DECOMPRESSION SICKNESS DURING THE SINKING OF A CAISSON

A STUDY OF SOME FACTORS IN THE PATHOGENESIS OF CAISSON DISEASE

BY

H. E. LEWIS and W. D. M. PATON

From the Department of Physiology, University College, London, and the Department of Pharmacology, Royal College of Surgeons, London

(RECEIVED FOR PUBLICATION MAY 25, 1956)

Most of the studies of decompression sickness in industry have been made during the construction of tunnels. The sinking of a caisson, however, is a much briefer undertaking involving certain special features not encountered in tunnel work. Work of this type has received relatively little attention in recent years, although caisson sinking is performed all over the world. This paper describes an investigation into the incidence of decompression sickness during the sinking of reinforced concrete caissons in the Thames between February and May, 1950.

The engineering problem was to build aqueducts to carry river water to a power station for cooling the turbines. The heated water had to be discharged by another outlet aqueduct down stream, well away from the inlet, so that the cooling efficiency would not be reduced by sucking in water already warmed by the turbines. The aqueducts were about 7 ft. in diameter. There were two stages in the construction of each aqueduct: first, caissons were sunk just below low water line in the river; tunnels were then driven out to join them. The aqueduct was completed so that its opening was well above the silt of the river bed. During the sinking the earth was removed by buckets and hoisted up through a “muck lock”. The air pressure in the caisson varied according to its depth below water-level, and was maintained to prevent water or mud rising into the caisson before it was sealed with concrete. The maximum pressure used was about 35 lb./sq.in., though it varied by 10 lb./sq.in. or more with the tide (Fig. 2). The working shift (usually four or five men, sometimes as many as eight) entered and left the caisson by a blister lock in which compression and decompression took place. They reached the caisson bottom by means of a vertical ladder. There were usually three eight-hour shifts. (See Fig. 1 for a diagram of the caisson.)

Methods

Data about cases of decompression sickness were obtained principally from the medical orderlies; they were always on the spot and were responsible for supervising the recompression in the medical lock. Records of all compressions were supplied by the engineer’s office.

Measurements were made of the carbon dioxide concentrations in the air in the blister lock of the caisson, during and at the end of a normal decompression. Since this lock is completely filled when it contains five men, there would have been no room for an observer and gas analysis apparatus in it. Accordingly arrangements were made to attach a length of hose to the outlet pipe, from which samples of the air escaping from the chamber during decompression were taken and sealed over mercury. It was thus possible to get estimates of the composition of the air in the decompression chamber while it was being used in the normal way, fully packed with men coming off shift. When a sample was taken another observer recorded the gauge pressure at the time. The oxygen and carbon dioxide were estimated by means of a Haldane gas analysis apparatus.

Results

The general course of the undertaking is shown in Fig. 2, in which are represented the mean weekly pressures under which the work was done, and the weekly incidence of bends. The main part of the figure is concerned with the inlet caisson; the right side of it includes the outlet caisson as well. In the middle of the undertaking work ceased for a period of three weeks. Over the whole period in which the pressure exceeded 18 lb. per sq. in. there were 79 cases of bends affecting 37 men in a total number of 1,800 compressions at the inlet caisson; at the outlet
Fig. 1.—Diagram of caisson, blister lock, access staging, and procedure for sampling air in lock during decompression.
SOME FACTORS IN PATHOGENESIS OF CAISSON DISEASE

Inlet
Outlet

WEEK NUMBER

INLET

OUTLET

WORKING PRESSURE (lb./sq.in.)

PERCENTAGE

INCIDENCE OF BENDS

WEEK NUMBER

FIG. 2.—Weekly incidence of bends (as % of all compressions) and working pressures (lb./sq.in.). Open circles, inlet caisson, filled circles, outlet caisson.

caisson there were another 10 cases in 310 decompressions. This gives an overall bends rate of about 4%. As is almost always observed in this type of undertaking, the bends rate was far from uniform over the whole course of the work. It only began to rise when the pressure exceeded 20 lb. then rose to a high level for a brief period, and tended to fall away thereafter, even though the pressure was maintained. This almost “epidemic” character of decompression sickness is a very common feature.

The bends, as is usual, consisted of aching pains most commonly affecting the joints. The legs were the most frequently affected, and next the arms; also affected were the knees, wrists, shoulders, head and the body in general. The bends were usually bilateral. The distribution of these symptoms is shown in Fig. 3.

There was the usual tendency for a few individuals to be repeatedly victims of mild bends, and two men had as many as six attacks each. Table 1 shows how the incidence was distributed.

In view of the rather high incidence of bends, particularly in the shift workers at special times, an attempt was made to define whether there were any special circumstances predisposing to decompression sickness. Four conditions seemed to present themselves:

(1) Faulty Decompression.—When accuracy of decompression was investigated, it was found that the decompression times were not always strictly adhered to. The decompression shown in Fig. 4a, performed during the test for vitiation of the air in the decompression lock, serves as an example. The lock pressure should have been lowered in about two minutes from 27 lb. to 6 lb. per square inch, and thereafter brought to atmospheric pressure during a further 30 minutes. In fact, the pressure fell nearly uniformly throughout, in a time slightly shorter than the total laid down for the decompression.

<table>
<thead>
<tr>
<th>FREQUENCY OF ATTACKS OF BENDS IN DIFFERENT INDIVIDUALS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Attacks per Man</td>
</tr>
<tr>
<td>---------------------------</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>6</td>
</tr>
</tbody>
</table>
Fig. 3.—Frequency of bends in various parts of the body (hatched columns indicate bilateral involvement).

(2) Vitiation of Air by Carbon Dioxide during Decompression.—Also shown in Fig. 4b is the rise of carbon dioxide percentage during the progress of the decompression. It has also been drawn in terms of the absolute tension of carbon dioxide, since it is this that is physiologically important. The absolute tension reached a maximum of 2-63% atmosphere after the decompression had been proceeding for 18 minutes. Thereafter it fell, although the percentage of carbon dioxide in the air in the lock continued to rise. The differing time course of the two measures of carbon dioxide is due, of course, to the changing lock pressure.

It is possible to make an approximate calculation from these figures of the total carbon dioxide production during the period of decompression. In doing this, it is necessary to include in the calculation the carbon dioxide lost in the gas vented from the chamber. When this calculation was made, knowing the capacity of the chamber to be 69 cu.ft., and allowing about 10 cu.ft. for the volume occupied by the five men themselves, we obtained a carbon dioxide production of approximately 0.51 per minute, corresponding to an oxygen consumption of about 0.6 l.

(3) Exercise.—It was found during the last war that vigorous exercise was a factor precipitating decompression sickness in aviators (Cook, 1951). The evidence for this is much weaker for decompression sickness resulting from exposure to high pressures, but it remains a possibility. It has been noticed in diving experience, for instance, that bends tend to occur in those muscles which have been most heavily worked during exercise. We believe it to be significant, therefore, that at the end of the shift the men had to climb a 70 ft. vertical ladder in order to reach the decompression lock. This ascent was usually made as fast as possible, and there was no waiting period before decompression began. Thus the decompression took place immediately after severe exercise, and this exercise may well be an important contributory factor to the incidence of bends.

(4) Chilling.—During the work at the bottom of the caisson the men are usually exposed to considerable heat. This is particularly the case on a warm day, or when concreting is done, since the setting of concrete is an exothermic reaction. On the other hand, when they come into the decompression chamber, the fall in pressure during decompression is associated with a considerable cooling of the atmosphere in which they are placed. This means that not only is the air around them cold and humid, but there is considerable condensation of moisture on their clothes, so that these, already damp, may become thoroughly wet and chilly. Experienced workers sometimes keep a set of dry clothes in the decompression chamber into which they change while waiting. The medical orderlies recorded the impression that men who were not careful about dry clothes or who found the decompression uncomfortably cold were those who seemed to be liable to decompression sickness.

Discussion

In many respects the decompression sickness observed during this undertaking resembled that seen during the construction of a tunnel (Paton and Walder, 1954). Thus it had an "epidemic" character; individuals varied considerably in their susceptibility; and the incidence in different parts of the body was more or less the same as that observed in tunnel work. On the other hand, the incidence was somewhat higher than has been recently recorded in tunnel work. It is probably important to recognize certain differences between the two types of undertaking. In the first place, the construction of a
SOME FACTORS IN PATHOGENESIS OF CAISSON DISEASE

Fig. 4(a).—Time course of fall of pressure in blister decompression lock actually observed (open circles) and prescribed by decompression tables (filled circles).

Fig. 4(b).—Carbon dioxide percentage (filled circles) and absolute tension in % atmosphere (open circles), during the decompression of five men shown in Fig. 4(a).
tunnel is usually a fairly lengthy operation. This allows time for the labour force to acclimatize to the work, for susceptibles to be weeded out, and for all the local details of an efficient decompression procedure to be developed. On the other hand, tunnel work may be more continuous and arduous (at least at the working face) than in a caisson, where there are pauses for the hopper to be hauled to the muck-lock, emptied, and returned. One gains the impression from veteran compressed air workers that bends used to be less of a problem in caisson work than in tunnel work; this might well be due to the existence, before the war, of a useful group of experienced and resistant workers developed by the various tunnel contracts of that time, for whom caisson construction would present no novelty. But with the suspension of tunnel-making during and immediately after the war, this cadre of experienced personnel must have disappeared. One would expect, therefore, that for some years the average bends incidence in short-term compressed air undertakings would be higher than normal, simply because there is no pool of relatively resistant individuals on which to draw.

The absence of a pool of insusceptible individuals does not, however, account satisfactorily for bends rates in excess of 2%. Decompression tables are intended for general use, and should be suitable even with inexperienced workers. For this reason, we have considered whether there are any processes involved in caisson sinking which might make the worker under these conditions relatively vulnerable. Four possible factors emerged.

1) Inaccuracy in Decompression.—The first was simple inaccuracy in decompression. This is a perennial difficulty with all compressed air working, and the chances of securing a sufficiently experienced, competent, and authoritative lock-keeper are usually fairly small, particularly for a relatively brief contract. He has to have a thorough understanding of his responsibility and the rationale of the procedures; but he also needs a personality strong enough not to be influenced by those inside the lock. It is not infrequent, for instance, that eagerness to go to a football match, or to collect and spend the week’s pay, leads to a severe outbreak of bends. To prevent over-rapid decompression, the best safeguard is to have a procedure which lays down a definite decompression procedure out of control of the lock-keeper, such as that used in Newcastle, where fixed jets were attached to the outlet pipe. It is comparatively easy to install this system in a tunnel where there is a relatively constant working pressure, but it would be more complicated for caisson work where the pressure is always altering.

A safeguard of a different type is a recording barograph attached to the chamber and secured by lock and key, which provides a permanent record of all the decompressions conducted. This enables examination of decompression procedures at leisure, and permits their correlation with any bends which subsequently occurred.

2) Vitiation of Air.—Vitiation of the air with carbon dioxide during the decompression in the small blister lock appears to be a contributory factor not hitherto recognized. It is known that carbon dioxide can contribute to the genesis of decompression sickness (Cook, 1951). It probably does this by assisting the initiation of the bubble which then grows by the later ingress of nitrogen from the supersaturated tissues. Usually, in tunnel work or in diving or aviation experience, the chambers are of such a size that there is no risk of accumulation of carbon dioxide. The blister lock of a caisson, however, is so small in relation to the number of men crammed into it that significant amounts of carbon dioxide can accumulate. In our observations the highest concentration achieved was about 2-6% of an atmosphere.

But the further question arises as to what this carbon dioxide percentage would have been if the decompression had been properly conducted, instead of having been, as was the case, too slowly falling in the early stages. This is a difficult problem to deal with precisely, since the situation is one whereby the amount of carbon dioxide lost from the blister depends simultaneously on the rate of carbon dioxide production, and on the rate of pressure fall. But an approximation can be obtained quite simply under the present circumstances. In general, if the pressure falls by an escape of the contents of the chamber, instantaneously to (say) half the initial absolute pressure, then the tension of any gas in the mixture of gases comprising the chamber’s atmosphere will also fall by half. Similarly if the absolute pressure falls to \( x \)\% of its original absolute pressure then the tension of carbon dioxide will fall to \( x \)\% of the original value. Now suppose that a major pressure fall takes place right at the beginning of decompression. In this particular case, suppose that the decompression was correctly done and the pressure fell within two minutes from 27 lb. to 6 lb. Now at this period CO\(_2\) can hardly have accumulated; thus virtually no carbon dioxide will be lost from the chamber. The only venting of carbon dioxide will be that achieved later in the further fall of pressure by 6 lb. over 30 minutes. What effect will this gradual loss have on the final carbon dioxide tension? It can be roughly estimated as producing a result halfway between the effects of two extreme


SOME FACTORS IN PATHOGENESIS OF CAISSON DISEASE

procedures, viz., a fall of 6 lb. rapidly at the beginning of the 30 minutes' period, which could not lead to the loss of any CO₂ and a fall of 6 lb. rapidly at the end of the 30-minute period, which would lead to a maximum loss of CO₂ (from 3.9% to \( \frac{15}{15 + 6} \times 3.9 \)
\( = 2.8\% \)). This yields a mean figure of \( \frac{3.9 + 2.8}{2} \)
\( = 3.35\% \) CO₂ as the expected final carbon dioxide concentration at the end of a correctly conducted decompression.

It is of interest that a badly performed decompression, such as in this particular case, should actually have lessened the carbon dioxide accumulation in the lock. This happened simply because the major fall of pressure in the lock came later than it should have done after an appreciable concentration of carbon dioxide had accumulated. It is in no way a justification for changing the decompression procedure from that already laid down and well tested.

To overcome this accumulation of carbon dioxide four measures could be taken, two of them easily. (a) The men could pause for about 10 minutes after the strenuous climb up the ladder, to allow their metabolism to sink back to normal levels. This should help to reduce their carbon dioxide output while confined in the decompression lock. (b) During the decompression the air in the main lock could be flushed out. One way of doing this would be, for instance, to arrange a continuous “bleed” of air into the lock, compensated by having the main outlet slightly more open than usual. It would be technically difficult, however, to achieve a sufficiently large turnover of air to prevent significant carbon dioxide accumulation. Another procedure would be to raise the pressure by a few pounds every quarter of an hour, and then blow it off immediately. The men would probably feel the effects of these changes of pressure, and would have to be warned accordingly. This procedure, too, would be of limited ventilatory efficiency; if the pressure changes were limited to 5 lb./sq.in., it would not change the air more than once during the decompression. (c) “Decanting” is a third measure to be considered. Here, the men leave the blister after a very rapid decompression lasting one to two minutes, and immediately go into a more comfortable chamber where they are very rapidly compressed again and then decompressed at the rate laid down. This method would be hazardous if there were any possibility of delay before entering the comfortable chamber. (d) If further study showed this to be a serious problem one could easily install a compact blower, operated perhaps by compressed air, circulating the air through a soda lime system. It might, however, be a fairly elaborate undertaking to secure sufficient clearance of carbon dioxide.

(3) Influence of Physical Work.—The influence on the incidence of decompression sickness of the physical work done in making the rapid ascent immediately before decompression probably manifests itself by creating nuclei for the formation of bends in the men’s muscles. The work of E. N. Harvey and his colleagues (Harvey, 1951) has shown that bubbles do not form in supersaturated solutions unless there is a centre from which the bubble may grow, possibly of the nature of a minute, gas-filled hydrophobic crevice. Damage, or even simple contraction of muscles, is effective in creating nuclei. If practicable, it would be desirable to have a compulsory rest of 10 minutes before entering the lock.

(4) Chilling.—Finally, the chilling of the men is probably a factor more prominent in caisson work than in any other type, simply because the temperature at the working face of a caisson reaches such high levels. At the top of the caisson itself room is so cramped that it seems difficult to insist on there being dry clothing available for all workers before they enter the blister. But some form of heating installation is in fact recommended by the Institution of Civil Engineers, and this should be insisted upon.

The enumeration of these factors is not intended as any criticism of those who sink caissons. The difficulties to be surmounted in such undertakings are considerable and much more work on the medical problems involved is needed. But it is hoped that, by indicating what seem to be the likely causes of disability in this type of work, it may help to reduce the incidence of sickness to levels which allow the working force to function at full efficiency without discomfort and without the risk of consequent serious damage to health.

Summary and Conclusions

This paper describes some of the factors in the pathogenesis of decompression sickness during the sinking of a caisson.

Over a period of three months, there were 89 cases of bends, in a total of about 2,100 decompressions.

Among the contributory factors of what was sometimes a rather high rate of decompression sickness were: (a) inaccuracy in following the decompression procedure; (b) accumulation of carbon dioxide in the blister lock in which decompression took place; (c) vigorous muscular exercise immediately before the decompression, due to the ascent of a 70-ft. ladder; (d) chilling because of the considerable temperature difference between the hot
working face and the cold decompression chamber. It is recommended: (1) That lock-keepers should be properly trained and given sufficient authority for their task, that a recording barograph should be attached to any chamber in which decompression of men takes place; (2) that the blister lock should be ventilated or "cleared" during decompression, and that the men should wait for 10 minutes after climbing the caisson ladder before entering it so that their carbon dioxide production should have sunk to resting levels. This waiting period will also tend to reduce the effect of exercise in predisposing to bends; (3) if possible, dry clothes should be provided before entering the decompression lock, and the lock should be kept warm during decompression.

We wish to thank the contractor's engineer for his assistance. During this work W. D. M. Paton was a member of the Medical Research Council's scientific staff at the National Institute for Medical Research.

REFERENCES

THE OCTOBER (1956) ISSUE
The October (1956) issue contains the following papers:


Carcinoma of the Lung in Workmen in the Chromates-producing Industry in Great Britain. By P. Lesley Bidstrup and R. A. M. Case.

Cancer in Coking Plant Workers. By D. D. Reid and Carol Buck.

The Cytological Diagnosis of Bladder Tumours Amongst Dyestuff Workers. By J. G. S. Crabbe, W. S. Cresdee, T. S. Scott, and M. H. C. Williams.


Miscellanea:
Occupational Mortality: Coronary Heart Disease. By J. N. Morris.

The British Occupational Hygiene Society: First Provincial Meeting.
Particle Measurement in Industrial Hygiene. By G. L. Fairs.
Control of the Fluorine Hazard. By J. D. Paterson.

Book Reviews.
Index.

A number of copies are still available and may be obtained from the Publishing Manager, British Medical Association, Tavistock Square, W.C.1, price 12s. 6d.
Decompression Sickness During the Sinking of a Caisson: A Study of some Factors in the Pathogenesis of Caisson Disease
H. E. Lewis and W. D. M. Paton

Br J Ind Med 1957 14: 5-12
doi: 10.1136/oem.14.1.5