movements between  
319 individual's post-stroke and healthy controls. Although not statistically significant,  
320 SD values were lower across all joints in healthy controls than individuals post-  
321 stroke. In contrast, ApEn values across all joints were significantly greater in  
322 healthy controls than individuals post-stroke. Based upon the optimal variability  
323 model, healthy controls exhibit an optimal nervous system, which may  
324 demonstrate chaotic temporal variations revealing optimum adaptability to meet  
325 the demands of everyday stresses placed on the human body. Deviance from the  
326 optimal variability model may suggest the presence of pathology; less than  
327 optimal variability may be representative of a more rigid, less adaptable system  
328 limiting the repertoire of movement strategies (Harbourne and Stergiou, 2009;  
329 Scholz, 1990). The results of this study suggest that temporal structure of  
330 variability is reduced in individuals post stroke, which potentially could alter the  
331 adaptability in their reach to grasp movements.

332       In healthy controls, ApEn was significantly greater in the index finger PIP  
333 joint than the shoulder, elbow and wrist joints. Lower ApEn values characterize a  
334 more stable or regular time series whereas; high ApEn values suggest an  
335 unstable or irregular time series. Hence, lower shoulder ApEn values suggest  
336 that shoulder is utilized primarily for stabilization of the arm during reach-to-



337 grasp. Alternatively, the PIP joint might have produced greater adjustments  
338 essential in manipulating the grasp around the can during the reach-to-grasp  
339 task. Greater ApEn values at the PIP compared to more proximal joints in the  
340 healthy controls are consistent with the current literature, which supports the  
341 versatile nature of hand (Lemon, 1993; Tallis, 2003). The advanced ability of the  
342 hand to grasp and manipulate objects of various sizes, shapes and textures is  
343 one of the key features of the human motor system (Begliomini et al., 2008).

344         In contrast to healthy controls, participants post-stroke demonstrated a  
345 significantly greater percent contribution from the wrist and elbow joints to total  
346 ApEn. Individuals post-stroke possibly made significantly greater adjustments  
347 with the wrist and elbow than with the PIP joint implicating an alternative  
348 compensatory strategy for accomplishing the reach-to-grasp task. The significant  
349 reduction in the percentage contribution of PIP joint ApEn values post- stroke  
350 could be due to the fact that motor neuron pools of distal UE segments are  
351 primarily innervated by the corticospinal tract, which is frequently compromised in  
352 stroke (Colebatch and Gandevia, 1989). Furthermore, Raghavan et al. (2010)  
353 also observed alternative movement strategies, where individuals with stroke  
354 compensated PIP joint flexion by increased flexion at the metacarpophalangeal  
355 joint during grasping of concave and convex shaped objects. Understanding how  
356 multiple effectors coordinate to produce a goal directed movement still remains a  
357 challenge to motor control researchers (Diedrichsen et al., 2009). Commonly  
358 referred to as the degrees of freedom problem (Bernstein, 1967), motor  
359 coordination is concerned with how work is distributed across multiple effectors

360 (muscles, joints) when multiple options exist to perform a task. Optimal control  
361 theory suggests that an optimization process might be a potential solution to the  
362 degree of freedom problem of motor control (Diedrichsen et al., 2009). Optimal  
363 control theory proposes that the selection of effectors for a particular task is the  
364 consequence of an optimization process based upon the cost function made up  
365 of the goal and the effort required to accomplish the goal. Stroke might change  
366 the cost function for a particular movement. For individuals with moderate UE  
367 deficits post-stroke, manipulating the index finger PIP joint around the soda can  
368 might require too much effort. Thus, the compensation strategy involving the  
369 wrist and elbow joints might involve re-optimization in setting up the new cost  
370 function and redistributing work across effectors. In fact, using the wrist may  
371 have made it easier to open and close the fingers due to the biomechanical  
372 properties of the long flexors (e.g., flexor digitorum superficialis), which cross  
373 both the wrist, and fingers.

374         We acknowledge certain limitations of this study. Given the heterogeneity  
375 observed in stroke, this sample size was relatively small, thus the lack of  
376 significant differences between groups in shoulder and elbow SD might reflect a  
377 lack of statistical power. The findings of this study are also limited to seated  
378 unimanual, discrete reach-to-grasp tasks. Further research is necessary to  
379 understand specific neurological mechanisms contributing to the changes in  
380 variability in UE joints post-stroke compared to other kinematic and functional  
381 variables. In particular, the effects of location and size of brain lesion, severity of  
382 the lesion, integrity of the descending motor pathways, individual degree of

383 spontaneous recovery, and the duration of stroke onset upon temporal structural  
384 of variability of UE joints needs to be explored. Additionally, future research is  
385 warranted to determine whether or not constraining the trunk might affect the  
386 temporal structure of variability. There is also a need to determine the effects of  
387 intervention on these variables.

## 388 **5. Conclusion and Implications for Rehabilitation**

389 Our findings reveal that the temporal structure of variability in reach-to-  
390 grasp movements is significantly reduced post-stroke. A measure of the temporal  
391 structure of variability seems to capture differences between the groups; even  
392 with a small cohort of individuals post-stroke we were able to significantly  
393 differentiate between healthy controls and individuals with stroke utilizing ApEn.  
394 In contrast, employing linear measures, such as the standard deviation, we failed  
395 to detect differences between healthy controls and individuals with stroke.

396 **Analyzing temporal structure of variability in UE movements provides a**  
397 **novel perspective on understanding motor impairments in individuals**  
398 **living with stroke. ApEn could potentially be utilized to measure the**  
399 **efficacy of UE rehabilitation intervention. Future research is warranted to**  
400 **establish the psychometric properties of ApEn prior to its use as an**  
401 **outcome measure.**

402

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535 Figure Captions

536 Figure 1. Upper extremity marker set.

537 Figure 2. Approximate entropy (ApEn) equation

538 Figure 3a. Standard deviation (SD) of various UE joints between healthy controls  
539 and individuals with stroke

540 b. Approximate Entropy (ApEn) of various UE joints between healthy  
541 controls and individuals with stroke (\* = significant)

542

543 c. Approximate entropy (ApEn) percent of each joint to total ApEn in  
544 healthy controls and individuals with stroke (\* = significant)