SOME OBSERVATIONS ON THE HAEMODYNAMIC EFFECTS OF EXTRACORPOREAL HAEMODIALYSIS

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Disturbances of the circulation in patients with renal failure are frequent and their consequences are often fatal. Beside the cardiotonic effects of extreme hyperpotassaemia even less striking derangements of other electrolytes and their intracellular and extracellular relations result in an impaired heart contraction (Tomášek and Ježek, 1961; Tomášek and Johanovská, 1965). Uraemic oxalosis of the heart adds further aggravating factors contributing to arrhythmias, conduction disorders and heart failure in uraemia (Herles, 1965). According to our experiences with 1200 extracorporeal haemodialyses, the circulatory disturbances are the main hazard of the artificial kidney treatment. Extracorporeal circulation propelled by a pump, changes of the blood volume and rapid shifts of electrolytes are the very factors affecting the circulation of the treated patient. In spite of their practical consequences the haemodynamic effects of the extracorporeal haemodialysis are still poorly known. To date, there have been published only some partial reports on the behaviour of blood pressure and heart rate, venous pressure (Pippig, Heiland and Klütsch, 1965), blood volume (Nieth and Müller, 1958; Andreas, Murphy and Zipf, 1959; Lubash, Cohen, Braveman and Luckey, 1959; Marshall and Yoffa, 1965) and cardiac output (Del Greco, Sher and Simon, 1964).

In this article, we present the preliminary results of our studies on the haemodynamic effects of haemodialysis with a dialyser of a constant low volume in patients with chronic renal failure.

Materials and methods

Eleven extracorporeal haemodialyses were performed in three male patients suffering from chronic renal failure of different origin. One patient was anuric whereas the remaining two had normal diuresis. No signs of circulatory failure were present. A varying degree of anaemia was found in all instances.

The haemodialyses were performed with a plate-type artificial kidney (Skegg-Leonards) of Russian manufacture with a constant capacity of 350 ml and a dialysis area of 15,000 sq.cm. The extracorporeal blood flow rate was maintained at 200 ml/min by means of a membrane pump. The extracorporeal circuit was connected through an arterio-venous shunt in all but two instances, when a veno-venous junction was used. The duration of haemodialyses ranged from four to seven hours. The effects of ultrafiltration during dialysis were continuously balanced by an appropriate fluid intake according to the body weight. Haemodialyses were uneventful with merely slight variations of blood pressure and no vasopressor agents were used.

The estimation of the haemodynamic parameters was performed from 15 to 30 minutes before the dialysis and within 15 minutes after its termination.

Cardiac output of plasma was determined by the direct dye-dilution method of Stewart and Hamilton. Samples of arterial blood were taken at two-second intervals following a rapid
intravenous injection of 20 mg of Geigy blue. The concentration of the indicator in the blood samples was estimated with a Zeiss spectrophotometer.

The plasma volume was determined from the arterial blood samples drawn 10 and 15 minutes after the injection of the same indicator.

The cardiac output of whole blood and the blood volume were calculated from the arterial haematocrit and from the cardiac output of plasma and plasma volume respectively.

The stroke volume was calculated as a ratio of the cardiac output and of the heart rate. The heart rate was determined simultaneously with the sampling of blood for the purpose of the dilution curve. The blood pressure was measured indirectly with a mercurial manometer.

The peripheral vascular resistance was calculated from the usual equation (Apéria, 1940).

Plasma-potassium and plasma-sodium were determined with a flame photometer; potassium in erythrocytes was estimated by the method of Lans.

Results

The mean values of the parameters gained from eleven explorations in three patients were used for the analysis and resultant evaluation.

The effectiveness of the haemodialyses was characterised by a significant decline of non-protein nitrogen (150-78 mg\%\_\_; p < 0.001) and plasma-potassium (6.13-4.67 meq/L; p < 0.001). Plasma-sodium, and potassium in the erythrocytes, revealed no significant change (Table I).

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<th>TABLE I</th>
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<td>Mean</td>
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<tr>
<td>Plasma NPN mg%__</td>
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<tr>
<td>Plasma K meq/L</td>
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<td>Plasma Na meq/L</td>
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<td>Erythrocyte K meq/L</td>
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Before dialysis, in comparison with the control group, the plasma-proteins (6.0 mg\%\_\_) and the haematocrit (25\%), were diminished. The blood volume was markedly expanded (6104 ml) owing to an augmented plasma volume (4913 ml). After dialysis only insignificant changes of the mentioned parameters occurred (Table II).

<table>
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<td>Mean</td>
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</tr>
<tr>
<td>Body weight kg</td>
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<tr>
<td>Plasma proteins g%__</td>
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<td>Hematocrit %</td>
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<td>Plasma volume ml</td>
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<td>Blood volume ml</td>
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The following haemodynamic values were found before dialysis (see Table III): the heart rate was almost within normal limits (76/min), the cardiac output (7830 ml/min) and the stroke volume (110 ml) were markedly augmented. The peripheral vascular resistance was slightly lowered (1331 dyn sec cm\^-2).
After dialysis the heart rate invariably increased to a marked tachycardia (76-97/min; $p < 0.001$). The cardiac output significantly rose (7830-9090 ml/min; $p < 0.02$) and the stroke volume revealed a significant drop (110-96 ml; $p < 0.01$), remaining still augmented. An insignificant decrease of the peripheral vascular resistance appeared.

A similar haemodynamic response was observed in the course of the extracorporeal haemodialysis where the determinations of the same parameters were done also after the first and the third hour of its duration (Fig. 1).
After the first hour of dialysis the initially elevated cardiac output revealed a further marked increase (7708 – 11261 ml/min). The high stroke volume maintained its original level (116.8-117.3 ml). The rise of the cardiac output was due to tachycardia (76-96/min).

After the third hour the cardiac output returned approximately to its predialysis level (7640 ml/min) and the stroke volume underwent a marked fall (81.3 ml). Tachycardia persisted (94/min).

After the fifth hour (at the end of dialysis) a slight increase of the cardiac output took place exceeding the initial predialysis level (8494 ml/min). A moderate increase of the stroke volume occurred yet the final postdialytic decrease was apparent (92.3 ml). Tachycardia was unaffected (92/min).

Only moderate variations of the blood pressure, the venous pressure in the inferior vena cava, the blood volume and the body weight developed in the course of the dialysis.

Comments and conclusions

The rise of the heart rate and excursions of the blood pressure during haemodialysis are a common feature and they can be traced in almost all reports on artificial kidney treatment wherever these parameters are mentioned. In patients with chronic renal failure in whom the state of the circulation before dialysis is often altered the haemodynamic response to the artificial kidney treatment easily exceeds the safe physiologic limits. Despite the fact that haemodialysis can improve the general circulatory state by abolishing the uraemic intoxication (Alwall, 1958), the failure of the circulation remains the leading risk of the artificial kidney treatment. Yet, the mechanisms and causes of these life-endangering circulatory derangements have been only poorly studied and are not fully understood. Recently Del Greco and colleagues reported on the changes in the cardiac output in the course of dialysis in a group of uraemic patients a greater part of which revealed signs of congestion and of impaired cardiac function. A significant increase of the cardiac output was found in patients undergoing uneventful dialyses whereas in patients requiring vasopressors during the procedure a marked decrease of the cardiac output was observed. In the opinion of these authors, the postdialysis rise of the originally normal cardiac output suggested improved cardiac function. The fall of the cardiac output was ascribed to a failure of the myocardium to cope with the haemodynamic burden induced by dialysis.

In our series of patients with chronic renal failure the cardiac output was markedly augmented already before dialysis, due particularly to a high stroke volume. Advanced anaemia together with the raised diastolic filling pressure due to the blood volume expansion is presumed to be responsible for the high cardiac output and stroke volume. After dialysis a significant rise of the initially augmented cardiac output was observed. Tachycardia was the underlying cause of this reaction. The stroke volume decreased after dialysis in all instances. The blood pressure, the blood volume and the anaemia revealed only insignificant variations and neither of them could be held responsible for the observed postdialysis haemodynamic changes.

In several separate studies in the course of haemodialysis the cardiac output revealed a remarkable increase already after the first hour of dialysis. In this phase the originally high stroke volume was still unaffected and tachycardia was the dominant feature. We suppose that the haemodynamic reaction observed in the initial phase of dialysis reflects particularly the adaptation of the circulation to the sudden load of the extracorporeal circuit.

In later phases of dialysis tachycardia persisted, the stroke volume diminished and the cardiac output decreased from its peak level though remaining usually above the predialysis level. The reasons for the decreasing stroke volume are not quite clear. Since other factors, such as the diastolic filling pressure, the arterial blood pressure and the peripheral vascular
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resistance, which can affect the stroke volume, did not change during the dialysis, the cause of the decreased stroke volume is probably the impaired contraction of the heart. Marked shifts of electrolytes, especially those of potassium, and the variations in their gradients across the myocardial cell membrane which appear particularly in the later period of the haemodialysis might play an important role in the progressive deterioration of the myocardial contraction, a decreased stroke volume being the result.

Thus, in contrast to the opinion of Del Greco and colleagues, we do not suppose that the rise of the cardiac output during dialysis always reflects an improved cardiac function or that it represents a more favourable circulatory state. According to our experience the immediate consequence of haemodialysis is a less advantageous haemodynamic situation in which the augmented cardiac output is maintained through tachycardia whereas the stroke volume diminishes. This presumption does not deny the possibility of later favourable circulatory effects of the haemodialysis which can be gained after the improvement of the metabolic derangement and after the restitution of the electrolyte relations at a more physiologic level.

Summary

The circulatory effects of the extracorporeal haemodialysis with a dialyser of a constant low volume (Skegg's-Leonards) with a pump were studied. In patients with chronic uraemia in which no signs of a circulatory failure were present different haemodynamic parameters were estimated before and after dialysis.

A markedly augmented cardiac output due to a high stroke volume was found in the predialysis investigation. After dialysis the cardiac output revealed a further increase at the expense of tachycardia whereas the stroke volume diminished. A similar haemodynamic response was observed also in patients undergoing serial examinations in the course of dialysis. The mechanisms and causes of the observed haemodynamic features are discussed.

REFERENCES


Herles, F. (1965): The heart in uremic oxalosis. Cor et Vasa, 6, 203.


DISCUSSION

The Chairman: Thank you, Dr. Valek. Any comments from the audience?

Unidentified Member: Some years ago we measured blood pressure in patients and dogs in severe uraemia and less severe uraemia, and we have seen that dogs not walking but lying on the table do not have a rise in pulse-rate and a fall in blood pressure; less severely ill dogs that are able to walk on the table have a rise in pulse-rate but no drop in blood pressure.

In the same way, we have seen that when we dialyse severely uraemic patients, we do not have a rise in pulse-rate.

The Chairman: What is your explanation?

Unidentified Member: That, in severe uraemia, there is no possibility of a rise in pulse-rate and therefore we have a drop in blood pressure.

Comty (London): Do you think you are getting pyrogenic reactions during the first few hours of dialysis?

I do not know how much ultrafiltration you are performing, during dialysis. But don't you think that this fall in cardiac output could be due to reduction in blood volume rather than an effect on the myocardium?

Valek (Prague): We did not ultrafiltrate during the dialysis.

Kerr (Newcastle): Pyrogenic reactions seem to me the obvious explanation.

Valek (Prague): No, we did not have pyrogenic reactions.