RENAL FUNCTION IN MARATHON RUNNERS*

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SUMMARY

Thirty-one competitors in a particularly strenuous 54-mile marathon race had their renal function assessed before and after the event. Seventy per cent of them had red cells in their urine and 60% showed proteinuria after the event, but in most cases the urine returned to normal apart from a trace of protein after 24 hours. Elevations in blood urea and potassium were moderate and not related to urinary changes. These abnormalities were also transient and in those competitors showing the most marked changes, creatinine clearance returned to normal within 4 months and usually within 2 weeks. No significant structural changes were noted in 4 patients who had pyelograms. The electrocardiograms of these runners showed no ischaemic changes, and the serum proteins remained normal.

The Comrades Marathon is an annual event run between Durban and Pietermaritzburg, a distance of 54 miles—over twice the distance of the Olympic Marathon. The route lies over hilly terrain, the weather is usually warm, and the race takes between 6 and 11 hours to complete. In spite of this it regularly attracts over 600 competitors! In 1967, and again in 1968, a runner developed anuria after the event.[1] We were interested to observe whether this severe exertion had any adverse effect on the kidneys of other runners.

Thirty-one randomly selected runners co-operated in the study and were examined medically during the week before the race. A 12-lead electrocardiogram, a mid-stream urine specimen and a blood sample were taken. The urine was examined microscopically and biochemically in 27 competitors and all had blood urea and electrolyte estimations. In 19 competitors serum proteins were estimated. All the tests were performed immediately on fresh specimens.

After the race a 12-lead electrocardiogram was repeated. The organizers kindly supplied a tent within 18 metres of the finishing line, and although some cardiograms were obtained within a minute of finishing the race, in others there was a delay of 5 - 10 minutes or longer. Blood samples were taken and analysed that same afternoon for urea, potassium and protein. All specimens of urine passed either during the race or during the 24 hours from the commencement were examined separately. In addition, the first sample of urine passed immediately after the race was examined in 26 other competitors so that a total of 53 competitors had urine examined after the race. Runners who showed abnormalities in blood or urine tests immediately after the race, returned 2 weeks later for additional tests which in some cases included creatinine clearance studies and intravenous pyelograms. Blood specimens were analysed using standard procedures.

RESULTS

An abnormal number of red blood cells (more than 5 per high-power field) were present in the urine of 23% of the runners, 40% had between 1 - 5 red blood cells/high-power field, and the remaining 37% had no cells present in the urine. Red cells when present in the urine were not present in all specimens and after 24 hours the urine was free of cells in all except 2 runners (No. 12 and 13).

Competitor No. 3 did not void urine during the race, but had 95 - 100 red blood cells/high-power field and a moderate proteinuria in the first specimen passed after the race. Five hours later he had 1 - 2 red blood cells/high-power field and no albumin and 24 hours after the commencement of the race the urine was completely clear.

Competitor No. 11 passed 4 small specimens of urine during the race (40 ml, 55 ml, 30 ml and 50ml). The first 2 specimens contained 1 - 2 red blood cells/high-power field, the third was clear and the fourth specimen contained 7 - 10 red blood cells/high-power field. The next specimen, passed 1 hour after the race, was completely clear.

Competitor No. 12 did not pass urine during the race but the first specimen passed after the race contained 10 red blood cells/high-power field, and moderate proteinuria. Three hours later a second specimen showed similar results and 24 hours after the commencement of the race there were still 2 - 3 red blood cells/high-power field, and a trace of protein.

Competitor No. 13 passed only 20 ml of urine during the race which contained 1 - 2 red blood cells/high-power field. Six hours later the urine was clear but 24 hours after the commencement of the race urine contained 5 - 6 red blood cells/high-power.

Competitor No. 26 passed 440 ml of urine during the race containing 5 - 6 blood cells/high-powered field. After the race the findings were the same but 24 hours after the commencement of the race his urine was clear.

Competitor No. 29 passed urine twice during the race —25 ml and 35 ml. The first specimen was clear but the second contained 4 - 5 red blood cells/high-power field. After the race his urine was clear.

Competitor No. 31 passed two small specimens (60 ml and 50 ml) during the race. The first contained 20 - 30 red blood cells/high-power field and the second 5 - 6 red blood cells/high-power field. After the race his urine was clear.

Competitor No. 34 did not pass urine during the race. The first specimen after the race contained 5 - 6 red blood cells/high-power field and was subsequently clear.

Four of the 26 runners who only had one specimen of urine examined had more than 5 red bleed cells/high-power field in this specimen. The actual numbers of red blood cells/high-power field in these 4 runners was 80 -90, 55 - 60, 30 - 40 and 15 - 17.

Proteinuria

This was present in 70% of the runners, but in most of them there was only a trace. Twenty per cent had a moderate proteinuria and in 30% no protein was present in any specimen of urine. One-third of the runners still had a trace of protein when examined 24 hours later. There was a relationship between proteinuria and red blood cells in the urine, as 10 of the 11 competitors who had moderate proteinuria also had red blood cells in the urine, although in only 5 were there more than 5 red blood cells/high-power field. Twelve of 16 runners who had no proteinuria were free of red cells in the urine.

Urinary Specific Gravity

As could be expected the urine was concentrated either during or immediately after the race. Only 10 out of 53 runners had a specific gravity of less than 1·030 during or immediately after the race. These varied from 1·025 to 1·028. There was no relationship between concentrations below 1·030 and the presence of red blood cells or protein in the urine. Five of the runners with specific gravity below 1·030 had a blood urea estimation and 3 of these had blood ureas elevated above 50 mg/100 ml at the end of the race. (Only 2 other runners had blood ureas above 50 mg.) One of the other 2 who failed to concentrate to 1·030 had a renal shutdown during a previous marathon.

The urine remained fairly concentrated in most runners for 24 hours—only 2 had specific gravities of less than 1·022 at the end of that time and some were still above 1·030.

Casts and Pus Cells in Urine

During or after the race hyaline casts were present in 26% of the competitors' urine, and pus cells of more than 1/high-power field in 37%. All runners who had pus cells also had red cells in the urine. Two runners had granular casts, and these also had hyaline casts, pus cells and red cells of more than 1/high-power field. One had further urine examined which was normal.

Pus cells were the most common abnormality in the urine of runners examined before the race and were present in excess of 1/high-power field in 20% of the runners examined and 9% of these runners also had 1 - 2 red blood cells/high-power field. No runner had protein or casts in the urine before the race. Those runners with pus and red cells before the race were no more liable to have red blood cells or protein in the urine after the race and all had urine concentrations above 1·030.

Blood Urea and Electrolyte Changes

The electrolyte changes in these runners have been discussed more fully elsewhere.[2] The mean rise in blood urea among 31 competitors was from 27·1 mg/100 ml before the race (range 21 - 33 mg/100 ml) to 41·7 mg/100 ml after the race (range 30 - 60 mg/100 ml). There was no relationship between the presence of red blood cells in the urine and rise in blood urea after the race. In eight runners who had more than 5 red blood cells/high-power field, the mean rise in blood urea after the race was 14 mg/100 ml and among 7 competitors whose urine remained free of red blood cells the mean rise was 11 mg/100 ml. This difference was not significant. Changes in potassium levels before and after the race were not related to urinary changes.

Creatinine Clearance

The creatinine clearance was estimated in 6 runners 2 - 3 weeks after the race. Three of these runners (No. 3, 11 and 12) had passed urine containing more than 5 red blood cells/high-power field during or after the race. The post-race blood urea was only moderately elevated in these 3 (44 mg/100 ml, 43 mg/100 ml and 38 mg/100 ml) a rise from the pre-race levels of only 12, 17 and 11 mg/100 ml respectively. The creatinine clearance figures for these 3 runners were 115 ml/min, 92 ml/min and 97 ml/min respectively.

The 3 other runners were selected to have creatinine clearance studied because they had *post*-race levels of blood urea above 50 mg/100 ml—50 mg/100 ml, 50 mg/100 ml and 60 mg/100 ml. The differences between the levels and *pre*-race levels were 24 mg/100 ml, 23 mg/100 ml and 27 mg/100 ml among the highest. The creatinine clearance figures for these runners 3 weeks later was 92 ml/min, 79 ml/min and 90 ml/min. Competitor No. 19, the only one with a low creatinine clearance, was the only competitor who collapsed at the end of the race (his blood pressure could not be recorded for about 1 hour and he was admitted to hospital overnight). Five months later his creatinine clearance was 99 ml/min.

Intravenous Pyelograms

Intravenous pyelograms were performed in 4 of these competitors 3 weeks after the race—Competitors No. 17, 19, 29 and 31. As mentioned previously both No. 17 and 19 had post-race blood urea of 50 mg/100 ml, No. 29 had a post-race blood urea of 58 mg/100 ml—a rise of 36 mg from pre-race levels, the highest—and No. 31 had 20 - 30 red blood cells/high-power field after the race. His blood urea only rose 11 mg/100 ml.

In 3 competitors (No. 17, 29 and 31) the renal outlines were normal. There was good symmetrical excretion of contrast medium. The pelvicalyceal system, ureters and the bladder were normal and drainage from the kidneys was good. In Competitor No. 19 a small diverticulum was noted in the upper calyx of the left kidney and there was a slight delay in drainage from the left side. Other features of the intravenous pyelogram were normal.

Blood Pressure

Pre-race diastolic blood pressures of 100 mmHg or more were recorded in 6 competitors, 4 of whom were under 35 years old. After the race the blood pressure remained elevated in 2 runners, but dropped in the others. The pre-race urines in all these runners were normal except for one who had pus cells. Blood pressures in other competitors were normal.

The 6 runners with elevated blood pressure were no more liable to develop urinary abnormalities, elevations of urea or potassium, or electrocardiographic changes than the others. Only 1 (No. 12) had more than 5 red blood cells/high-power field in the urine, and another had moderate proteinuria. Two of these runners had an increase in the T-wave amplitude of greater than 5 mV, but no other abnormal electrocardiographic changes were recorded. All finished the race in good physical condition.

In most runners the blood pressure was moderately lower after the race, and in only one it could not be recorded.

Electrocardiograms

In 2 runners the amplitude of the P wave was doubled after the race but in the remainder it remained normal. One runner developed a right bundle-branch. Other electrocardiographic changes were confined to the T wave, and in nearly all of the runners this showed an increased amplitude. In 11 (40%) this increase measured 5 mV or more and had the symmetrical pointing suggestive of hyperpotassaemia (Fig. 1).
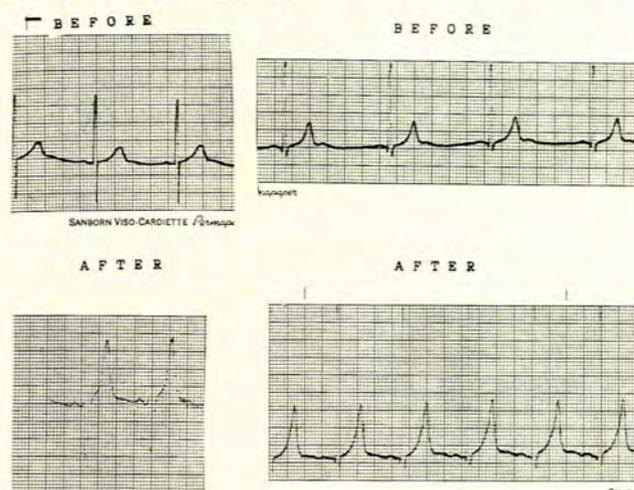

Fig. 1. Electrocardiograms before and after the race showing changes in T waves. In both runners the potassium increased 0·7 mEq/litre during the race (competitors No. 12 and 18).

The mean increase in serum potassium was 0·73 mEq/litre in the 11 runners who had T-wave increases over 5 mV compared with 0·54 mEq/litre in those whose T wave did not increase this much. In 5 runners whose T wave increased by 8 mV or more, the average serum potassium increase was 0·98 mEq/litre (Table I).

Serum Proteins

There was a slight but not significant drop in serum albumin after the race, which had no relation to proteinuria (Table II). We found no change in serum protein levels 2 weeks after the event, unlike McKechnie et al.[3,4] who noted a rise immediately after the race and a drop 2 weeks later.

DISCUSSION

Urinary abnormalities were first noted in marathon runners 60 years ago.[5] The incidence of proteinuria and red cells in the urine in these runners is much the same as has been found in participants in other sports including boxing, baseball, ice hockey, football, rowing, swimming, lacrosse and track events[6] (the incidence was actually higher among the last three, particularly long-distance races). The underlying renal pathology, its aetiology and significance are still uncertain.

There are a number of physiological effects following severe exertion which could predispose to renal abnormalities. Vasoconstriction of renal arteries occurs as part of the general splanchnic vasoconstriction enabling blood to reach muscles and skin in greater volume.[7,8] Ephedrine liberated as part of the stress associated with marathon running potentiates this effect.[9] As long as the renal circulation is not reduced to critical levels, glomerular filtration continues, but if dehydration is excessive then critical levels leading to anuria may be reached. In milder degrees of renal circulatory disturbance, ischaemic damage to glomeruli and tubules may be limited but sufficient to result in red blood cells, protein and casts appearing in the urine.

Castenfors,[10] who studied the problem of exercise pro-

TABLE I. ELECTROCARDIOGRAPHIC AND SERUM POTASSIUM CHANGES

Competitor No. Group with increase in T wave of 5 mm or more	T-wave amplitude (mm)			Serum potassium (mEq/litre)		
	Before race	After race	Difference	Before race	After race	Difference
1	3	8	5	3·7	4·2	0·5
6	2	8	6	4·3	4·5	0·2
7	8	17	9	4·1	5·5	1·4
11	9	17	8	3·7	4·4	0·7
12	4	17	13	4·4	5·1	0·7
13	7	14	7	4·4	5·2	0·8
19	5	19	14	4·0	4·7	0·7
23	3	9	6	3·4	4·0	0·6
29	9	17	8	3·9	5·3	1·4
31	8	15	7	5·4	5·9	0·5
33	5	10	5	3·8	4·3	0·5
						Mean 0·73
Group with increase in T wave of less than 5 mm						
3	5	5	0	3·9	5·0	1·1
5	2	2	0	4·0	4·5	0·5
8	2	4	2	3·5	4·8	1·3
9	4	6	2	3·9	4·3	0·4
15	2	4	2	4·0	5·0	1·0
16	5	7	2	4·2	4·6	0·4
17	15	15	0	3·7	4·0	0·3
24	7	9	2	3·6	4·0	0·4
25	3	6	3	4·3	4·3	0·0
26	4	5	1	4·0	4·3	0·3
34	2	6	4	4·1	4·3	0·2
						Mean 0·54

TABLE II. SERUM PROTEINS (g/100 ml)

Competitor No.	Before race			Immediately after race			Two weeks later		
	Total	Albumin	Globulin	Total	Albumin	Globulin	Total	Albumin	Globulin
1	7·3	4·4	2·9	6·8	4·2	2·6	6·7	4·2	2·5
5	7·5	5·3	2·2	7·6	4·3	3·3			
7	7·1	4·5	2·6	7·9	4·9	3·0	7·4	3·9	3·5
8	7·4	4·4	3·0	7·9	4·7	3·2			
9	7·3	4·6	2·7	6·8	4·2	2·4	8·0	5·2	2·8
11	7·3	4·6	2·7	7·3	4·0	3·3	7·5	4·5	3·0
12	7·6	5·0	2·6	7·3	3·8	3·5	6·7	3·7	3·0
13	7·5	5·0	2·5	7·2	4·2	3·0	6·9	4·3	2·6
16	7·3	4·3	3·0	7·3	4·3	3·0	7·4	4·2	3·2
19	7·4	4·6	2·8	—	—	—	—	—	—
21	6·5	4·5	2·0	7·5	4·3	3·2	6·7	4·3	2·4
23	7·3	4·6	2·7	6·5	3·8	2·7	7·6	4·6	3·0
24	7·6	5·2	2·4	7·1	4·4	2·7	7·3	4·7	2·6
25	8·0	5·2	2·8	7·6	4·0	3·6	6·9	3·5	3·4
26	7·5	4·8	2·7	6·5	3·8	2·5	7·1	4·7	2·4
31	7·8	4·8	3·0	7·3	4·7	2·6	—	—	—
32	6·5	4·5	2·0	—	—	—	—	—	—
33	7·3	4·4	2·9	7·9	4·5	3·4	—	—	—
34	8·0	4·8	3·2	7·6	5·0	2·6	—	—	—
3	—	—	—	—	—	—	7·3	4·3	3·0
Average	7·4	4·7	2·7	7·3	4·3	3·0	7·2	4·3	2·9

teinuria in detail, concluded that an increased glomerular permeability and the renal vasoconstriction following exertion were the main factors responsible and that there were individual differences in the increased permeability of the glomerular membrane following exertion.

Kleinman[11] demonstrated radiological changes in the kidney after exertion, particularly a distortion of the upper calyx of the right kidney which he considered to be possibly due to trauma of that kidney against the 12th rib, either directly from a blow, or from repeated and rapid changes of body posture. These changes predisposed to repeated urinary infection. He also considered that frequent forced crouching could lead to renal hypoxia through mechanical compression of vessels and viscera, although this would seem an unlikely mechanism in marathon runners. None of the 4 runners we examined radiologically had changes in the right kidney, and it is probable that the diverticulum in the left renal calyx observed in the one runner was not related to his marathon running.

Another factor in precipitating renal pathology is the high temperatures which occur during marathon running.[12] The abnormal renal histology in heat-stroke has been well documented.[13] None of these runners, however, developed temperatures in excess of 102°F and it was unlikely to have been a cause of abnormal renal function we observed in the runners. Heat-stroke was the probable cause of anuria in two competitors of previous Comrades Marathons and has occurred after army training.[14]

In none of these competitors could any disturbance in renal circulation be attributed to a central cause for collapse. The only competitor whose blood pressure could not be recorded at the end of the race had no urinary abnormalities although his blood urea was moderately elevated and his creatinine clearance was the only one which was low 3 weeks after the race. It returned to normal 4 months later. No other runner had any clinical signs of angina or cardiac collapse which might have disturbed the renal circulation, and the cardiograms were remarkable in that not one showed ischaemic effects.

There was no change affecting the J point or ST segment. The most frequent changes in these cardiograms after the race was the elevation in T waves. These were related to serum potassium increases.

Should these urinary changes be taken to indicate permanent renal damage and a reason to stop running? Any elevation of blood urea or potassium which occurred in these runners was not alarming, and was not related to the runners who had red cells or protein in the urine. Blood and urinary abnormalities were temporary in nearly all the runners. Only 2 had red cells in the urine after 24 hours although one-third still showed traces of protein. In the runners with more marked changes, creatinine clearance showed normal function when performed 2 weeks later in all but one runner, and he returned to normal 4 months later. No structural changes were noted in the 4 cases who had intravenous pyelograms.

There is, therefore, evidence that these renal abnormalities associated with marathon running are not serious. The renal histology in these cases has not been established, nor whether repeated renal damage could possibly lead to more long-lasting effects. We know of no evidence that long-distance runners are more liable to develop renal complications than others, but abnormal pyelograms have been seen in athletes who have had repeated haematuria.[11]

A tendency to repeated urinary infections has been found in boxers who had similar urinary findings to these runners.[11] Alyea and Parish[6] are inclined to take a more serious view of these urinary abnormalities occurring after effort. They suggest that anyone who has had acute renal disease—acute nephritis or pyelonephritis—should not take part in strenuous exercise.

There is probably an individual variation among athletes susceptible to renal damage after exertion, and those who frequently have haematuria would be well advised to stop severe exertion as permanent renal damage could occur. It would seem likely that the marathon runner who has had a single episode of haematuria is not at risk, but one cannot be absolutely certain of this.

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