BLOOD PRESSURE CONTROLLED BY LOW REACTIVE LEVEL DIODE LASER THERAPY (LLLT)

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The effects of low reactive level laser therapy (LLLT) with an infrared diode laser on blood pressure, particularly the hypotensive effect on hypertension were studied. Essential or primary hypertension is frequently encountered, but its aetiology and consistent control elude present day medicine. Experimental animal studies have shown that hypertension causes destructive changes in the medulla oblongata and in the brain stem: arteriosclerosis, and destructive changes in brain stem cells have been demonstrated in hypertensive human patients. Various biological effects on vascular tissue have been reported for the diode laser, and so the author evaluated the GaAlAs diode laser for the treatment of hypertension by controlling the blood pressure regulatory system, irradiating the area adjacent to the medulla oblongata. Thirty patients were included in the study. Following treatment the results were graded as excellent in 6 patients (20%); good in 11 patients (37%); fair in seven cases (23%), and ineffective in 6 cases (20%), giving an overall effective rate of 80%. In 12 of the 30 patients, whose hypertension had not been effectively controlled by conventional hypotensive treatment, LLLT gave excellent results in 6, good in 5 and fair in 1 patient. In contrast, in a second group of 15 relatively normotensive control patients, there were no excellent or good results, and only 3 fair results, the remainder being graded as ineffective. It was concluded that in general there was no clear-cut hypotensive effect following LLLT with the diode laser, but when applied to the pathological condition known as essential hypertension, LLLT was noticeably effective. These findings warrant further study on this application of LLLT.

KEY WORDS Essential hypertension LLLT Laser bioactivation Hypertension control

Introduction

Hypertension is one of the major diseases of the present day, with a high rate of mortality. Hypertension can be divided into essential primary and symptomatic secondary types, as shown in Table 1.¹ Essential hypertension remains a mystery as far as its causative factor, and very often fails to respond to conventional medical therapy. Secondary symptomatic hypertension is easier to control, by treating the underlying disease or abnormal condition. Figure 1 shows the World Health Organisation's criteria for the classification of hypertension: normal is classified as being below a systolic/diastolic range of 140 over 90. From 140 over 90 to 160 over 95, the classification is marginal, and patients with blood pressure above 160 over 95 are classed as frankly hypertensive.

Table 2 lists the main hypertensive factors. Increased activity of the sympathetic nervous system increases cardiac output, while simultaneously caus-

ing vasoconstriction of distal arteries: this combination raises the pressure of the blood, and hypertension is the result. Renin is an enzyme formed in the kidney, and released into the blood stream. It is an important component in the formation of angiotensin, a powerful pressor agent. Excess renin synthesis can lead to spasmodic arterial vasoconstriction, which in turn causes hypertension. Sodium chloride, or common salt, is extremely hygroscopic. Excess intake of NaCl therefore causes fluid retention in the body, thus increasing the volume of body fluids, causing an increase in blood pressure. Aldosterone is a potent electrolyte-regulating hormone secreted by the adrenal gland. Excess aldosterone causes enhanced sodium retention in the body, leading to an increase in body fluid retention as for sodium chloride, with the same end result. The kidney contains a number of hypotensive agents, such as members of the prostaglandin group, thus a decrease in the synthesis of these substances which occurs with reduction of kidney function will lead to hypertensive changes. Neural factors, blood vascular system factors, endocrinous factors, factors within the kidney itself all exist to control the blood pressure, together with other reported blood press-

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Table 1. Essential and symptomatic hypertension

Essential Hypertension (Primary)

Symptomatic Hypertension (Secondary)

- A. Nephritic influences on blood pressure
 - Acute/Chronic
 - Glomerulonephritis
 - Acute pyelonephritis
 - Cystic nephritis
 - Nephritis with diabetes mellitus
 - Collagen disease nephritis
- B. Effects from kidney blood vasculature
- C. Endocrinous influences on blood pressure
 - Primary aldosterone disease
 - Cushing's disease
 - Brown cell tumour
 - Renin-producing tumour
- D. Miscellaneous others

Figure 1. WHO classification of blood pressure ranges

Table 2. Blood pressure hypertensive factors

- A. Acceleration of activity of sympathetic nervous system
- B. Endocrinous acceleration of renin synthesis
- C. Hyperuptake of salt (NaCl)
- D. Endocrinous acceleration of electrolyte corticoid
- E. Defect in kidney hypotensive function

ure control substances. All of these factors are in turn controlled by the central nervous system;² any irregularities in the latter will naturally lead to hypertensive changes. All blood vessel walls are under vasoconstrictive sympathetic neural control. Sympathetic system control also exists over the carotid sinus and the aortic band in the vessel wall. It can therefore be said that these areas constitute pressure receptors.¹ A build-up of pressure over

normal will be felt by these receptors as they physically expand, and a signal is sent to the central nervous system master control system, which routes it to the centre of the pons and brain stem, where the blood vascular system pressure control complex is housed. Signals are sent to the heart via the parasympathetic vagus nerve, the pulse rate is reduced, and blood vessels are dilated, thus lowering pressure to normal. The opposite occurs when low pressure is detected by the sensors. Pulse rate is increased, sympathetic system-mediated vasoconstriction of blood vessels simultaneously occurs, and blood pressure is raised.³ From this it can easily be seen that any disturbance of CNS function will greatly affect hypo- and hypertension control factors. The above system is schematically represented in Figure 2.

There are many known hypertensive factors, and even more which to date remain unclear. In this trial, it was decided to concentrate treatment on the central nervous system, in particular paying special attention to the pons and brainstem. Accordingly, in the present study, the diode laser was applied to the occipital area opposite the pons, concentrating treatment in the area extending from the upper cervical vertebrae up to the occiput.

Materials and Methods

The patient population for the study consisted of 30 patients diagnosed as having essential hypertension, ages ranging from 37–81, 12 male and 18 female patients with mean ages of 65 and 64.6 respectively. A second group of comparatively normotensive patients was also treated in the same manner, as a control, to study the possible effects of LLLT on a normally-operating blood pressure control system.

The chosen target area was the centre of the back of the upper neck, just below the occiput, angling the laser slightly upwards. This was chosen to involve as much of the blood pressure motor control centre as possible and associated afferent and efferent nerve fibres.

The laser used was the OhLase-3D1® (Proli Japan), a gallium aluminium arsenide (GaAlAs) system, giving a continuous wave output power of 60 mW, at a wavelength of 830 nm in the near infrared. The system was used with the hand-held probe in light contact with the target area for 120 s per treatment session. The range of treatment sessions was from 4 to 12, with a mean of 8.

Patients were made as comfortable and relaxed as possible before treatment to help eliminate any adverse psychic factors, and their blood pressure was measured. The laser was applied as above, and soon after irradiation, blood pressure was taken again. The pre- and postirradiation results were then compared. Evaluation of treatment effectiveness was divided into four levels depending on the degree

Figure 2. Schematic representation of control circuit of the blood pressure motor centre. Note the complete feedback loops between the control centre in the pons, the various pressure receptors, and the heart and blood vessels, allowing for both blood vessel vasoconstriction (sympathetic system control) to raise blood pressure, and vasodilation (parasympathetic control) to lower blood pressure, coupled with vagus nerve influence directly on the heart

of reduction of systolic and diastolic pressures, as seen in Table 3: a reduction of the systolic pressure of more than 20 mm Hg, together with a 10 mm Hg reduction in the diastolic reading was rated 'excellent'. If one but not both of these levels of reduction was achieved, then the result was graded 'effective'. A 10–19 mm Hg systolic reading reduction together with a 5–9 mm Hg diastolic level

reduction was graded as 'fair'. Values of less than the above, or no change, were graded as 'ineffective'.

Results

The results are summarized in Table 4 and Figures 3 and 4 (hypertensive subjects) and Table 3, Figure 5 (normotensive control subjects). Of the 30 patients, 6 (20%) had an excellent result, 11 (37%) had an effective result, 7 (23%) were graded fair, and the remaining 6 (20%) were graded as ineffective. In other words, 24 (80%) out of the 30 patients had an effective treatment result. These patients were all on hypotensive control drugs. The 6 'ineffective' patients were actually within the WHO 'normal' guidelines. Of the 24 effectively treated patients, 12 were not responding well to the drugs. Of these 12, 6 cases were excellent, 5 effective, and 1 fair. In these difficult-to-treat cases, LLLT gave a 100% effective rating compared to conventional pharmaceutical control.

The control group of 15 normotensive patients was composed of 6 males and 9 females, with ages ranging from 26–77, (average ages: males–55, female–47). As seen from Table 3 and Figure 5, only 2 were graded as fair, with the rest ineffective. However, it is interesting to note from Table 3 that in some cases, what could be graded as hypotensive conditions were actually improved: in other words

Table 3. Criteria used in evaluation of laser therapy

Excellent (+++):	Reduction of over 20 mm Hg in systolic BP <i>plus</i> Reduction of over 10 mm Hg in diastolic BP
Effective (++):	Reduction of over 20 mm Hg in systolic BP <i>or</i> Reduction of over 10 mm Hg in diastolic BP
Fair (+):	Reduction of from 10–19 mm Hg in systolic BP <i>plus</i> Reduction of from 5–9 mm Hg in diastolic BP
Ineffective (–):	Reduced levels less than above, or no reduction at all

Figure 3. Line graph representation of pre- (left of line) and post- LLLT (right of line) blood pressures for first group of 15 treated hypertensive patients, corresponding to top group in Table 4. The post LLLT value was taken almost immediately after irradiation

Figure 4. Line graph representation of pre- (left of line) and post- LLLT (right of line) blood pressures for second group of 15 treated hypertensive patients, corresponding to bottom group in Table 4. The post LLLT value was taken almost immediately after irradiation

Figure 5. Line graph representation of pre- (left of line) and post- LLLT (right of line) blood pressures for control group of 15 normotensive treated patients, corresponding to group in Table 5. The post LLLT value was taken almost immediately after irradiation

LLLT had an opposite hypertensive effect in hypotensive subjects.

Discussion

When used to treat essential hypertension, the effect of LLLT is very clear. Animal experiments have

demonstrated pons and brainstem cell damage as sequelae of hypertension, and evidence exists of similar brainstem cellular damage, and arteriosclerosis in hypertensive humans. It is considered that irregularity of the vascular motor control centre in the pons is the main cause of essential hypertension, however the data of the present study point to LLLT as being an effective hypotensive agent in

Table 4. Results and effective rate of diode laser on hypertension in 30 patients

Age	67	82	67	45	70	69	78	62	72	77	81	66	70	72	37
Sex	F	F	M	M	F	F	M	F	F	F	M	F	F	M	F
blood pressure before treatment (Sys/Dias)	190/120	134/68	144/90	172/100	156/90	140/82	138/88	176/90	142/76	120/80	160/90	186/90	150/84	152/90	146/88
blood pressure after treatment (Sys/Dias)	170/80	108/60	144/84	150/90	130/82	140/76	124/82	152/80	136/60	120/72	160/86	156/86	140/70	140/84	118/82
Effectiveness	+++	++	-	+++	++	+	+	+++	++	-	-	++	++	+	++
Age	50	58	71	53	82	70	59	76	67	66	56	77	48	50	61
Sex	M	F	F	M	F	M	F	F	M	F	F	M	F	M	M
blood pressure before treatment (Sys/Dias)	178/108	150/90	140/80	152/110	158/92	150/88	136/74	168/90	144/90	140/86	160/80	120/72	130/90	148/80	146/86
blood pressure after treatment (Sys/Dias)	156/90	130/84	124/76	130/90	140/80	134/70	130/70	140/72	125/84	126/80	142/70	120/70	124/82	126/84	140/80
Effectiveness	+++	++	+	+++	++	++	-	+++	+	+	++	-	+	++	-

Table 5. Control patient population and results of diode laser therapy

Age	48	50	56	50	77	49	48	50	51	26	56	45	43	29	38
Sex	M	M	M	M	M	F	M	F	F	F	F	F	F	F	F
blood pressure before treatment (Sys/Dias)	128/78	136/86	130/96	132/88	110/72	140/86	100/64	92/50	92/60	110/68	90/40	104/50	100/58	102/64	114/70
blood pressure after treatment (Sys/Dias)	120/72	120/78	128/88	120/80	120/80	128/80	102/60	96/54	100/70	120/80	90/54	104/60	88/58	108/66	110/88
Effectiveness	-	+	-	+	-	+	-	-	-	-	-	-	-	-	-

cases of essential hypertension, when used as discussed on the area opposite the pons.⁴ The pons-vago motor disturbance theory is certainly a reasonable assumption for one of the possible causes of essential hypertension, and the application of the diode laser in LLLT on the area of the pons has showed in this trial good results.

Further controlled studies should be carried out with other treatment techniques and wavelengths, including double-blind studies, before LLLT can be hailed as a definitive treatment for essential hypertension, in order to lower blood pressure, and protect against brain, heart and kidney damage. However, the results of the present study point to the possible effectiveness of LLLT with the diode

laser for this puzzling and often difficult-to-control condition.

References

1. Ebihara, A. (1976). Physiologic circulation for the clinical doctor, in *The Mechanisms of Hypertension*. Shinko Koeki, Tokyo. p. 293 (in Japanese).
2. Guyton, A. C., Coleman, T. G., Cowley, A. W. Jr. and Ferguson, J. D. (1974). A systems analysis approach to understanding long-range arterial blood pressure control and hypertension. *Circulation Research*, **35**, p. 159.
3. Lewis, R. K., Harcrig, C. G., Fricke F. J. and Russel, R. O. (1972). Therapy of idiopathic essential hypertension. *Archives of Internal Medicine* **129**, p. 943.
4. Pickering, G. K. (1968). *High Blood Pressure*. Churchill, London.