Complete and Voluntary Starvation of 50 days

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ABSTRACT: A 34-year-old obese male (96.8 kg; BMI, 30.2 kg m^{-1}) volitionally undertook a 50-day fast with the stated goal of losing body mass. During this time, only tea, coffee, water, and a daily multivitamin were consumed. Severe and linear loss of body mass is recorded during these 50 days (final 75.4 kg; BMI, 23.5 kg m^{-1}). A surprising resilience to effects of fasting on activity levels and physical function is noted. Plasma samples are suggestive of early impairment of liver function, and perturbations to cardiovascular dynamics are also noted. One month following resumption of feeding behavior, body weight was maintained (75.0 kg; BMI, 23.4 kg m^{-1}). Evidence-based decision-making with the fasting or hunger striking patient is limited by a lack of evidence. This case report suggests that total body mass, not mass lost, may be a key observation in clinical decision-making during fasting and starvation.

KEYWORDS: severe fasting, hunger strike, caloric restriction

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Introduction

A 34–year-old male presented for general examination on day 49 of a 50-day self-imposed fast, seeking approval to continue his fasting behavior. During this period, the patient reported only consuming water, tea, coffee (with no milk or sugar), and a daily multivitamin tablet (Centrum Performance; Pfizer).

Patient history prior to fasting behavior revealed a family history of obesity and the presence of pathologies related to body mass. Personal history involved an oscillating mass between 85 and 134 kg over a 10-year period. At the start of fasting, the patient reported body mass as 96.8 kg, and 29.2% body fat, as recorded by a personal bioelectrical impedance device. History following the onset of fasting behavior was unremarkable, but included severe abdominal cramps between days 7 and 21 of fasting, development and frequent occurrence of vasovagal syncope on transition from sitting to standing, and the near-cessation of fecal movement. Eructation frequency was significantly increased. Self-reported physical activity and mobility was reduced, but not prevented; this participant was not bedridden. Substantial water and coffee intake was noted (8–12 cups of coffee daily and 2–3 bottles of water daily). As expected, body mass loss during fasting was severe and noticeable (Fig. 1).

Initial examination showed maintenance of motor and cranial nerve function. Resting metabolic rate (30 minutes by indirect calorimetry) was 1626 kcal day^{-1}. During the 30-minute rest period, ECG and blood pressure were monitored. Resting ECG appeared morphologically normal, patient was bradycardic (52 bpm) and notably hypertensive (150/78 mmHg). Body composition was measured by whole-body air displacement plethysmography (Bod Pod) as 19.8% body fat, while height was 180.9 cm and mass was 75.4 kg, with a BMI of 23.5 kg m^{-1}, all recorded on

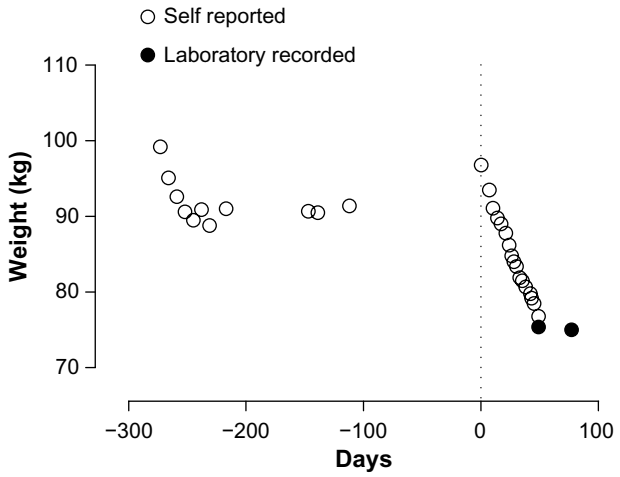

Figure 1. One-year history of body mass.
Notes: Day 0 indicates start of fast. Open circles indicate self-reported values, closed circles indicate laboratory recorded. Mass is given in kilogram.

calibrated laboratory devices. Sum of four skinfolds[1] was 30.0 mm, approximately half of that of a reference population of similarly aged males (62 ± 25 mm, aged 30–39). It was visually noted that subcutaneous fat in the abdominal region was still present, despite severe losses in the peripheral limbs, protruding ribs, and lack of prominent chest and shoulder musculature. A grade 1/2 panniculus resulting from rapid abdominal adipose loss was present (Fig. 2). During a six-minute walk test, this individual covered 342 m, which was below expected performance for a healthy male adult.[2] A mild scoliosis was observed while walking; however, it is unclear if this is related to fasting.

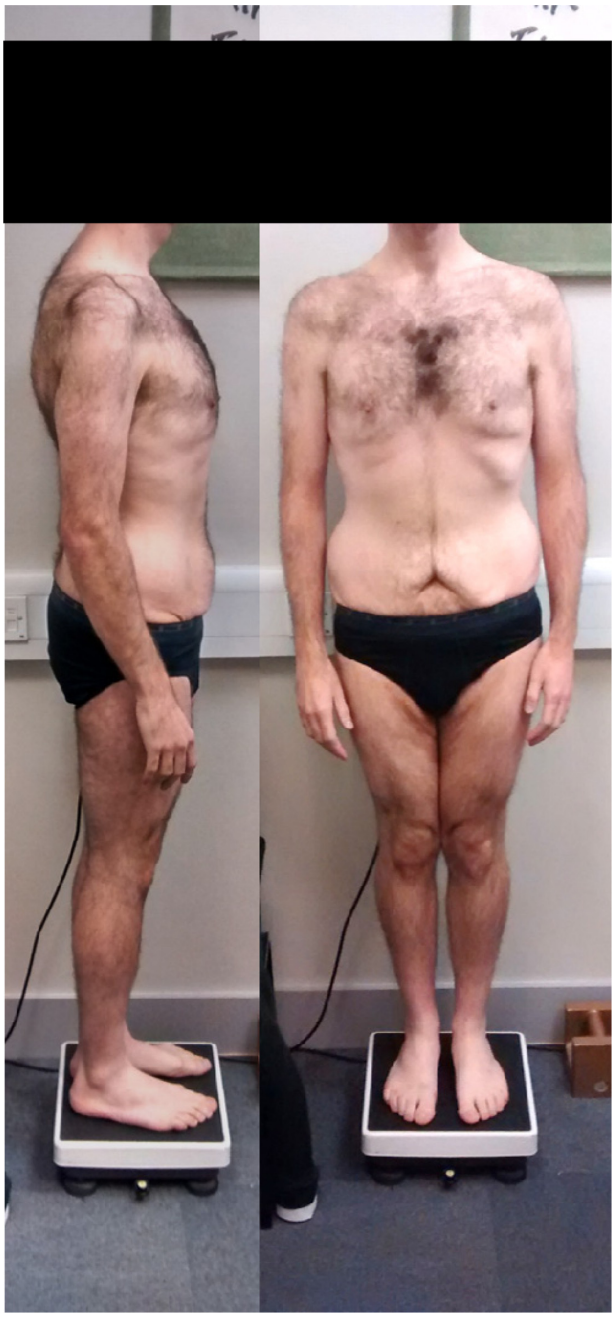

Figure 2. Anterior and lateral view whilst standing. Day 49 of fast.

Venous blood was taken for serum markers of metabolism, liver, and renal function. Results are given in Table 1. Noteworthy findings include plasma glucose of 3.1 mmol L^{-1} and alkaline phosphatase of 36 U L^{-1}. Total bilirubin was 19 µmol L^{-1}, direct bilirubin was 7 µmol L^{-1}. Uric acid was 620 µmol L^{-1}.

Urine was straw colored, lacked sediment, and was noticeably sweet in aroma. Rapid dipstick analysis (Multistix 2161; Siemens) was negative for blood glucose, contained trace proteins, and indicated urine pH of below 5. Urine was highly ketonuric (>16 mmol L^{-1}).

One Month Past Breaking of Fast

One month following initial presentation, the patient returned for follow-up examination during which body composition, resting metabolic rate, urine, and venous blood were recorded. Resumption of feeding initially involved liquids such as vegetable soups, with the resumption of solid foods three days following breaking of fast. Days 3–5 involved one solid meal per day, with 2–3 solid meals during days 5–7, and the resumption of three solid food meals per day for 7 days following breaking of fast. In the following 23 days, diet was

Table 1. Serum biochemical values at day 49 of fast and 28 days post cessation of fasting.

	DAY 49 OF FAST	28 DAYS POST RESUMPTION	UNITS
Albumin	49	44	g·L^{-1}
Alkaline phosphatase	**36***	42	U·L^{-1}
Alanine aminotransferase	34	19	U·L^{-1}
Aspartate aminotransferase	33	23	U·L^{-1}
Calcium	2·40	2·30	mmol·L^{-1}
Adjusted calcium	2·22	2·22	mmol·L^{-1}
Cholesterol	**3·4***	3·7	mmol·L^{-1}
Creatinine	90	80	µmol·L^{-1}
Gamma-glutamyl transferase	24	27	U·L^{-1}
Glucose	**3·1***	4·0	mmol·L^{-1}
HDL-cholesterol	1·50	1·37	mmol·L^{-1}
LDL-cholesterol	2·0	2·1	mmol·L^{-1}
Phosphate	1·3	1·1	mmol·L^{-1}
Potassium	4·6	4·6	mmol·L^{-1}
Sodium	142	148	mmol·L^{-1}
Total bilirubin	19	18	µmol·L^{-1}
Direct bilirubin	**7·0***	**4·6***	µmol·L^{-1}
Total protein	73	71	g·L^{-1}
Triglycerides	0·8	0·5	mmol·L^{-1}
Urea	2·3	3·4	mmol·L^{-1}
Uric acid	**620***	355	µmol·L^{-1}

Note: *Values outside normal range for age and gender.[12]

reported to be of caffeine and alcohol free, low carbohydrate (a piece of bread every 1–3 days), and high servings of fruit and vegetables. Self-estimation of diet by volume was 20% animal protein (chicken and fish) and 80% fruit or vegetables; validated, quantifiable measures of intake and caloric content are not available. Heart rate remained bradycardic at rest (48 bpm), and blood pressure was notably reduced (132/78). Metabolic rate was reduced (1413 kcal day^{-1}), and body composition was unchanged (75.0 kg; BMI, 23.4 kg m^{-1}; 16.1% fat mass). Urinary ketones were reduced, but not removed. Normal fecal movements were reported to begin seven days following resumption of feeding. Blood biochemistry returned to within normal range, with the exception of direct bilirubin (Table 1). The six-minute walk test was repeated, and the patient covered 401 m.

Discussion

Clinical examinations and research models reporting such severe starvation are absent in modern literature. Clear ethical barriers preclude structured research into the physiology of such stimuli. It is noteworthy, therefore, in this case study that moderate physical function appears maintained at day 49, as assessed by general observation and results of the six-minute walk test. Frommel et al.[3] suggested that fasting in healthy lean individuals was well tolerated until 18% of body mass was lost; the patient reported here lost 20.7% of body mass without substantial loss of function. One prior report of long-term fasting in obese individuals for 30–40 days demonstrated body mass losses between 10.6% and 20.5%, but does not report on function of individuals.[4] It seems self-apparent and supported by this limited evidence, such that the starting mass is a key variable in survival of extreme fasting, and not the amount of loss. This patient started fasting while obese and ended his fast when anthropometric variables were within the targeted ranges for the general population. Indeed, among the few (nonpublished) examples of complete fasting and mortality,[5] patients were frail between 30 and 50 days, and death was noted to occur between days 43 and 70, which this case study has reached without obvious or significant frailty occurring. These findings were reported in nonoverweight individuals.

Current World Medical Association guidelines prioritize autonomy, recommending nonintervention in fasting to the point of harm and/or death, if the patient makes a written informed statement of intent.[5] Lack of research into this field precludes evidence-based decision-making or advice. In the limited light of this case study and historic data of fasting in obesity,[4] it is tempting to suggest that guidelines to physicians for advice to the fasting individual reflect body mass, and specifically mass of adiposity at start of fasting behavior. Survival in starvation is ultimately governed by physics, the calories available to maintain metabolic function. Availability of adipose tissue for metabolic usage may thus delay the catastrophic degradation of muscular protein, increasing survival time.

That said, small animal models reveal organ-specific effects of starvation, with loss of liver and gut size and function noted after only four to six days.[6] Functional damage to digestive organs cannot be ruled out, and indeed, the elevated direct bilirubin is suggestive of reduced liver filtration. Of concern, direct bilirubin remained elevated 30 days post breaking of fast. Elevated uric acid is likely due to reduced kidney filtration, as has been reported in caloric restriction previously.[7]

Further, hypertension was an unexpected finding. This finding is counter to one of the few published examinations of cardiology in severe starvation, where bradycardia and hypotension (~20 mmHg below fed state) were noted in juvenile pigs following 29 days of complete starvation.[8] The porcine heart contains several structural differences relative to the human, most related to bipedal vs. quadrupedal stance.[9] As frequent syncope was also noted in that our case study, it is plausible that dysregulated autonomic regulation of blood pressure underlies the observed hypertension. Blood pressure was measured in a supine position, increasing venous return in the human relative to standing position. Simple Frank–Starling mechanics combined with a failure of autonomic regulation may thus explain elevated blood pressure. Thus, it is tempting to suggest that differences observed may be species dependent; however, this is highly speculative on a single case study. Further, long-lasting cardiovascular deficits were noted in the above porcine model on refeeding, as well as chronically following experimental conclusion.[8] As death resulting from starvation often occurs via cardiovascular complications, this elevated blood pressure during fast is key to note; this should be carefully monitered in fasting individuals.

Caloric restriction and weight loss in overweight or obese individuals is often associated with subsequent regain of weight.[10,11] Having access to this case study 30 days following resumption of feeding was of interest due to a chance to examine any occurrence of such a rebound effect. It is of interest to note that this was not seen here; however, future gains in weight cannot be ruled out.

Here, we report the maintenance of physical function of an individual after 49 days of complete fast. This report does not indicate support for such behavioral choices, and the projections of metabolic energy provision by starting adiposity assume no other perturbations to homeostasis, such as impaired immune response, liver and renal function, or cardiovascular events. Such a voluntary behavioral choice is difficult to support.

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Author Contributions

Conceived and designed the experiments: BE. Analyzed the data: BE. Wrote the first draft of the manuscript: BE. Contributed to the writing of the manuscript: BE, MM, CF. Agreed with manuscript results and conclusions: BE,

MM, CF. Jointly developed the structure and arguments for the paper: BE, CF. Made critical revisions and approved the final version: BE. All the authors reviewed and approved the final manuscript.

REFERENCES

1. Durnin JV, Womersley J. Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *Br J Nutr*. 1974;32(1):77–97.
2. Chetta A, Zanini A, Pisi G, et al. Reference values for the 6-min walk test in healthy subjects 20–50 years old. *Respir Med*. 2006;100(9):1573–8.
3. Frommel D, Gautier M, Questiaux E, Schwarzenberg L. Voluntary total fasting: a challenge for the medical community. *Lancet*. 1984;1(8392):1451–2.
4. Owen OE, Felig P, Morgan AP, Wahren J, Cahill GF Jr. Liver and kidney metabolism during prolonged starvation. *J Clin Invest*. 1969;48(3):574–83.
5. O'Hagan S. Hunger: the real maze men speak. *The Guardian*. 2008. Available at: http://www.theguardian.com/film/2008/oct/19/northernireland.
6. Steiner M, Bourges HR, Freedman LS, Gray SJ. Effect of starvation on the tissue composition of the small intestine in the rat. *Am J Physiol*. 1968;215(1):75–7.
7. Kirch W, von Gicycki C. [Renal function in therapeutic starvation (author's transl)]. *Wien Klin Wochenschr*. 1980;92(8):263–6.
8. Smith GS, Smith JL, Mameesh MS, Simon J, Johnson BC. Hypertension and cardiovascular abnormalities in starved-refed Swine. *J Nutr*. 1964;82:173–82.
9. Crick SJ, Sheppard MN, Ho SY, Gebstein L, Anderson RH. Anatomy of the pig heart: comparisons with normal human cardiac structure. *J Anat*. 1998; 193(pt 1):105–19.
10. Hemmingsson E, Johansson K, Eriksson J, Sundstrom J, Neovius M, Marcus C. Weight loss and dropout during a commercial weight-loss program including a very-low-calorie diet, a low-calorie diet, or restricted normal food: observational cohort study. *Am J Clin Nutr*. 2012;96(5):953–61.
11. Tsai AG, Wadden TA. The evolution of very-low-calorie diets: an update and meta-analysis. *Obesity (Silver Spring)*. 2006;14(8):1283–93.
12. Instrumentation Laboratory. *Applications Manual ILab Aries*. Bedford, MA: Instrumentation Laboratory; 2014. Revision 06-01-14.