Toxic torts: a beginner's guide

By Peter Long

Chemical injury is the greatest threat to our quality of life in the 21st century, yet is probably the most misdiagnosed ailment facing the medical profession. Against this background, litigation lawyers are being consulted by clearly debilitated clients who usually have little, if any, medical evidence as to what has caused their disability.

For personal injury lawyers accustomed to less challenging litigation, such clients can present a daunting and often confusing prospect that can take them well out of their comfort zone.

THE HYMN BOOK

The key is in the history taken. This is where the lawyer may have an advantage over the medical profession, which has been forced by our national health system to adopt a 'wham-bam-thank-you-ma'am' approach to obtaining the patient's factual history.

Recent moves by state governments to restrict the fees that a lawyer can charge for taking instructions are, however, forcing some lawyers into the same approach. Like treating doctors, practitioners adopting shortcuts and arbitrary brevity in instruction-taking do so at their own risk.

The first step must be to take a complete proof of evidence, even though this task may take several days and usually has to be broken up because of client fatigue. No lawyer would accept a builder who erected their $500,000 family home without its foundations. Equally, no client should

be exposed to a $500,000 case being constructed without solid foundations.

Never forget that your client is his or her own best witness, expert doctor and lawyer. The client is intimately aware of what has occurred in his or her life and is often quite well read on the issue of chemical injury, given that usually no one else has been able to assist them. Every step outlined below must be done in complete and open consultation with the client, and specific instructions taken in relation to every report or document received or obtained.

This proof of evidence must be submitted to the client for approval and, once approved, it becomes the hymn book from which everyone involved in the case sings thereafter.

LAY EVIDENCE

The lawyer obviously has to identify the chemical that caused the injury, if possible, and obtain a material safety data sheet and a copy of the label from the chemical manufacturer. Such documents usually provide details on the adverse health effects of the chemical and may well corroborate your client's symptoms.

In numerous instances, the identity of the precise harmful chemical will not be known. For example, cigarette smoke has 4,000 chemicals, some 400 of which are known to be toxic, but no one knows precisely which of those, either individually or in combination, lead to the development of those conditions which the medical profession now readily accepts are due to cigarettes.

The synergistic effect of chemicals when combined can be overwhelming, and little research has been conducted on this aspect of toxicity. The lawyer may be faced with a situation in which the readily identifiable chemical in a particular case may not have been present in sufficient quantities, or not itself toxic enough to cause the problem, yet the client clearly has an injury. It could well be that other chemicals have combined to cause the damage. In such situations, the client may be left with little other than a temporal nexus.

Chemical injury is rarely confined to one person and statements must be taken from others within the same workplace, building, paddock, etc, as to their experiences with the subject fume or spray event. Do not be put off by the fact that some people with the same levels of exposure do not have the same symptoms as your client: that is entirely anatomically consistent. Chemicals such as organo-phosphates act as enzyme inhibitors. The more enzymes a person has, the less impact the organo-phosphate has on them. Humans do not all have exactly the same levels of enzymes.

It is trite to say that evidence of prior events and complaints, if available, must be gathered.

MEDICAL EVIDENCE

In toxic tort cases, requesting a report from a treating medical provider is fraught with risk as that doctor may not have been able to diagnose the condition, identify its cause or take an accurate history. The lawyer must be prepared to provide the client's history to the treating doctor when requesting the report, so as to ensure the doctor is singing from the same hymn book. Busy doctors invariably appreciate this history and are then able to write a more complete and useful report.

Chemical injury litigation is not for the faint-hearted.

Be prepared to see the word 'depression' used repeatedly: it is the final refuge of an overworked general practitioner or specialist physician.

Qualified medico-legal experts are rare, and extreme care must be taken in selecting an expert with the necessary qualifications, expertise and experience. Chemical injury litigation is expensive enough without wasting thousands of dollars on a useless report. The best experts also usually run busy practices and the lawyer will find that 'garbage in, garbage out' isn't a maxim that is solely applicable to the computer industry. The lawyer's task is to properly brief each such expert, which brings us back to the hymn book, together with such treating medical evidence and other evidence as is obtained.

A face-to-face session with each medico-legal expert is both mentally rewarding for the lawyer and enhances the return for the client's dollar spent on that expert.

EXPERT LIABILITY EVIDENCE

Good luck! No, seriously: selection and briefing is everything. Easy to say but how does one find an expert who can provide an opinion on hydraulics, mechanical engineering, noxious smells, chemistry, spray droplet size and the like? The same way lawyers go about making a car: they don't. They find someone who does. There are businesses out there who will find an expert for the lawyer. Let them do their job so you, as the lawyer, can do yours, but vet the ones they find to ensure that they are right for the particular case.

Often, the lawyer will need to obtain an expert report on liability in order to be able to draft the pleadings, yet such expert opinion must be conditional upon what is obtained via discovery, and ought to be expressed as such.

THE PARTIES

There is nothing different here to any other personal injury claim.

THE PLEADINGS

Aside from what every litigation lawyer already knows from running more conventional personal injury litigation; the following rules apply:

Rule No. 1: include everything, including the allegation that the known chemical on its own or in combination with other chemicals caused the injury. It is always easier to amend pleadings by deleting anything that is subsequently shown to be superfluous than to add additional information.

Rule No. 2: defendants are always 'embarrassed' by a plaintiff's pleadings in chemical injury cases, so get used to the complaint and stick to what you know is right.

Rule No. 3: expect to be in court more times on interlocutory >>

applications to strike out, dismiss, etc, in one chemical injury case than all of the hearings attended in all other cases in the last decade. It's called the 'deep pocket syndrome'.

PARTICULARS

Apparently, the sole reason for the invention of the wordprocessor. No different to the usual, just more of them.

SUBPOENA

Do it. Do it right. Do it early. Anyone who has a document that may assist the lawyer to properly prepare the client's case ought be asked to produce it. This includes the Australian Pesticides and Veterinary Medicines Authority, but expect to have to give undertakings regarding confidentiality.

The defendants will oppose any that may hurt them, so be prepared for more time in court. The more specific the schedule to the subpoena, the more chance of succeeding against such opposition.

DISCOVERY

Essential. Get leave early if leave is required. Specificity is a must, but also use separate catch-all requests. Expect cuteness from the defendant's legal representatives as the proceedings move away from the deep-pocket arena and into the semantics of particular words used in the notice of categories and classes.

This is the time when counter-attack is critical. If a defendant has not complied with discovery either at all or fully by the due date, file a motion immediately and set out the defects in a lengthy affidavit. Get it right and stand your ground. Time to give the defendant something worth being embarrassed about. Do it again and again until full compliance has been made. The duty judge or case management judge will soon start to see the trend and jump on the semantics.

RE-VISITING THE EXPERT EVIDENCE

Invariably, in toxic tort litigation, a prudent lawyer has obtained expert evidence prior to commencing proceedings. Obviously, once documents have been obtained under subpoena and by way of discovery, consideration must be given as to whether all or some of those documents ought to be placed before particular experts for comment. Again, it doesn't hurt to do this face to face.

Chemical injury is rarely confined to one person...but people with the same levels of exposure can present with very different symptoms.

NOTICE TO ADMIT FACTS AND AUTHENTICITY OF DOCUMENTS

A useful tool in chemical injury litigation and one often overlooked. The eventual hearing will be long enough. Take advantage of this mechanism to narrow down matters in dispute.

OFFERS OF COMPROMISE

An absolute 'must' in cases such as these where a three-to-four-week hearing is the norm rather than the exception. Obviously, such offers are effective only if they are made when the defendant is sufficiently well-informed to give the offer its due consideration. But the offer should be made at the earliest possible opportunity so as to maximise the client's chances of recovering the significant solicitor/client costs that can be incurred in such cases.

THE BURDEN OF PROOF

(See Appendix Causation – an illustration using organo-phosphate poisoning on page following.)

As stated so succinctly by Priestley JA in 2000 in the NSW Supreme Court in *Allen v Department of Agriculture*:

> 'The decision at first instance is a paradigm example of a feature of fact finding often found in cases involving medical issues. That feature is the major cleavage between proof of a fact in non-criminal court cases to the satisfaction of the fact-finding tribunal on the balance of probabilities and proof of a fact for scientific purposes to the satisfaction of those expert in the particular field of science. The latter kind of proof is much more rigorous and demanding than the former.
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> The two kinds of proof are quite different in their objects and methods, but are frequently the cause of confusion when medical issues are concerned. In many such cases, experts in the field of the relevant medical science give evidence of their expert opinion concerning the medical issue. Trained in the scientific method of proof, some such experts find difficulty in adjusting themselves, when giving evidence in court, to the lesser requirements of legal proof which, looked at from their scientific standpoint, they regard as inferior and unreliable. An expert who gave evidence for a party in litigation where there has been an adverse result reached by application of the legal standard of proof is quite likely to advise the party that the result is an unscientific and unsound one. Hence, many appeals by the indignant losing party.'

THE ANSWER

Chemical injury litigation is not for the faint-hearted. If you are up to the challenge of this form of litigation, you will find it intellectually stimulating, rigorous and highly rewarding. Just as civil litigation has been responsible for a great deal of beneficial social change and adverse behaviour amelioration, so chemical injury litigation offers benefits to the wider community. ■

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Appendix Causation – an illustration using organo-phosphate poisoning

Significant progress has been made over the last 25 years by the bio-scientific and medical professions in developing tests to detect the presence of harmful chemicals in mammals, including humans, and birds, a development that has received significant impetus since the first Gulf War.

Neurons communicate through the medium of the transmitter substance, acetylcholine ('ACh'), an acetic acid ester, in the synaptic vesicle. The neuron giving the message is referred to as a pre-synaptic neuron, at the end of which there is the synaptic knob. This knob contains the synaptic vesicles. Adjoining the synaptic knob, but separated by the synaptic cleft, is the post-synaptic neuron which receives the message. On the surface of the post-synaptic neuron is the post-synaptic membrane, which holds the ACh receptors.

The neurons communicate at the neuromuscular junction in order to affect action potential. When an action potential arrives at the synaptic knob, the synaptic knob absorbs Ca2+ from the surrounding extracellular fluid in the synaptic cleft and there follows exocytosis of ACh. The ACh crosses the cleft to bond with the receptors, which results in depolarisation of the post-synaptic membrane. This then initiates the action potential.

The enzyme acetylcholinesterase ('AChE') then removes the ACh while the action potential is propagated through to the next neuron. It is vital to remove the ACh quickly, as otherwise the action potential keeps going. Thus, people whose ACh is not removed quickly will get constant tremors.

Synthetic organo-phosphates are tailor-made to inactivate or inhibit the AChE enzyme. Tetraethyl pyrophosphate was the first to be synthesised in Germany in 1854 and dimethyl, diethyl and diisopropyl phosphoro-flouridates followed. They were first developed as insecticides during World War II and later as warfare agents aimed predominantly at the muscles that constrict the lungs. They sought to ensure that the message to breathe out was not removed but rather maintained, so that victims simply suffocated to death. The difference between an insecticide (phosphate) and a nerve agent (phosphonate) is one atom.

Organo-phosphates inhibit the effect of an AChE enzyme by causing the formation of the Michaelis complex; phosphorylation of the enzyme; a reactivation reaction; and aged phosphonyl enzymes. This results in muscarinic effects such as increased secretions; constriction of the pupils and the bronchi; abdominal cramps; and bradycardia (slow heart). There are also the nicotinic effects of fasciculations and tachycardia; and the effects on the central nervous system of headaches, dizziness, anxiety, confusion, convulsions, and coma.

Organo-phosphorus esters cause the neurotoxic actions known as cholerinergic neurotoxicity, organophophorus ester-induced delayed neurotoxicity ('OPIDN'), and organophophorus ester-induced chronic neurotoxicity ('OPICN').

Further research is needed into the interactions of exposures to different organo-phosphates simultaneously; the interactions of acute exposure to organo-phosphates after chronic pre-exposure to organo-phosphates; and the link with neuro-degenerative diseases. Current research on the effect of combinations of pesticides has found a significant synergism between some pesticides, which is more than simply additive. This synergy is referred to as the 'potentiation of toxicity'. The permitted levels of pesticides are set by the regulatory authorities without taking into account these proven synergistic qualities and their obvious effects on the potentiation of toxicity.

METHODS OF PROVING ORGANO-PHOSPHATE DAMAGE

The current methods being used to detect whether a mammal has been the victim of neuro-toxic damage include measuring neurite growth where the NB2a neuroblastoma cells are examined for the effect on neurite growth of the organo-phosphate, diazinon. Other research with neuropathic and non-neuro-pathic isomers of tricresyl phosphate ('TCP') has established that TCP inhibits the neurite growth in mice.

In addition, research has been undertaken into the effects of exposure to the organ-phosphate Sarin at high levels in the Matsumoto City (1994) and Tokyo subway trains' (1995) incidents, and at low levels in 1991 during the Gulf War destruction of the Khamislyah munitions dump by US forces. The two Japanese events caused cholinergic neurotoxicity, whereas the Iraqi experience resulted in OPIDN, which is a neurod-egenerative disorder characterised by a latency period ranging between six and 14 days; neuropathological lesions in the medulla of the brain, spinal cord and sciatic nerve; primary degeneration of the axon, followed by secondary degeneration of the myelin; species and age sensitivity; and inhibition of the neurotoxicity target esterase.

Of 237 organo-phosphates tested for OPIDN characteristics in one study, 106 were positive. One of those, which is a common industrial chemical, is tri-ortho-cresyl phosphate ('TOCP'). The metabolic activation of TOCP has been investigated and works as follows: first there is dearylation, via the Phase I oxidase enzyme CYP 450, of TOCP to di-o-cresyl phosphate; then hydroxylation of a methyl group; followed by cyclisation into saligenin cyclic-o-tolyl phosphate.

Individuals with increased activity of CYP 450 are more susceptible to TOCP-induced neurotoxicity, and the combined exposure to chemicals that increase the activity of the CYP 450 has been found to enhance TOCP neurotoxicity. By way of example, safrotin does not produce OPIDN while chlorpyrifos produces mild ataxia at 100 mg/kg, yet when safrotin and chlorpyrifos are combined at 100 mg/kg, the resulting compound causes paralysis. This synergistic effect arises out of the fact that safrotin binds to the plasma butyrylcholinesterase enzyme ('BChE') and prevents the BChE binding to the chlorprifos, leading to its persistence in circulation and increased delivery to the neurotoxicity target in the nervous system.

Research has found that repeated >>

doses of organo-phosphorus compounds are more efficient in producing OPIDN than large single doses. In fact, a low dose applied over a 72-day period led to the same OPIDN as a single dose 500 times higher. Further, organo-phosphorus compounds have more access to the nervous system and the neurotoxicity target through inhalation and skin penetration than through the gastro-intestinal tract. Inhalation is the second most effective route of entry, preceded only by intravenous injection.

Research has also identified a third mode of neurotoxic action, OPICN. This results from both large toxic doses or small sub-clinical doses of organo-phosphates. The clinical signs, which continue for weeks or years, consist of neurological and neurobehavioural abnormalities and damage is greater in the central nervous system than in the peripheral nervous system. The symptoms include headache; drowsiness; dizziness; anxiety; increased tension; apathy; restlessness; labile emotions; anorexia; insomnia; bad dreams; weakness; lethargy; fatigue; inability to concentrate; cognitive and memory deficits; depression; social isolation; neurological deficits; irritability; confusion; reduced motor co-ordination; and tremors.

Neuronal cell death resulting from early necrosis or delayed apoptosis is seen in various brain areas including the cerebral cortex, the hippocampal formation and the cerebellum and, because central nervous system injury predominates, improvement is slow and recovery is unlikely. OPICN is exacerbated by concurrent exposure to stress or other chemicals that cause neuronal cell death or oxidative stress. The apoptosis was confirmed using an apoptosis-specific stain transferase-mediated UTP nick end-labelling and by neuronal nitric oxide synthase immunohistostaining.

Significant death of cerebral cortex neurons results in muscular weakness and loss of strength, while loss of a significant amount of hippocampus neurons leads to progressive memory loss and results in learning disabilities. The loss of the purkinje cells in the cerebellum is believed to cause delays in initiating and terminating movements, terminal tremor at the end of movement, and disorders in the spatial co-ordination of hand and finger muscles.

Plasma can be analysed for TOCP and its various metabolites by high-performance liquid chromatography and liquid scintillation counting. It has been found that TOCP reached its highest concentration in plasma at 12 hours, while its metabolites attained their maximum concentration levels between 24 and 48 hours after dosing. The disappearance of TOCP from the plasma followed a mono-exponential kinetics with a half-life of 1.2 days. Di-o-cresyl hydrogen phosphate and o-cresyl dihydrogen phosphate were the major metabolites in the plasma, while dihydroxymethyl TOCP was present in trace amounts. Appreciable concentrations of saligenin cyclic-o-tolyl phosphate, which is believed to be the active neurotoxic metabolite, were detected in the plasma at all time points.

Thus, clients who were considered to have probably suffered from the organo-phosphate TOCP exposure could submit their plasma for analysis for concentrations of saligenin cyclic-o-tolyl phosphate. Those with concentrations of saligenin cyclic-o-tolyl phosphate must have been exposed to TOCP. This provides what so many judges want to see, namely, pathology. Further, the brain produces certain proteins only when there has been damage to the CNS and the blood can be analysed for the presence of these proteins.

The symptoms of some 40 farmers and farmhands exposed to organo-phosphate esters in sheep dip in the UK suggested symmetrical distal sensory neuropathy, which was confirmed by neuro-physiological analysis of the distal peripheral nerves. The researchers reported a characteristic pattern of involvement of 15 functional indices of autonomic nervous system dysfunction. In addition, the symptoms suggested that the thinly myelinated nerves were targeted by the esters and, again, the neuro-physiology was consistent with this.

Unfortunately for humans, all the organs of the body involve thinly myelinated nerves and the autonomic symptoms suggested that the target organs are in the skin, the large blood vessels, and the brain. This focus is different to other diseases and thus clearly distinguishable. A patient who has this suite of autonomic dysfunctions is the victim of organo-phosphate toxicity. Further, the skin's sympathetic mono-aminergic thermoregulation is affected but the cholinergic sudomotor is preserved, while the baroreceptors' and the brainstem's monoaminergic functions are affected but their cholinergic functions are preserved. Patients exposed to organo-phosphates showed evidence of malfunction of cardiac chemoreceptors: a unique cardiac effect where patients have the paradox of a normal heart rate, but it is mostly resting bracdycardia in association with abnormally low cardiac vagal tone.

The same research has studied miners with long-term effects of accidental carbon monoxide poisoning who were complaining of ill-health. Target-organ specific examinations of the autonomic nervous functions showed that the miners had different patterns of autonomic dysfunctions to the farmers and farmhands.

Other diagnostic methods being used include neuro-psychological assessment; analysis of the neuro-immunoendocrine function after toxic exposure; single photon emission computed tomography ('SPECT') scans; PET scans; and the test of variables of attention ('TOVA') psychometric test. Significant positive response has been achieved when patients with toxic encephalopathy have been treated with repeat mild hyperbaric oxygen treatment ('mHBOT').

SPECT-imaging of the brain for patients before and after the mHBOT has revealed stark changes in brain function. PET scans of the metabolic activity reveal similar results. TOVA is a continuous performance test which measures attention span, self-control, reaction speed and consistency, and has been used to evaluate patients with acquired ADD as part of their toxic encephalopathy. The test does not suffer from practice effect and is therefore excellent for evaluating progress following each mHBOT session. The mHBOT also improved the immune function of the patients and allowed impaired brain cells to recover. Similar improvements were achieved with pulmonary and endocrine function. ■